Las Memorias del V Convencion anual de la Asociacion Nacional de Especialistas en Ciencias Avicolas

Proceedings of 29th Western Poultry Disease Conference and 14th California Health Symposium

Acapulco, Gro, Mexico
April 22-25, 1980
22 al 25 de Abril de 1980

and

Riverside and Turlock, California
April 29-30, 1980

Cooperative Extension
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Asociacion Nacional de Especialistas en Ciencias Avícolas y Western Poultry Disease Conference dan su más sincero reconocimiento y gracias por la assistencia económica especial de los patrocinadores y miembros sostenientes mencionados aqui. A los contribuidores mayores y a los organizaciónes que han enviado oradores a nuestra conferencia para hacer de ella un programa sobresaliente; para todos ellos, nuestros conferencistas e invitados, muchas gracias.

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The leadership of Drs. Mosqueda and Ghazikhanian, the support of participants and others who contributed so very much to programs and arrangements, and the enthusiastic attendance helped tremendously in putting on meetings and preparing the proceedings. To all who contributed, our sincere thanks.

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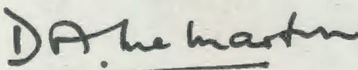

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DEDICATORIA EN MEMORIA DEL DR. P.P. LEVINE

Brillante y noble idea, la de los organizadores de esta Convención, el haberla dedicado a la memoria de ese maestro de muchas generaciones de estudiantes y brillante investigador de las enfermedades aviares, el admirado doctor P. Philip Levine.

Su carrera profesional se inicia al obtener el títulode bachiller, del Colegio de la Ciudad de Nueva York, dedonde era nativo. Continúa sus estudios en la Universidad de Cornell, a la que dedica toda su esistencia. Esta Universidad le otorga, en el mismo año, la maestria y el título de médico veterinario. Más adelante le confiere el doctorado.

Maestro, por naturaleza y por convicción, abraza la docencia en 1934, con el título de instructor. Diez años después, tras de recorrer todo el escalafón, se le nombra profesor y en 1961 Jefe del Departamento de Enfermedades de las Aves, del Colegio de Medicina Veterinaria de la Universidad de Cornell. Culmina su labor de enseñanaza al ser nombrado, al jubilarse, Professor Emérito.

Sus investigaciones en ornitipatología fueron variadas y fructíferas, dando testimonio de ello 74 publicaciones, en las que figura como autor o colaborador. Sus trabajos comprenden, entre otras, la parasitologá, virología y bacteriología aviares, siendo relevantes sus investigaciones en Eimeriasis, sobre todo las que se refieren al empleo de las sulfonamidas, trabajos pioneros y clásicos en el uso de los coccidiostáticos. Su admiración y respecto hacia dos de sus más queridos maestros se ponen de manifiesto al bautizar con los nombres de Hagan y Burnett a dos de las Eimerias por él descubiertas.

Como si fueran pocos sus méritos anteriores, se aboca, con su tesón caracteristico, a publicar una revista especializada en la que se aglutinen todos los escritos sobre Medicina Aviaria. Su labor fructifica en 1957, cuando aparece el primer número de Avian Diseases, órgano oficial, ahora, de la Asociación Americana de Patólogos Aviares, siendo el doctor Levine el fundador y primer editor de esta revista.

Viajero incansable, en 1947, tiene su primer contacto con Latinoamérica al visitar Brasil, país del que queda profundamente impresionado, al igual que de Reis y Nobrega, autores del "Tratado Sobre Enfermedades de las Aves". Aprende el portugués y más adelante el castellano, idiomas que le sirven para establecer, unto con su bonhomia y don de gentes, en casi toda América, sólidos y percederos lazos profesionales y de amistad.

Que *mejor tributo*, para este embajador universal de la ornitopatología que ofrecer su memoria esta Convención de profesionales de la Medicina Aviaria, que reune en el la a muchos de los que estavimos relacionados con él, como lectores, discípulos o amigos.

Eduardo Rivera-Cruz, MVZ, MS
2a. Cerrada de San Angel Inn #21
Mexico 21, d.f., Mexico

DEDICATION TO THE MEMORY OF DR. P.P. LEVINE

In organizing this Congress and Conference it is most fitting and a great and brilliant idea to dedicate it to the memory of that outstanding mentor and friend of many generations of students and the outstanding researcher on avian diseases, the esteemed Doctor P. Philip Levine.

His professional career was initiated with a bachelor;s degree from the City College of New York, of which city he was a native. Dr. Levine continued his studies at Cornell University from which he received his D.V.M. (1932), his M.S. and Ph.D. degrees.

A teacher by nature and conviction, he began as an instructor at Cornell in the Veterinary College in 1934, spending his entire professional career there. Ten years later he became Professor and in 1961 was made head of the Department of Avian Diseases. He finished his career as a Professor Emeritus, Cornell University.

Dr. Levine;s activities in investigating bird pathology were varied and fruitful; he authored and collaborated on 74 publications in avian pathology. These publications include investigations on parasitology, virology and bacteriology of the avian species. Among the prominent studies in avian pathology are those related with coccidiosis and the treatment of the disease with sulfonamides. Dr. Levine pioneered new methods for the control of *Eimeria* in poultry by using coccidiostats. After discovering two species of coccidia, Dr. Levine manifested his respect and admiration to his teachers when he named two species of coccidia that he isolated after Drs. Hagan and Burnett.

Besides conducting research and teaching, Dr. Levine sought to establish a journal on avian diseases. With the help of others, but serving as editor and manager, the first issue of "Avian Diseases" was published in 1957. This journal is now the official publication of the American Association of Avian Pathologists, as Dr. Levine wished. He was the first editor and after initiating it so successfully was named Honorary Lifetime Editor when A.A.A.P began publication.

Dr. Levine also gained valuable international experience by visiting Latin American countries where he learned Portuguese and Spanish to communicate and establish professional relationships with these countries. In 1947 he went to Brazil where the country and work of Reis and Nobrega and their classic "Treatise Concerning the Diseases of Birds" made a great impression on Dr. Levine, but he also contributed greatly to that country. He had great good relationships with people of all the American nations and for that matter all the world.

What better tribute to this avian pathology ambassador than to dedicate this Congress of professionals, students, and associates to the memory of their great good friend, teacher, and leader -- Dr. P. Philip Levine.

Eduardo Rivera-Cruz, MVZ, MS
2a. Cerrada de San Angel Inn #21
Mexico 21, d.f., Mexico

THE IMPORTANCE OF THE POULTRY INDUSTRY IN MEXICO

Enrique Salinas Aguilera, M.V.Z.
Director-General de Avicultura y Especies Menores

SUMMARY

The National Poultry Industry Became outstanding During the early 60's as the most dynamic activity, having the most advanced technology and accelerated growt, this provoked - during the initial months of 1971, a crisis of over production and forced incubators to reduce their capacity up to 1980, when they again operated at 100% of their installed capacity, and appearred new hatcmeries.

The estimated investment in aviculture amounts to $40'404'244,800.00, the estimated National Inventory Accounts for 173'176,832 fowls with an eggy production of 700,000 tons.

Main problems affecting present National Aviculture are:

A) Supply of Row Materials - Sorghum.- Poultry and swine requirements are estimated at 6'300,000 tons., production of sorghum is estimated at 4'700,000 tons.

B) D i s e a s e s.- High incidence of pullorosis and fowl typhord has caused great damages in the second semester of 1979 and during the 1980 months which have elapsed.

Policies established, to solve the more important problems affecting the Poultry Industry are:

A) Raw Materials-Sorghum.- The cultivated area will be increased by 24.1% in relation - with 1979 and imports will be programmed at 2"000,000 tons.

B) Handling of overproduction and eggy Price.- Due to convenience of National policies, - it is not possible to eliminate the official price of eggs, because of this, the government - has backed egg producers construct a plant will take care of seasonal excessive produc--- tion, to do this, the National bank of Rural Credit has granted $86'446,224.00 .

C) An agreement has been reached between CONASUPO and the National Union Of. Poultry -Growers, by means of which this official Enterprise (CONASUPO), guarantees the direct supply 70% of their sorghum needs, at the price of $2,450.00 per ton. and authorizes the - importation of the remaining 30% at the subsidized price.

Has been given to Poultry growers granting them priority in trailers with the official Enterprise Dina Nacional.

E) Pulorosis and Fowl typhoid.- the permanent comission for the control and erradica-- tion of Pulorosis and fowl typhoid was created, it proceeded prepare the necessary docu-- ments for the establishment of the eampaign. Its activities culminated on February 26, - 1980 with the publication, in the official Daily of which the National Campaign of Pulorosis and Fowl typhoid is Put into effect.

F) Overture for the eggs Hatching.- The decrease in the offer of recently hatched chicks- and Laying pullets provoked by the delayed increase in the hatching capacities and investments in new plants gave rise to the promotion of an overture for the explotation of light - and heavy Reproducers on the part of the Aviculture and minor species General Direction. It should be pointed out that within these policies backing has been given to small and medium Poultry growers so that they may obtain their integration and this remain in the market and finally the temporary authorization to import newly hatched Reproducers, fertile - eggs and newly hatched chicks for fattening.

O'ur Production Goals.- In 1989 we are shooting for 584,659.2 tons. and 792.053.1 tons. of fowl meat which represents and indrease of 48.32% and 100.93%.

In egg production we will shooting for 968,739 tons and 1'36 9,286 tons. which represent- 50.03% and 112.07% increase in relation with the 1980 Production.

PANORAMA ACTUAL DE LA AVICULTURA EN MEXICO

Enrique Salinas Aguilera, M.V.Z.
Director-General de Avicultura y Especies Menores

INTRODUCCION

ANTECEDENTES

En abril de 1977 fuí invitado por la Asociación Nacional de Especialistas en Ciencias Avícolas, para dar una plática en sureunión anual celebrada en la Ciudad de Monterrey, N.L. En esa ocasión, el tema que presenté se tituló "ANALISIS Y PERSPECTIVAS DE LA AVICULTURA EN MEXICO". Ahora vengo a' platicar con ustedes sobre el Panorma Actual de la Avicultura en el País.

Con objeto de establecer un marco de referencia a la plática - que nos ocupa en esta ocasión, mencionaré algunos de los - aspectos más importantes sobre los antecedentes de la Avicultura en México.

La Avicultura se sigue destacando como la actividad pecuaria - más dinámica y con tecnología más avanzadañ por tal motivo, México pasó de ser un País deficitario e importado -- de carne y huevo en la década de los 50, a ser un País que cubre sus necesidades a partir de la década de los 60. Sin embargo, no obstante este crecimiento y desarrollo acelerado, a fines del año de 1970 y a principios de 1971 se tuvo una crisis de sobre producción de huevo y pollo, que obligó que el Comité Nacional de Planificación Avícola, decidiera reducir la producción de pollita ponedora y pollito para engorda rcie´n nacidos, con objeto de regularizar la oferta con la demanda de estos productos y evitar un deterioro económico más grave a los avicultores, que el que ya habían sufrido a causa de esta crisis. El mecanismo utilizado, consistió en bajar el inventario de Reproductoras Ligeras y Pesadas y reducir la capacidad de incubación instalada.

Los avicultores dedicados a la cría de aves reproductoras y a la incubación de huevo, celebraron una Asamblea el 30 de Agosto de 1972, en la que se tuvo como resultado una proposición al Gobierno para generar un sistema de distribución de reproductoras, tendiente a disminuir la producción de pollita y pollito. El mecanismo propuesto consistió en una tabla de reducción a las capacidades de incubación, a través de la que se aplicaba una disminución del 2% sobre cada 74,999 huevos de capacidad instalada a partir de empresas incubadoras con capacidad de incubación superior a 200,000 hoevos. Con el mecanismo anterior hubo empresas que redujeron sucapaciad de incubación en un 4% otras en un 16% y algunas, las de mayor tamaño tuvieron que reducir hasta cerca del 40%.

La capacidad de incubación nacional para pollo de engorda pasó de 24' 568,000, lo que representa una reducción del 14.3%. En el caso de las Reproductoras Ligeras la capacidad de incubación se redujo de 8' 916,000 a 6' 076 000 huevos, lo que significa el 31.8%. A partir de este punto, la fijación anual de la cantidad máxima de Reproductoras en Producción, quedó a criterio del Comité Nacional de Planificación Avícola.

Este mecanismo se siguió llevando hasta el año de 1978, en que las compañías llegaron al 100% de su capacidad de incubación instalada. Para este año, las empresas avícolas dedicadas a la incubación-reproducción se habían recapitalizado y estaban preparadas para un crecimiento fuerte y sostenido.

Por otra parte se abrieron oportunidades para la entrada de nuevos incubadores, de manera particular para Asociacionesó Sociedades de Pequeños y Medianos Avicultures.

SITUACION DE 1977 A 1980

En el año de 1977, el Inventario fué de 1' 750,000 Reproductoras Pesadas y 473,000 Reproductoras Ligeras. Esto significó una producción de más de 350,000 toneladas de pollo y de 22'500,000 cajas de huevo.

Durante el primer trimestre de 1980, la producción de poll.ita ponedora de huevo com-

ercial, ha sido de un promedio de 3' 890,000 cada mes y la producción de huevo blanco ha llegado a 2'708,300 cajas mensuales ó sea 54,165 toneladas. Por otra parte se producen 3,600 toneladas mensuales de huevo rojo correspondiente a aves semipesadas comerciales. En cuanto a la producción rural de huevo, se consideran 10o,900 cajas mensuales con un peso aproximado de 2,038 toneladas. Esta producción proviene de la promoción avícola que por medio del Programa Nacional de Paquetes Familiares, se ha hecho en apoyo al Sector Rural.

De seguir este ritmo de producción de casi 60,000 toneladas mensuales de huevo, estimanos que en 1980 se producirán poco más de 700,000 toneladas. Esto representaría un incremento de 16.6% sobre el año de 1979, en que se produjeron 600,583 toneladas.

Por lo que se refiere a la producción de pollo de engorda, durante el primer trimestre del presente año se ha tenido una producción de 26'850,000 pollos rendidos, lo que representa un volúmen de 33,562 toneladas. De continuar con estre rit mo de producción, llegarémos a 402,744 toneladas. Sobre este particular estamos un poco abajo de las metas oficiales, que previeron para 1980 un volúmen de producción de 424,000 toneladas. La razón de esta situación, es la falta de aproximadamente, 450,000 Reproducturas Pesadas en producción, que se han perdido fundamentalmente a causa del problema de Tifoidea y Pulorosis Aviar y que nos representa un promedio de 5'265,000 pollos al mes, equivalentes a 7,900 toneladas de carne de pollo mensual. Sobre este particular se comentará con más detalle en el transcurso de esta plática, así como las medidas que se han tomado para contrarestar su efecto en las metas de producción de 1980.

El valor de la producción de Huebo y Pollo durante 1979 fué de $25,459;000,000.00 y participó con el 16.97% del valor de la producción ganadera. Para 1980 se estima en $27,361'000,000.00 y participará con un porcentaje del 17.21% del valor total de la producción ganadera.

INVENTARIO AVICOLA ESTIMADO AL 31 DE MARZO
DE 1980

	Crianza	Producción
Progenitoras Pesadas	107 873	76 682
Progenitoras Ligeras	14 453	20 069
Reproductoras Pesadas	1' 875 693	2' 174 000
Reproductoras Ligeras	394 212	514 935
Gallina Ponedora	20' 795 915	50' 000 000
Pollo de Engorda al ciclo		84' 786 000
Gallina Ponedora Semipesada	1' 440 000	3' 600 000
Aves criollas		7' 377 000
Sub-Total:	24' 628 146	148' 548 686
T O T A L:		173' 176 832

La Inversion estimada en la Avicultura es de $ 40,404'244,800.00.

PRECIO OFICIAL DEL HUEVO

En la actualidad el precio oficial del huevo es de $ 22.00 Kg. al público y de $19.80 al productor. Como consecuencia de que en México el huevo tiene un precio oficial, se ha notado cierto desaliento en los productores sobre todo en los más pequeños, cuyos costos de producción son más elevados. Larazón de ésto, es que constantemente se encuentran con que sus costos de producción rebasan el precio de venta, provocándoles pérdidas económicas significantivas.

Con esta política, hemos visto que no obstante nuestros esfuerzos para proteger y cuidar a los avicultores pequeños, estos han ido desapareciendo y solo permanecen aquellos que incrementan sus volumenes de operación y se integran en forma vertical, con objeto de abatir sus costos y tener mayor competitividad en el mercado. En el transcurso de esta plática se indicarán las políticas oficiales fijadas para resolver ó por lo menos amortiguar esta situación.

A partir del año de 1971 se han llevado registros cuidadosos de los precios vigentes, a través de los cuales se han podido identificar variaciones estacionales claramente

marcadas en cada año; de esta manera se puede observar que en los meses de Enero a Junio se presenta una baja sistemática en el precio del huevo que invariablemente coincide con una mayor oferta del mismo en el mercado y por consiguiente, la situación es contraria en los meses de Agosto a Diciembre, en que aparentemente se presentan faltantes de huevo, de manera particular en el mes de Noviembre, como consecuencia de una demanda temporal excesiva motivada por hábitos de consumo del Mexicano y una baja estacional en la producción del huevo.

Hasta el año de 1979 no se había hecho nada concreto pr parte del Gobierno ó de los productores para resolver esta situación, pero a partir de 1980 se ha planteado la primera alternativa de solucion, a través de la instalación de una Planta Industrializadora de Huevo.

PRINCIPALES PROBLEMAS QUE AFECTAN A LA AVICULTURA EN LA ACTUALIDAD

MATERIAS PRIMAS

El problema de abastecimiento de Materias Primas para la Avicultura, es uno de los más importantes en la actualidad y nos obliga a prestarle atención prioritaria, con objeto de no ver afectadas nuestras metas de producción. Para el presente año, hemos estimado necesidades de Sorgo para la Avicultura y la Porcicultura, del orden de 6' 300 000 toneladas y la producción estimada es de 4; 700 000 toneladas, por lo que el déficit de Sorgo es de 1' 600 000 toneladas. Si a lo anterior le añadimos un márgen de seguridad, para contar con reservas mínimas permanentes de 60 días de Sorgo almacenado en el País, las necesidades de importacion se elevan a 2' 000 000 de toneladas en números gruesos, requeridas para mantener el ritmo de crecimiento previsto para la Avicultura y la Porcicultura.

ENFERMEDADES

Tal como mencioné a ustedes hace un momento, en el renglón de enfermedades la presencia de Tifoidea y Pulorosis Aviar ha causado verdaderos estragos en la Avicultura de nuestro País en el segundo semestre de 1979 y lo que va de 1980.

El Dr. Angel Mosqueda Taylor, hará el comentario específico, desde el punto de vista epizootiológico de esta enfermedad, razón por la que unicamente me concretaré a indicar que los daños ocasionados por la misma, han traído como consecuencia un grave impacto económico en la Avicultura Nacional y un desajuste en la organización de la producción, de manera particular en el área de Progenitoras, Reproductoras Pesadas y pollo de engorda.

La confusión que provocó la utilización inadecuada de productos inmunizantes, como es el caso específico de la Vacuna R-9, ocasionó que lejos de desechar parvadas de Progenitoras y reproductoras infectadas, se hicieran diversos y múltiples intentos de tratamientos combinados con antibióticos que lo único que dió como resultado fué el provocar una diseminación de la enfermedad con altas mortalidades en las parvadas de Reproductoras y de Pollo de Engorda. El impacto económico provocado por esta enfermedad se refleja en la falta de 450,000 Reproductoras, ya que en este momento el País debiera tener más de 2; 600 000 Reproductoras Pesadas y cuenta unicamente con 2' 174 000 en producción. Además, parte de la producción de pollo proviene todavía de parvadas infectadas y por lo mismo, se tienen mortalidades que fluctúan entre el 15 y 30%.

Al márgen de las pérdidas directas que está provocando esta enfermedad en la parvada nacional, nos hemos visto precisados a autorizar importaciones temporales de Reproductoras, Huebo Fértil y Pollito recién nacido, que ya no eran necesarias en los últimos años, con objeto de que no se vea disminuída la producción de pollo de engorda, representando esto salida de divisas.

POLITICAS ESTABLECIDAS PARA LA SOLUCION DE LOS PROBLEMAS MAS IMPORTANTES QUE TIENE LA AVICULTURA EN MEXICO

MATERIAS PRIMAS - S O R G O

Con objeto de tener una mayor producción de Sorgo Nacional la superficie que se sembrará en este año es de un 24.1% mayor a la de 1979 y la producción esperada que es de 4; 677 000 toneladas, significará un incremento del 26.1%, con respecto al año anterior. Sin embargo, en virtud de que la demanda de Sorgo del Sub-Sector Pecuario es superior, se está requiriendo la importacion programada de 2; 000 000 de toneladas, con objeto de abastecer la totalidad de las necesidades.

MANEJO DE EXCEDENTES Y PRECIO DEL HUEVO

En virtud de que por conveniencia de Política Nacional, en apoyo a los intereses de los consumidores, no se podrá eliminar el precio oficial del huevo, el Gobierno apoyó a la

Unión Nacional de Avicultores, para la construcción de una Planta Industrializadora de Huevo, que maneje los excendentes esta cionales y les permita vender durante todo año al precio máximo autorizado.

Sobre este particular, el Banco Nacional de Crédito Rural, S. A., otorgó un crédito de $ 86;446,224.00 para la construcción de esta planta, la cual se está edifcando en la Ciudad de Querétaro, con una capacidad para captar 4,000 cajas diarias, así como para producir 4.6 toneladas de huevo líquido y 6.5 toneladas de huevo en polvo diariamente.

Este es el primer precedente formal, para regularizar el abastecimiento de huebo en el País y normalizar el precio del mismo.

CONVENIO ENTRE LA UNION NACIONAL DE AVICULTORES Y LA COMPAñIA NACIONAL DE SUBSISTENCIAS POPULARES

Con objeto de que el Gobierno sea congruente con los productores de huevo, en el sentido de que al fijarles un precio oficial máximo, les garantice que sus costos de producción se mantendrán dentro de un nivel que les permita tener la rentabilidad necesaria sobre sus inversiones, formalizó un Convenio entre la Unión Nacional de Avicultures y la CONASUPO, a través del que esta Empresa Estatal, les garantiza el abastecimiento directo del 70% de sus necesidades de Sorgo, al precio oficial de $ 2,450.00 la tonelada y les autoriza la importación del 30% adicional, a precio subsidiadoñ es dicir, que el Gobierno le devuelve al productor la cantida que exeda los $2,450.00 en sus diferentes compras de importación. Bajo esta premisa, de garantizar el abastecimiento del grano y unprecio máximo del mismo, es como se puede mantener rentable la producción de huevo, aún teniendo un precio tope oficial.

TRANSPORTE

Se ha dado apoyo a los avicultores otorgándoles prioridad en la adquisición de trailes y camiones ante la Empresa Estatal Dina Nacional, para que puedan movilizar con toda oportunidad sus Materias Primas y su producto terminado. Lo anterior es en virtud de que el País tiene en la actualidad un serio problema de falta de transporte.

ALMACENES, RASTROS Y FRIGORIFICOS

Existen créditos y líneas de descuento especial para invertir en Almacenes, Rastros y Frigorificos, así como créditos de avío amplios para la adquisición de Materias Primas.

TIFOIDEA Y PULOROSIS AVIAR

En Agosto de 1978 se reunieron en la Dirección General de Avicultura y Especies Menores, un grupo de Avipatólogos preocupados por la alta incidencia de Pulorosis y Tifoidea Aviar en el Pás, con la finalidad de analizar esta problemática y proponer medidas de acción, integrándose un grupo Inter Institucional y habiéndose constituído como consecuencia, la Comisión Permanente para el Control y Erradicación de la Pulorosis y Tifoidea Aviar, la cual se avocó a preparar los Manuales de Normas y Procedimentos, así como el Programa para establecer la Campaña y el Convenio que involucra a los Sectores Avícolas, culminando con la publicación en el Diario Oficial de la Federación el 26 de Febrero de 1980 del Acuerdo mediante el cual se establece en el Territorio Nacional con carácter general, obligatorio y permanente. "La Campaña Nacional contra la Pulorosis y la Tifoidea Aviar".

El Programa contempla la realización de tres etapas en la forma siguiente:

A) En la primera, se incorporará al 100% de las parvadas de Progenitoras y al 25% de la población de aves Reproductoras con duración aproximada de dos años.

B) La segunda comprende la incorporación del 100% de las parvadas de Reproductoras Ligeras y Pesadas de los Estados de Sonora, Nuevo León, Morelos, Puebla, Querétaro y Jalisco, con duración aproximada de dos años.

C) La tercera etapa, contempla la incorporación de las parvadas de Reproductoras del resto de País, con una duración de dos años.

Como aspecto importante de Programa, cabe mencionar el que quedará prohibido el uso de agentes inmunizantes en las parvadas de Progenitoras, así como en las Reproductoras que se encuentren dentro de la Campaña.

APERTURA PARA LA INCUBACION DE HUEVO

La alta incidencia de Salmonelosis y la lentitud en la ampliación de las capacidades de incubatión, así como de inversiones de nuevas plantas, trajo como consecuencia una disminución en la oferta de pollito recién nacido y pollita ponedora de huevo comercial, agravada por una demanda exagerada de pollito de engorda, que se originó por los altos

precios de otras carnes en el mercado. Por tal motivo, la Dirección General de Avicultura y Especies Menores, tuvo que promover la apertura de la explotación de Reproductoras Pesadas y Ligeras, ya que los incrementos convenidos en el último ciclo avícola con la Unión Nacional de Avicultores, no fueron suficientes para permitir el crecimiento adecuado de los incubadores ya existentes y de los nuevos autorizados.

Esta apertura temporal conará con un sistema de registro que incluirá a las operaciones ya existentes y a las Empresas de nueva creación. Para mantener el orden en el crecimiento de la Avicultura se cuenta con un sistema de información permanente que está siendo procesado en computadora.

APOYO A LOS PEQUEÑOS AVICULTORES.

Con objeto de facilitar la permanencia en el mercado de los Pequeños y Medianos Avicultores, se procede de acuerdo con los lineamientos del Plan Nacional Avícola a dar el primer paso para la integración vertical de los mismos, organizándolos y elaborándoles estudios de factibilidad para la instalación de Plantas de Alimentos Balanceados, Incubadoras y Granjas Productoras de Huevo y Pollo. Igualmente se les proporciona asistencia técnica en proyectos de comercialización, así como el posible financiamiento de los mismos; sea por medio de la Banca Oficial ó Privada.

AUTORIZACIONES TEMPORALES

Otra acción tomada para aliviar el desequilibrio de la oferta, ha sido la autorización temporal para la Importación de Reproductoras recién nacidas de Tip Pesado, Huevo Fértil y Pollito de Engorda recién nacido. Estas autorizaciones han sido calculadas en base a la deficiencia de Reproductoras Pesadas en Producción, que se tenían planeadas, constatando además en forma permanente, la demanda de pollito recién nacido. Las cantidades Importadas de Febrero de 1979 a Febrero de 1980 son:

Reproductoras Pesadas	702,473
Huevo Fértil para Reproductoras Pesadas	360,000
Huevo Fértil para producir pollito de Engorda	21'744,000
Pollito de Engorda recién nacido	1'924,000

El valor de estas importaciones es de $ 93'274,249.00.

METAS DE PRODUCCION DE HUEVO Y POLLO PARA FINES DE LA DECADA DE LOS 80

La población estimada en el País para 1980 es de 71'910,772 habitantes. Para 1985 se estima en 86'437,370 y para 1989 en 94'031,284.

Nuestras metas de producción para 1985 y 1989 en carne de ave son de 584,659.2 toneladas y 792,053.1 tons respectivamente, lo que representa un incremento de 48.32% y del 100.93%.

Por lo que se refiere al huevo esperamos alcanzar la producción de 968,739 toneladas y 1'369,286 tons., lo que representa el 50.03% y 112.07% de incremento con relación a la producción de 1980.

Respecto al incremento en el consumo per-cápita de carne de pollo será del 23.36% para 1985 y de 53.28% para 1989; en el caso del huevo será del 24.83% y del 62.14%.

Con lo expuesto a ustedes en párrafos anteriores, podemos tener la sequridad, que la Avicultura se sequirá comportando como la actividad más dinámica de la Ganadería Nacional y que cumplirá con sus metas previstas, participando adí de una manera relevante en el aporte de proteína de origen animal a la dieta del Mexicano.

SITUACION EN MEXICO DE LAS ENFERMEDADES AVIARES

M.V.Z. Angel Mosqueda Taylor
Departamento de Producción Animal:Aves
Facultad de Medicina Veterinaria y Zootecnia
Universidad Nacional Autónoma de México.
México 20, D. F.

RESUMEN:

Las diez enfermedades de las aves más frecuentemente diagnosticadas en el Departamento de Producción Animal: Aves de la Universidad Nacional Autónoma de México, fueron: Colibacilosis, Infección de la Bolsa de Fabricio, Enfermedad de Marek, Infección del Saco Vitelino,-Enfermedad Respiratoria Crónica, Tifoidea Aviar, Enfermedad de Newcastle, Encefalomielitis Aviaria, Coccidiosis y Paratifoidea, habiendo otras, como la Coriza Infecciosa y la Bronquitis Infecciosa, de similar importancia. Se comentan algunos factores relacionados con la -- existencia de las enfermedades aviares en el país.

SUMMARY:

The ten more frequently diagnosed Avian diseases at the Department of Avian Production of the National Autonomous University of Mexico were: Colibacilosis, Infectious Bursal Disease, Marek's Disease, Egg Yolk Infection, Chronic Respiratory Disease, Fowl Typhoid, Newcastle - Disease, Avian Encephalomyelitis, Coccidiosis and Paratyphoid, although others, such as -- Infectious Coriza and Infectious Bronchitis are of similar importance. Some comments about factors related with the existence of avian diseases in the country are made.

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En esta plática haré algunas consideraciones sobre el significado y dimensión de las enfermedades aviares en nuestro país. No es mi intensión exponer en forma precisa las estadísticas sobre el tipo de problemas patológicos y su frecuencia, sino más bien abordar en forma general la problemática de la Avicultura desde el punto de vista médico.

En México nos preocupa cada vez más el cuidado de los procedimientos de diagnóstico y control, tomando en cuenta las repercusiones de los errores que -- cometemos. De todas formas seguimos padeciendo de las "epidemias de diagnóstico', Algunas de las cuales aparecieron al surgir la infección de la bolsa de Fabricio, la artritis viral, la aflatoxicosis y otros problemas tóxicos, y ahora el síndrome de la baja de postura (EDS).

En resúmen, la panorámica es la siguiente:

En los años 1978/1979, las diez enfermedades más frecuentemente diagnosticadas en la Universidad Nacional Autónoma de México fueron:

Enfermedad	No. de casos *	%
1.- Colibacilosis	261	10.15
2.- Inf.de la Bolsa de Fabricio	198	7.69
3.- Enfermedad de Marek	121	4.70
4.- Infeccion del Saco Vitelino	94	3.65
5.- Enf.Respiratoria Crónica	88	3.42
6.- Tifoidea Aviar	58	2.25
7.- Enfermedad de Newcastle	44	1.71
8.- Encefalomielitis	33	1.28
9.- Coccidiosis	30	1.16
10.-Paratifoidea	27	1.04
*De un total de	2576	= 100%

Algunas de estas enfermedades son muy antiguas pero de presencia constante, y a las cuales no hemos podido vencer; me refiero a la Tifoidea Aviar y Pulorosis, cuyo combate por fortuna ya ha sido debidamente planeado y reglamentado mediante una muy necesaria Campaña Nacional de Control y Erradicación.

Otros padecimientos que representan serias pérdidas económicas son la Coriza Infecciosa (sobre todo por la explotación de parvadas de diferentes edades - en la misma granja), el Síndrome Asciti la Laringotraqueítis Aviar (en --- ciertas regiones del país), la Viruela Aviar y la Bronquitis Infecciosa.

Hay un buen número de padecimientos de importancia médica y económica, cuya existencia no queda constatada en ningún archivo de casos clínicos, pero sí en los registros de producción de la granja y en el bolsillo del avicultor, como son los abundantes casos de baja de producción de huevo o producción -- subóptima y las diarreas inespecíficas, las que no han sido investigadas a profundidad.

Tal vez valga la pena enlistar los recursos de diagnóstico más empleados en nuestra rutina de trabajo en México:

- exámenes bacteriológicos
- exámenes virológicos
- histopatología
- serología

Los recursos antes mencionados son aplicados primordialmente a la atención - de enfermedades de gallinas, siendo poco empleados en casos de padecimientos de otras aves. Esto se debe a que en México apenas se inicia la cría y explotación de especies como los pavos, patos, codornices y otras, como las de -- ornato. En éstas, es decir, las de ornato, hemos reconocidos varios casos de aspergilosis sistémica, colibacilosis, enfermedades neoplásicas, viruela, tifoidea aviar y pulorosis.

Hemos atendido algunos casos de probable clamidiosis en loros, sin que se -- haya precisado el diagnóstico.

En cuanto a la eficiencia que poseemos para la solución de problemas a nivel de campo y de laboratorio, podría decirse que nos vemos limitados en ciertos aspectos como el de la toxicología, serología, y aún en los campos de la virología y bacteriología.

Existen factores que obstaculizan el control de las enfermedades aviares en México, como por ejemplo:

a) Carencia de una reglamentación sobre la construcción de granjas avícolas tomando en cuenta: distancias entre ellas, propósito zootécnico, etc.

b) Falta de organización y exigencia para el reporte obligatorio - de enfermedades como la Salmonelosis, Enfermedad de Newcastle, y otras.

c) Insuficientes centros de capacitación en medicina aviar.

d) Reducido número de laboratorios de diagnóstico avícola

e) Pobre comunicación entre médicos veterinarios (congresos, conferencias y demás reuniones técnicas y científicas)

f) Empirismo en el diagnóstico y tratamiento de las enfermedades, lo que las convierte en perniciosas y sumamente costosas.

Finalmente, hay preguntas de las que nos gustaría tener alguna respuesta, tales como:

¿Cuánto están costando las enfermedades aviares al avicultor y al consumidor?

¿Cuándo exigiremos la adquisición exclusivamente de aves libres de Mycoplasma, Salmonella, etc., tanto de México como el del extranjero?

¿Cuándo se atacará el abuso en el empleo de antibióticos y otros - antimicrobianos?.

RESUMEN:
En México se han reconocido prácticamente todas las enfermedades económicamente importantes de las aves, siendo la Colibacilosis, Infección de la Bolsa de Fabricio, Enfermedad de Marek, Infección del Saco Vitelino, Enfermedad Respiratoria Crónica, Tifoidea Aviaria, Enfermedad de Newcastle, Encefalomielitis Aviaria, Coccidiosis y Paratifoideas los 10 padecimientos más frecuentes diagnosticados en el Departamento de Producción Animal: Aves de la Universidad Nacional Autónoma de México. Se comentan algunos factores relacionados con la presencia de enfermedades en el país.

SUMMARY:
In Mexico it is well recognized the presence of all the economically important avian diseases, being Colibacilosis, Infectious Bursal Disease, Marek's Disease, Egg Yolk Infection, Chronic Respiratory Disease, Fowl Typhoid, Newcastle Disease, Avian Encephalomyelitis, Coccidiosis and Paratyphoid Infectious. The ten more frequent diagnosis at the Universidad Nacional Autónoma de México. Some factors related with the existence of Avian diseases in the country are discused.

Los recursos antes mencionados son aplicados primordialmente a la atención de enfermedades de gallinas, siendo poco empleados en casos de padecimientos de otras aves. Esto se debe a que en México apenas se inicia la cría y explotación de especies como los pavos, patos, codornices y otras, como las de ornato. En éstas, es decir, las de ornato, hemos reconocido varios casos de aspergilosis sistémica, colibacilosis, enfermedades neoplásicas, viruela, tifoidea aviar y pulorosis.

Hemos atendido algunos casos de probable clamidiosis en loros, sin que se haya precisado el diagnóstico.

En cuanto a la eficiencia que poseemos para la solución de problemas a nivel de campo y de laboratorio, podría decirse que nos vemos limitados en ciertos aspectos como el de la toxicología, serología y aún en los campos de la virología y bacteriología.

Existen factores que obstaculizan el control de las enfermedades aviares en México, como -- por ejemplo:

a) Carencia de una reglamentación sobre la construcción de granjas avícolas tomando en cuenta: distancias entre ellas, propósito -- zootécnico, etc.

b) Falta de organización y exigencia para el reporte obligatorio de enfermedades como la Salmonelosis, Enfermedad de Newcastle, y otras.

c) Insuficientes centros de capacitación en medicina aviar.

d) Reducido número de laboratorios de diagnóstico avícola.

e) Pobre comunicación entre médicos veterinarios (congresos, conferencias y demás reuniones técnicas y científicas).

f) Empirismo en el diagnóstico y tratamiento de las enfermedades, lo que las convierte en perniciosas y sumamente costosas.

Finalmente, hay preguntas de las que nos gustaría tener alguna respuesta, tales como:

¿Cuánto están costando las enfermedades aviares al avicultor y al consumidor?

¿Cuándo exigiremos la adquisición exclusivamente de aves libres de Mycoplasma, Salmonella, etc., tanto de México como del extranjero?

¿Cuándo se atacará el abuso en el empleo de antibióticos y otros antimicrobianos?

POULTRY DISEASE REPORTING IN THE WESTERN REGION (A.A.A.P)

R.K. Edson, D.V.M., M.P.V.M.
Department of Epidemiology and Preventive Medicine
School of Veterinary Medicine
University of California, Davis, CA

The Western Region (Mexico, 4 western provinces of Canada, and 13 western states of the USA) began reporting disease diagnoses to the AAAP Committee for Disease Reporting and Nomenclature in 1969. The region has reported in 8 of the 10 years from 1969 to 1978. Canada has been the most consistent reporter (94%), followed by Mexico (63%)and the western states (38%). The objectives of this report to ANECA-WPDC are five:

1) To discuss briefly the objectives of the AAAP Committee and its report;
2) To discuss the AAAP disease reporting procedure;
3) To describe the report format, its evolution, and possible shortcomings;
4) To illustrate use of the report in studying disease trends with respect to time and location;
5) To propose that the Western Region establish a standing committee comprised of Mexican, Canadian, and USA members whose responsibility it would be
   (a) to compile the AAAP report each year;
   (b) to report the year's disease developments at the WPDC meeting each year;
   (c) to suggest further improvements in and uses for the AAAP report to the AAAP Committee.

COMUNICADO DE LAS ENFERMEDADES AVIARES EN LA REGION OCCIDENTAL (A.A.A.P.)

R. K. Edson, D.V.M., M.P.V.M.
Departmento de Epidemiologia y Medicina Preventiva de la
Escuela de Medicina Veterinaria de la
Universidad de California, Davis, CA

La región Occidental (Mexico, 4 provincias occidentales de Canada y 13 estada occidentales de U.S.A.) emperzaron reportando el diagnostico de enfermedades al Comité de la AAAP para el Comunicado de Enfermedades y su Nomenclatura en 1969. La region ha reportado en 8 de los 10 años desde que se inicio, de 1969 a 1978. Canada ha sido el más consistente en reportar (94%), sequido por México (63%) y los estados occidentales de U.S.A. (38%). Los objectiva de este reporte a la ANECA-WPDC son sincoñ

1. Discutir someramente los objectivos del Comité de la AAAP y su reporte
2. Discutir el procedimiento del comunicado de las enfiermedades a la A.A.A.P.
3. Describir el formato del reporte, su evolucion y posible jallas
4. Ilustrar el uso del reporte en el estadio de las tendencia de las enfermedades con refecto el tiempo y lugar
5. Proponer que la Region Occidental establezca un Comité lijo compreudido por miembros de México, Canada y U.S.A., y cuya responsabilidad seráñ
   a. Recopilar el reporte de la A.A.A.P. cada año
   b. Reportar el desarrollo de las enfermedades su el año ante la reunión anual de la W.P.D.C.
   c. Sugerir más mejoramientos y usos del reporte A.A.A.P al Comite de la A.A.A.P.

DISEASE REPORTING IN THE WESTERN REGION

R. K. Edson, D.V.M., M.P.V.M.
Department of Epidemiology and Preventive Medicine
School of Veterinary Medicine
University of California
Davis, California 95616

The objectives of this report are four:

1) to discuss the objectives of the AAAP Disease Reporting and Nomenclature Committee.

2) to propose the establishment of a permanent Disease Reporting Committee for the Western Region.

3) to discuss the format and possible shortcomings of the current AAAP report.

4) to demonstrate use of the report in study of avian disease trends.

1. Objectives of the AAAP Committee. The Committee has two primary objectives. The first is to establish a uniform disease nomenclature for use in describing and identifying avian diseases. The second objective is to assemble an annual avian disease report by region (state, province, etc.) for Canada, Mexico and the USA. The goal of standardizing nomenclature has largely been achieved; periodic name changes still occur as "new" diseases are described and as word usage changes. The disease reporting goal has been achieved for over 10 years, although the reports tend to be rather variable in terms of the number of areas reporting. The Committee does not undertake to analyse or summarize the disease reports.

2. Establishment of a Disease Reporting Committee in the Western Region. Since the inception of the AAAP report the contributions of the Western Region have been one-person efforts. Those who have performed this task in recent years feel that the job would be better done by a committee whose members (perhaps 4 to 6) represented all 3 reporting countries. A committee effort would decrease the individual responsibility, and it would probably produce a more complete report. For example, in 1978 only 4 of 13 Western Region States are represented in the report, and the best record achieved for this region was a reporting of 10 of 13 states in 1969. Also, in past years reports from Mexico have usually come from only one source. Mexican committee members would be better able to develop other reporting sources. Reports from Canada currently to present little problem, since these are abstracted from one official government document for the entire country.

3. Format and Possible Shortcomings of the Shortcomings of the Current AAAP Report. The format of the report can be seen in Avian Diseases 24(2):533-568. The author of this WPDC report discussed the AAAP avian disease report with several colleagues and the following possible limitations of the report were noted:

a) There are no longer any data summaries.

b) The desirable regional nature of the USA portion of the report has been lost due to the change to complete alphabetic format.

c) The report is difficult to read, possibly due to number of entries, or due to unnecessary inclusion of zeros.

d) Two-letter state abbreviations (e.g., AL, AK, AR, AZ) cause confusion, especially for non-USA readers.

e) The list of diagnoses is too limited; some others should be considered.

f) Non-reporting regions (states, etc.) should be noted.

g) Some use of graphics might make the report more "palatable."

h) Data are not meaningful because populations-at-risk are not specified. Therefore, it is not possible to compute disease rates.

i) The report does not adequately relate diseases to types (e.g., broiler, layer) or age of poultry. Some felt that the "other" category of birds needed greater definition.

j) Information is not in retrievable form for analytic purposes. The yearly reports should be stored on computer disk or magnetic tape for easier use in future.

These criticisms will be brought before the AAAP Committee in July 1980 at the AVMA Meeting in Washington, D. C. A report of that discussion will be given at WPDC in March 1981.

4. An Example of Use of the AAAP Report in the Study of Avian Disease Trends. Fowl cholera is an important disease problem of turkeys. During the past decade there has been a profound change in fowl cholera vaccination practices in the USA. There has been a change from almost exclusive use of killed *Pasteurella multocida* bacterins to the use of live orally-administered "avirulent" *P. multocida* vaccines. Reports of results are confusing. Some say that the live vaccine (alone or in combination with bacterins) has solved the cholera problem. Others feel that the turkey fowl cholera problem is still increasing. The problem is one of determining whether the fowl cholera rate has increased or decreased over the past 10 years. My approach to this question was as follows:

a) Count the number of cases of fowl cholera in turkeys in Minnesota, Missouri, and in the combined states of North and South Carolina for each year from 1969-1977 (Fig. 1). Add all 4 states' data together to get total fowl cholera diagnoses (Fig. 2).

b) Estimate the number of turkeys grown in each of the states from data given in Agricultural Statistics: 1970-1979 (U.S. Government Printing Office) (Fig. 3). Add these data together to get the 4-state population total (Fig. 4).

c) Divide the number of fowl cholera diagnoses in each of the 3 regions by the number of millions of turkeys raised in each of the regions to produce a fowl cholera rate of sorts (= number of diagnoses per million turkeys raised). Compute the same rate for the population totals. All 4 rates (Missouri, Carolinas, Minnesota and combined) are shown in Fig. 5.

Results. The turkey populations and the numbers of fowl cholera diagnoses increased during the 9 year period of study (Figs. 1-4). However, the rate of cholera diagnosis appears relatively stable over the 9 year period (Fig. 5). If we assume that disease incidence is the major influence on number of diagnoses, then it appears that the changing vaccination strategy of the 1970's has had little effect on fowl cholera in turkeys. It also appears that fowl cholera was no greater a problem in 1977 than it was in 1969. Failure to reduce the fowl cholera diagnostic rate suggests that new studies of the epidemiology of this disease are warranted.

A similar approach was used to study the trends in diagnosis of *Arizona* and *Mycoplasma gallisepticum* in California turkeys (Figs. 6 and 7) and for Marek's Disease in California chickens (Fig. 8) form 1969-1978. There was a marked decline in the diagnostic rate for each of these diseases during the study period. These results suggest that the disease intervention strategies employed (egg dipping, day-old poult injection for *Arizona*; serologic testing and slaughter for *M.g.*; vaccination for Marek's Disease) were at least partly successful.

Fig 1. Fowl Cholera diagnoses by state and year.

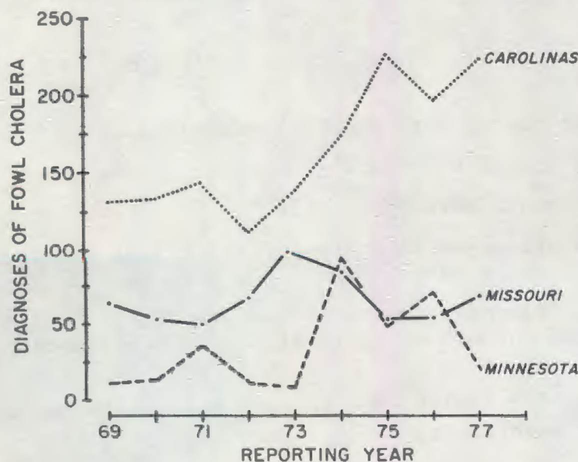


Fig. 2. Fowl Cholere diagnoses (accumulated for Minnesota, Missouri, the Carolinas): 1969-77

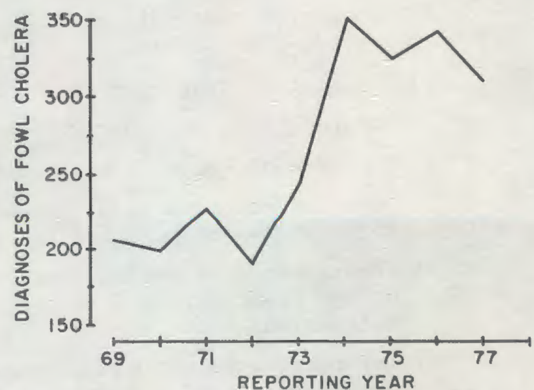


Fig. 3. Numbers of turkeys raised by state and year: 1969-77.

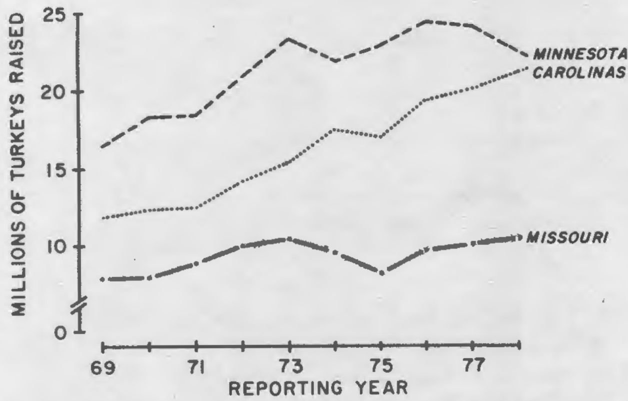


Fig. 4. Numbers of turkeys raised (accumulated for Minnesota, Missouri, the Carolinas): 1969-77.

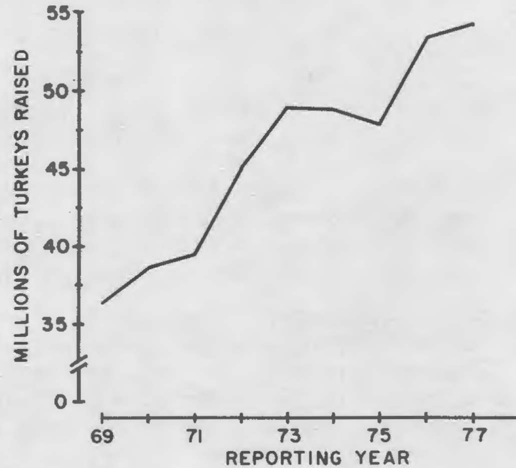


Fig. 5. Fowl Cholera rates (number of diagnoses per million turkeys raised) by state and year.

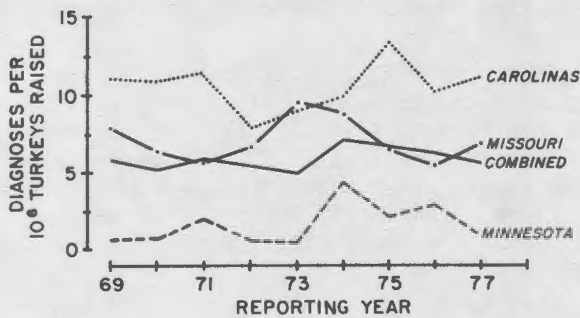
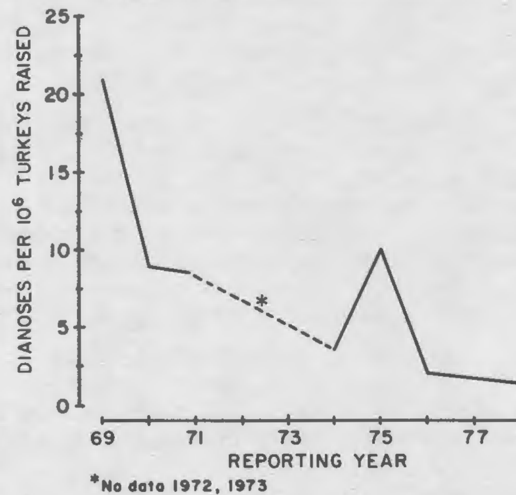
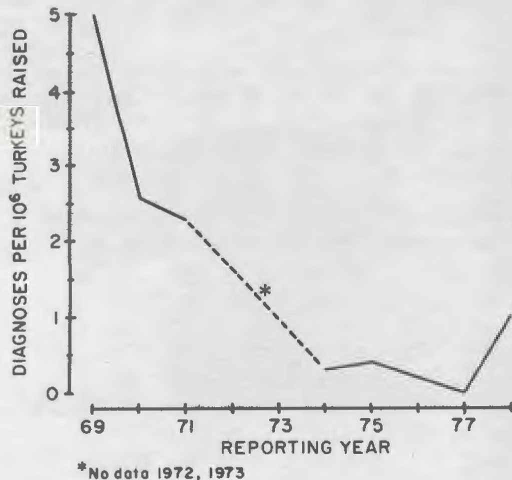


Fig. 6. Arizona rate for California: 1969-78



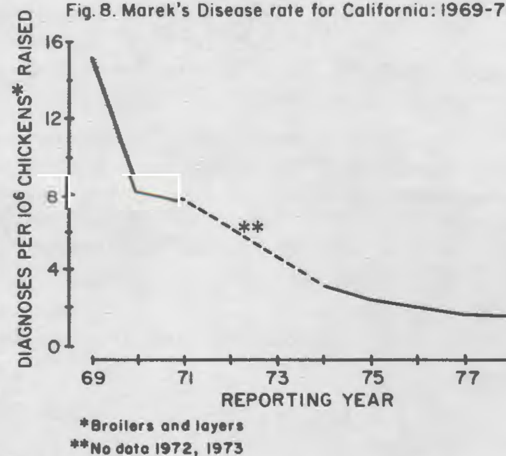
*No data 1972, 1973

Fig. 7. Mycoplasma gallisepticum rate for California: 1969-78.



*No data 1972, 1973

Fig. 8. Marek's Disease rate for California: 1969-78.



*Broilers and layers
**No data 1972, 1973

NOTE: The following paper is presented with comments by prior readers because of its pertinence to the subject of disease reporting. Detail tables are not included but are available from the authors if desired.

A FOUR-YEAR SUMMARY OF AVIAN DISEASES DIAGNOSED IN CANADIAN PATHOLOGY LABORATORIES

J. Biely and D. Douglas
University of British Columbia, Vancouver, BC, V6T 1W5

"This summary of avian disease diagnoses in Canada for the years 1973-76 is an interesting way of handling the data presented annually. AAAP should discuss the possiblity of adding such a summary to the annual report or to replace the detailed report with an analysis such as this."

"This paper forces one to consider the value and deficiencies of the current disease reports in Avian Diseases and in the Canadian Veterinary Journal. The authors are quite correct in their assertion that these published reports require analysis and interpretation to grasp the significance of the data. It is also true that the data published does not allow one to ascertain such highly significant points as the population at risk or the economic significance of the different diseases diagnosed."

Records of the important avian diseases diagnosed in Canada and the USA have been published for a number of years by both the Annual Poultry Diseases Conference in Canada and the American Association of Avian Pathologists respectively. In order to make the data comparable, a uniform terminology has been adopted by most diagnostic laboratories. The data reveal the incidence of diseases diagnosed in poultry flocks in the respective provinces and states.

Summaries of avian disease reports from 40 states in the USA, 10 provinces in Canada and Mexico were published in the January-March issue of Avian Diseases (Vol. 23 No. 1, 1979 pages 241-277). Summaries of poultry diseases diagnosed by Veterinary Laboratories in Canada have been published in the Canadian Veterinary Journal since 1973 (2-5). These reports record the important diseases diagnosed by Veterinary Laboratories across Canada. Wherever possible, the specific name of the disease is used. Some incompletely defined diseases, because of their frequent recurrence, were also included. The terminology corresponds closely to that recommended by the American Association of Avian Pathologists.

The data made available by each of the laboratories in Canada and the USA provide an invaluable source of statistical information. However, unless analyzed and interpreted, it is difficult to grasp the significance of the data. In the present paper, an attempt has been made to classify or categorize the diagnoses according the specific causative agent of the disease (virus, bacteria, protozoa, etc....) or common trait or entity (metabolic disorder, faulty management, etc...).

DATA

The data for the present report was obtained from summaries published for the years 1973, 1974, 1975, 1976 (2-5). The diagnoses in some of the reports were classified under four headings: B--broilers, C--chickens, T--turkeys, and O--other avian species. For this study, however, in order to focus the salient findings of the detailed reports the diagnoses were combined.

In all of Canada, 55,277 consignments were diagnosed, of which 9,436 were from the province of British Columbia. A consignment consists of birds of the same source and environment and may include one or more birds. Thus the total number of birds submitted for diagnosis for the whole of Canada may represent 100,000 individuals during the four-year period. Over 100 different causes of disease were recorded.

The diagnoses reported by the B.C. Animal Pathology Laboratory and all Canadian Animal Pathology Laboratories, for British Columbia and Canada are shown in graphical form in Figures 1 and 2, and in tabular form in the tables.

DISCUSSION

It is interesting to note from Table 1 that six categories made up slightly more than 50% of the total diagnoses in Canada. With the exception of a severe outbreak of infectious laryngotracheitis in B.C. in 1978 and a few sporadic outbreaks of Newcastle disease in Ontario, no serious epidemics occurred in the four years 1973-1976. Among the infectious diseases, those of bacterial origin were responsible for 15.20% of the diagnoses

while viral and protozoal diseases were 6.50% and 5.60% respectively.

Disorders of the digestive and excretory systems were second in percentage of occurrence (9.28%). Enteritis, hepatitis and nephritis may be caused by a specific agent or as an aftermath of bacterial, viral or mycotoxin infection or intestinal parasitism. Constitutional disorders accounted for 8.70% of the diagnoses; this category includes pendulous crops in turkeys, internal egg laying, egg bound, peritonitis, bumble foot, trauma, vices such as cannibalism, vent picking, beak necrosis, etc. According to Gordon (1) constitutional disorders in flocks where infectious conditions are not a factor constitute a serious total loss of some 20% of the total yearly mortality in laying birds.

Metabolic disorders were diagnosed in 7.12% of the consignments in B.C. and 7.49% in the whole of Canada. This ill-defined group includes such conditions as fatty liver syndrome, dissecting aneurysm in turkeys, starve-outs in baby chicks and poults, laying cage fatigue, acute death syndrome in broilers, leg disorders, gout, watery droppings, etc.

It is rather disconcerting that 24% of the diagnoses were of an undetermined or sporadic nature. This category may include such conditions as impaction of the crop, proventriculus, or gizzard; intestinal intussusception, vent gleet, laceration, acute hemorrhages from the liver, ovary, heart and unidentified tumors, etc. The occurrence of sporadic conditions is unpredictable and quite often is detected only on post-mortem examination.

SUMMARY

The results of diagnoses of avian diseases carried out in Canadian Veterinary Laboratories during 1973, 1974, 1975, and 1976 were analyzed as to cause and incidence. A total of 55,277 consignments were subjected to examination in Canadian Patholog Laboratories. Over 100 specific causes and entities were diagnosed; 52.8% of the cases accounted for six disease categories, 23.1% by 10 categories and 24% of the diagnoses were of undetermined nature or sporadic occurrence. Greater awareness on the part of the poultrymen of disease preventive and control measures are essential if morbidity and mortality are to be held to a minimum.

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Table 1. Percentage of consigments by causative agents: 1973-1976

Causative Agent	British Columbia 1973	1974	1975	1976	Avg.	Canada 1973	1974	1975	1975	Avg.
1. Leukosis	4.1	0	7.2	6.1	4.35	2.5	2.4	4.5	3.7	3.28
2. Marek's disease	2.0	12.3	8.7	4.4	6.85	3.6	3.8	4.0	3.2	1.65
3. Tumors	0.6	0.5	0.7	2.1	0.98	2.2	2.1	1.9	2.2	2.10
4. Viral	15.6	14.8	6.3	9.9	11.65	6.5	7.7	4.8	7.0	6.50
5. Mycoplasma	4.2	2.6	3.7	3.2	3.43	3.2	3.1	2.1	2.9	2.83
6. Bacterial	15.3	12.5	10.9	8.1	11.70	15.1	16.2	14.9	14.6	15.00
7. Protozoan	4.3	3.9	4.5	2.3	3.75	7.7	5.1	5.3	4.3	5.60
8. Fungal	0.7	0.8	0.5	0.4	0.60	2.7	2.8	2.1	1.7	2.33
9. Internal parasites	0.8	0.8	1.2	0.4	0.80	1.4	1.4	1.5	0.9	1.30
10. External parasites	0.3	0.6	0.5	5.3	1.68	0.8	0.8	1.6	1.9	1.28
11. Nutritional	5.8	2.4	2.4	2.7	3.33	4.4	3.6	2.5	2.8	3.33
12. Metabolic disorders	3.8	6.1	5.6	10.4	6.48	8.1	6.7	7.6	7.6	7.50
13. Digestive & exretory disorders	4.9	4.4	4.7	6.4	5.10	10.6	9.1	9.3	8.1	9.28
14. Constitutional disorders	6.8	6.5	10.6	7.5	7.85	7.9	6.9	10.6	9.4	8.70
15. Toxicosis-poisoning	1.2	1.2	0.2	0.9	0.88	0.4	0.6	0.7	0.9	0.65
16. Accidental-Misc.	2.7	1.8	3.1	3.5	2.78	2.3	2.1	2.6	2.7	2.43
17. Undetermined and sporadic conditions	26.8	28.8	29.2	26.2	27.75	20.6	25.6	24.0	25.9	24.03
Total	99.9	100.0	100.0	99.8	99.96	100.0	100.0	100.0	99.8	99.99

Table 2. Percentage of causative agents of diseases diagnosed in British Columbia and Canadian laboratories, 1973-1976.

Cause		British Columbia	Canada
Bacterial diseases		11.7	15.2
Digestive and excretory disorders		5.1	9.3
Constitutional disorders		7.9	8.7
Metabolic disorders		6.5	7.5
Viral diseases		11.7	6.5
Protozoan diseases		3.8	5.6
	Total	46.7	52.8
Marek's disease		6.9	3.6
Nutritional disorders		3.3	3.3
Leukosis		4.4	3.3
Mycoplasma diseases		3.4	2.8
Accidental-Miscellaneous		2.8	2.4
Fungal diseases		0.6	2.3
Tumors		0.1	2.1
Internal parasites		0.8	1.3
External parasites		1.7	1.3
Toxicosis-poisoning		0.9	0.6
	Total	24.9	23.1
Undetermined and sporadic conditions		28.0	24.0

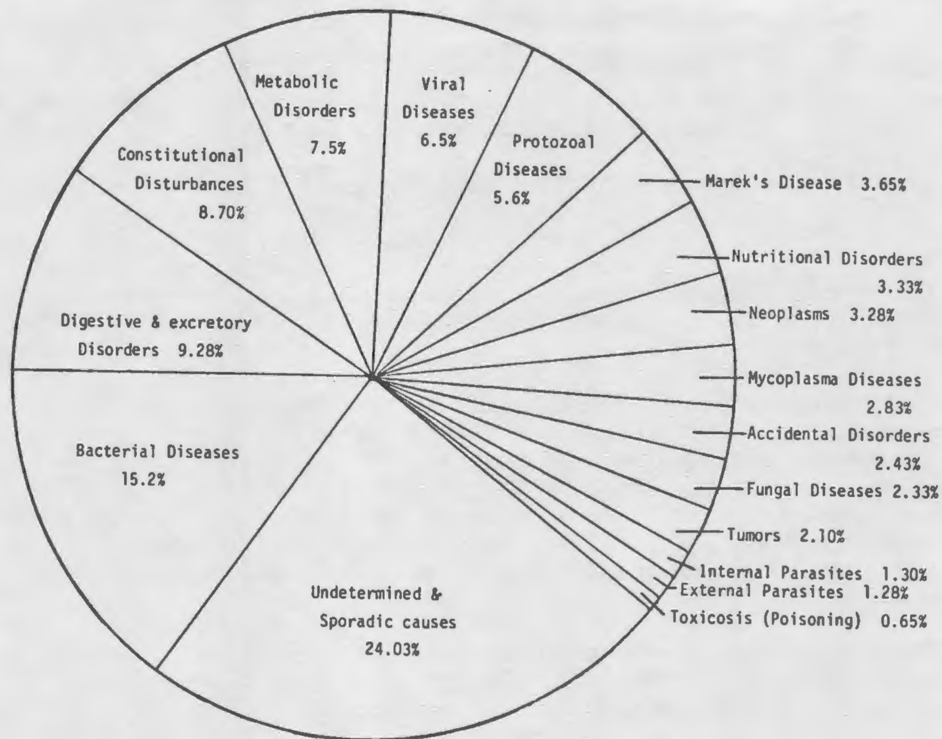

Figure 1. Average percent of diseases diagnosed at all Canadian pathology laboratories, 1973-76 (54,688 Total Consignments)

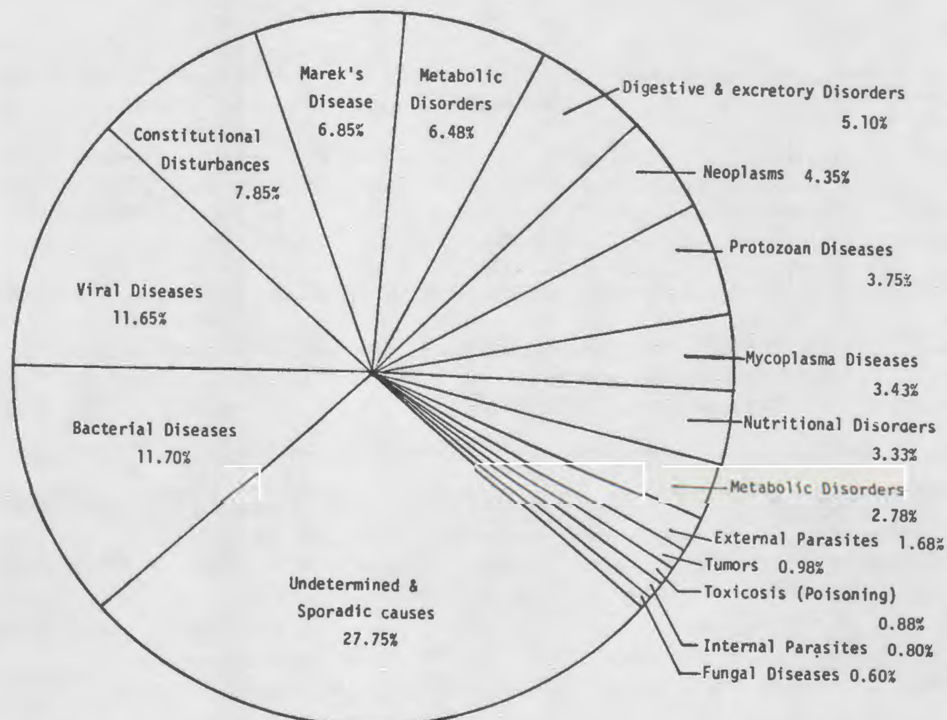

Figure 2. Average percent of diseases diagnosed at all B.C. provincial pathology laboratories, 1973-76 (9,441 Total Consignments)

EFECTO DE LOS ANTICUERPOS PASIVOS SOBRE LA INFECCION DE LA BOLSA DE FABRICIO Y SU INMUNIZACION ACTIVA.

Benjamín Lucio M. Departamento de Producción Animal: Aves. Facultad de Medicina Veterinaria y Zootecnia. Universidad Nacional Autónoma de México. México, 20, D.F.

Stephen B. Hitchner. Department of Avian and Aquatic Animal Medicine. New York State College of Veterinary Medicine. Cornell University. Ithaca, N.Y. 14853.

En una serie de experimentos, algunos de ellos ya publicados, se estudió la interacción entre la inmunidad pasiva y el virus de la infección de la bolsa de Fabricio (VIBF).

Las gallinas adultas inmunizadas con una emulsión múltiple de VIBF inactivado reaccionaron con niveles elevados de anticuerpos, que se mantuvieron estables durante las 50 semanas del periodo experimental. Los pollitos obtenidos de ellas resistieron totalmente la atrofia bursal cuando se les desafió con VIBF en las primeras cuatro semanas de edad. La protección conferida a los pollitos nacidos de gallinas vacunadas con virus muerto en suspensión, o no revacunadas, fué parcial desde el primer desafio a la semana de edad y fué de menor duración.

La inmunidad pasiva del pollito interfirió con las vacunas de virus muerto en suspensión o en emulsión múltiple, o cuando se inyectó un VIBF vivo modificado por via intramuscular o intrabursal.

Los pollitos con niveles elevados de anticuerpos, al ser inoculados con VIBF de campo no sufrieron atrofia bursal ni inmunodepresión y su respuesta activa fué inferior a la de los pollitos totalmente susceptibles. Además en este experimento los anticuerpos pasivos protegieron al pollito de la inmunodepresión durante cuatro semanas, dos semanas mas de lo que duró la protección a la atrofia bursal.

EFFECT OF PASSIVE ANTIBODIES ON INFECTIOUS BURSAL DISEASE AND ACTIVE IMMUNIZATION.

In a series of experiments, some already published, the interaction of passive immunity and infectious bursal disease (IBD) was studied.

Adult hens revaccinated with a killed-virus multiple-emulsion responded with high levels of antibodies that remained stable for the 50-week experimental period. Chicks obtained from these hens were fully protected against bursal atrophy when challenged with IBD virus (IBDV) in the first four weeks of life. Protection of chicks derived from hens revaccinated with killed-virus suspension, or unrevaccinated was partial at all times of challenge.

Passive immunity interferred with the response to killed-virus suspension or killed-virus emulsion, or with a live-modified IBDV given through the intramuscular or intrabursal routes.

Inoculation with wild IBDV did not result in bursal atrophy, nor immunosuppression in chicks with high antibody levels, and elicited a lower antibody response than in susceptible chickens. Furthermore, in this experiment it was found that passive antibodies protected the chick against immunosuppression during the first four weeks of age, two weeks after protection against bursal atrophy had disappeared.

[a] Parte de la tesis doctoral del autor principal.
Part of the senior author's Ph. D. thesis.

EFECTO DE LOS ANTICUERPOS PASIVOS SOBRE LA INFECCION DE LA BOLSA DE FABRICIO Y SU INMUNIZACION ACTIVA

Benjamín Lucio - Martínez.- Departamento de Producción Animal:Aves Facultad de Medicina Veterinaria y Zootecnia Universidad Nacional Autónoma de México. México 20, D.F.

Stephen B.Hitchner. Department of Avian and Aquatic Animal Medicine. New York State College of Veterinary Medicine. Cornell University. Ithaca, N.Y. 14853.

La inducción de una inmunidad adecuada contra la infección de la bolsa de Fabricio (IBF) es difícil debido a que la gran mayoría de los pollitos comerciales poseen anticuerpos maternos contra el virus de la IBF (VIBF).

Estos anticuerpos maternos protegen al pollito contra el VIBF y contra la inmunodepresión que él induce durante las primeras semanas de vida (1,5,9,14). Por desgracia,los anticuerpos maternos también interfieren con las vacunas elaboradas con virus atenuados (1,4,8,10,13,15).El problema se complica aún más debido a que los niveles de anticuerpos maternos varian de una parvada a otra y aún entre pollitos de la misma parvada (2).

Estos estudios,algunos de ellos ya publicados (6,7),se llevaron a cabo para determinar:(A) si era posible inducir niveles elevados de anticuerpos en gallinas adultas (B) cuál era su efecto sobre la atrofia de la bolsa de Fabricio inducida por el VIBF (C) el efecto de los anticuerpos pasivos sobre la inmunización activa inducida por el VIBF vivo virulento,atenuado el VIBF inactivado y (D) el efecto de los anticuerpos pasivos sobre la protección a la inmunodepresión y sobre la respuesta inmune producida por una cepa virulenta.

En la primera fase,en la que se estudió la respuesta de las gallinas adultas a la vacunación y la protección transmitida a la progenie,se utilizaron: un lote de gallinas libres de anticuerpos y otro lote vacunado a las dos semanas de edad con virus vivo modificado. A las 16 semanas de edad las gallinas se vacunaron con virus muerto en suspensión o en emulsión y se alojaron en un gallinero comercial junto con otras gallinas de diversas edades y procedencias.

Durante todo el periodo experimental las gallinas susceptibles permanecieron esencialmente negativas a anticuerpos neutralizantes contra VIBF.La respuesta de las gallinas susceptibles vacunadas con virus muerto fue pobre,durante todo el periodo experimental sus títulos de anticuerpos neutralizantes fueron de 10^2 (expresado como el logaritmo inverso de la más alta dilución del antisuero capaz de neutralizar 100 dosis infectantes para los cultivos celulares),mientras que los títulos de anticuerpos neturalizantes de las gallinas susceptibles vacunadas con virus muerto en emulsión eran de aproximadamente 10^4.

Los títulos de las gallinas inmunes no revacunadas se mantuvieron durante todo el experimento alrededor de 10^4, siendo el título de las gallinas revacunadas con virus muerto en suspensión sólo ligeramente más alto.Por otro lado, la respuesta de las gallinas inmunes revacunadas con virus muerto en emulsión,aún cuando fue más lenta,mantuvo un título de alrededor de 10^7 durante las 50 semanas que duró el experimento.

Para determinar la protección conferida a la progenie,veinticinco semanas después de la vacunación se obtuvieron huevos de estas gallinas.Los anticuerpos contra el VIBF presentes en la yema y en el suero de los pollitos se titularon y se encontró una estrecha correlación entre ellos y los títulos de anticuerpos en el suero de la madre.

Con los pollitos obtenidos se formaron grupos que fueron desafiados de la la. a la 6a.semana de edad.Ningún pollito descendiente de gallinas revacunadas con virus muerto emulsionado sufrió atrofia de la bolsa de Fabricio al ser -

desafiado antes de la 5a. semana de vida y a las 5 semanas de edad sólo el 60% sufrió de atrofia de la bolsa.En contrasta, en los grupos de pollitos - obtenidos de gallinas revacunadas con virus en suspensión hubo entre un 20 y un 40% de animales susceptibles de la 1a. a la 4a.semana de edad, y en la 5a. hubo un 80%. Los pollitos provenientes de gallinas no revacunadas mostraron un 60% de resistencia sólo en el desafio de la primera semana y 25% en la 2a., de ahí en adelante siermpre resultaron susceptibles a la atrofia de la bolsa de Fabricio.

El efecto de los anticuerpos pasivos sobre la inmunización activa inducida po el VIBF se estudió utilizando virus muerto en suspensión o en emulsión en pollitos con o sin anticuerpos pasivos. Se encontró que una sola aplicación de virus muerto en suspensión no indujo inmunidad en pollitos con o sin anticuerpos pasivos. La vacuna de virus muerto en emulsión indujo resistencia a la atrofia de la bolsa de Fabricio en pollitos sin anticuerpos pasivos cuatro semanas después de su aplicación. En los pollitos con anticuerpos la resistencia inducida fue muy irregular.

En las segunda parte de esta investigación se usó virus vivo atenuado,aplicado por vía ocular,intramuscular o intrabursal.La aplicación ocular de este virus no indujo inmunidad en pollitos con o sin anticuerpos pasivos.Por otro lado,su aplicación intramuscular o intrabursal indujo protección a la bolsa de Fabricio en pollitos sin anticuerpos pero no en pollitos con anticuerpos pasivos.

En vista de estos resultados y de que en los inicios de la inmunización contra la IBF se utilizó el virus vivo virulento a temprana edad, con aparentes buenos resultados (2,3,11),se decidió hacer una prueba preliminar en la que se infectaron pollitos con o sin anticuerpos pasivos con VIBF virulento. Los pollitos inmunes desarrollaron títulos de anticuerpos inferiores a los de -- los pollitos susceptibles,pero no sufrieron de atrofia de la bolsa de Fabricio.

En la siguiente fase experimental se estudió la interacción de la edad y de los anticuerpos pasivos sobre la inmunodepresión causada por cepas más virulentas.Para ello se inocularon grupos de pollitos con o sin anticuerpos maternos a diferentes edades con "Bursa-Vac" (Sterwin Laboratories, Millsboro, Delaware) o con un virus de campo.Los resultados obtenidos con ambos virus - fueron practicamente iguales.En los pollitos sin anticuerpos el VIBF indujo inmunodepresión,evidente durante la dos primeras semanas de vida en la respuesta a la albúmina de suero bovino y durante 4 semanas en la respuesta a <u>Salmonella pullorum</u>.

Los anticuerpos pasivos protegieron de la atrofia de la bolsa de Fabricio - en el desafio de los 7 a los 14 días de edad.La protección a la inmunodepresión continuó hasta los 28 días de edad.

Los hallazgos más importantes de estos estudios fueron que:(a) es posible inducir títulos de anticuerpos elevados y uniformes en gallinas adultas utilizando virus muerto en emulsión (b) los pollitos con anticuerpos pasivos pueden responder en forma activa a las cepas virulentas del VIBF sin sufrir --- atrofia de la bolsa de Fabricio; (c)los niveles elevados de anticuerpos pasivos bloquean la respuesta inmune al VIBF vivo virulento,vivo modificado o - inactivado; (d) hay niveles en los que los anticuerpos pasivos permiten una respuesta activa al VIBF virulento sin que haya atrofia de la bolsa de Fabricio;(e) hay un punto en el que los anticuerpos pasivos previenen la inmunodepresión,aún cuando no protejan de la atrofia de la bolsa de Fabricio y (f)la edad a la que el pollito ya no sufre de inmunodepresión varía de acuerdo con el antígeno al que está respondiendo.

En base a los resultados obtenidos se puede pensar que la infección de la -- bolsa de Fabricio puede ser prevenida mediante una combinación de inmunidad pasiva e inmunización activa.Los niveles de anticuerpos elevados y uniformes, logrados por la aplicación de virus muerto emulsionado en las reproductoras, protegerian al pollito de la inmunodepresión durante las primeras semanas de edad.Una vez que el peligro de la inmunodepresión hubiera pasado una vacuna de virulencia media que protegería al pollito por el resto de su vida comercial.

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FIELD TRIALS WITH INACTIVATED INFECTIOUS BURSAL DISEASE VIRUS VACCINE

R. Ramirez, D.V.M.
Vineland Laboratories (Mexico), S.A. de C.V.
Queretaro, Qro. Mexico

SUMMARY

Several workers have shown that if chickens can be protected with maternal antibody for at least 2 weeks following hatching, the immunosuppressive effects of the IBDV can be minimized. Effective programs of live vaccination of broiler chicks are difficult to manage because of the varying levels of maternal antibody and immunity conferred by parents to their progeny which will interfere with vaccination.

Most broiler breeders in the United States are serologically positive to the IBDV prior to the onset of production as a result of field exposure or live virus vaccination. This immunity is effectively transferred to progeny. - However, as the breeder flock ages there is an almost linear decrease in the IBDV antibody titers with an increase in age. This results in decreased ---- titers hence a loss of protection at a time critical in terms of the development of the immune system.

This report describes experiences we have had in field trials with an inactivated oil emulsion IBDV vaccine for use in broiler breeders.

Infectious Bursal Disease Virus neutralization titers in breeders and their progeny were significantly higher in inactivated IBDV vaccinated birds than in nonvaccinates for the duration of the study. Progeny from breeder flocks vaccinated with inactivated IBDV generally perform better than progeny from non-vaccinated flocks. This is particularly true when these birds are reared on "problem" broiler farms. Progeny from breeder flocks vaccinated with inactivated IBDV demonstrated protection against high-virulent artificial challenge.

Despite positive AGP tests in all chicks the SN titers of chicks from vaccinated dams ranged from a low of 1:16 to 1:512, with most above 1:32; the controls ranged from <1:16 to 1:64 with most at 1:32 or lower.

RESUMEN

Varios investigadores han mostrado que si los pollitos pueden ser protegidos con anticuerpos maternos por lo menos las dos primeras semanas de vida, los efectos inmunosupresores del virus de la Infección de la Bolsa pueden minimizarse. Programas efectivos con vacunas a virus vivo, en pollo de engorda son difíciles de alcanzar, debido a la variación en los niveles de anticuerpos - maternales y a la inmunidad transmitida por las madres a su progenie, la cual interfiere con los programas de vacunación. La mayoría de las reproductoras pesadas son serológicamente positivas al virus de la Infección de la Bolsa antes de alcanzar su máxima producción con resultado de una exposición de -- campo y de una vacunación con virus vivo. Esta inmunidad le es transmitida - efectivamente a la progenie. Sin embargo, conforme la edad de las gallinas - va aumentando, existe una disminución lineal en los títulos de los anticuerpos. Esto resulta en una pérdida de protección en la fase crítica del desarrollo del sistema inmunocompetente. Este reporte describe algunas de las -- experiencias que tenemos de las pruebas de campo con una vacuna inactivada y emulsionada en aceite, para utilizarse en reproductoras. Los títulos de suero neutralización en las reproductoras, y en su progenie fueron significativamente más altos en las aves vacunadas que en las aves no vacunadas durante el tiempo que duró la prueba. La progenie procedente de reproductoras vacunadas con la vacuna inactivada emulsionada se comportó dando menores resultados que la progenie procedente de reproductoras no vacunadas, especialmente cuando estas aves eran alojadas en granjas problema. La progenie de aves - vacunadas con la vacuna emulsionada demostró protección contra el desafío -- con un virus virulento.

PRUEBAS CON UNA VACUNA COMERCIAL INACTIVADA Y EMULSIONADA DE VIRUS DE LA INFECCION DE LA BURSA

R. Ramirez, M.V.Z.

La incidencia de infecciones clínicas de la Enfermedad de la Bursa de Fabricius puede ser baja, pero serológicamente hay evidencia de que las infecciones subclínicas están ampliamente defundidas y son comunes en la mayoría de las explotaciones avícolas.

La infección clínica (Enfermedad clásica), causa pérdidas económicas directas, aumentando la mortandad y afectando la productividad. Sin embargo, la interacción del virus de la infección de la Bursa con otras infecciones es probablemente de mayor importancia. Ha sido demostrado per varios investigadores que el virus de la Infección de la Bursa, cuando es inoculado a edad temprana de los pollitos puede dañar la respuesta inmunológica e incrementar la susceptibilidad de estas aves hacia agentes infecciosos, como virus de la Enfermedad de Newcastle, de Bronquitis Infecciosa, *Salmonella*, *e. coli*, Enfermedad de Marek y *Mycoplasma*, etc.

Varios investigadores han mostrado que si los pollitos pueden ser protegidos con anticuerpos maternales por lo menos las dos primeras semanas de vida, los efectos inmunosupresores de Virus de la Infección de la Bursa pueden minimizarse. Progresos efectivos con vacunas a virus vivo, en pollo de engorda son difíciles de alcanzar, debido a la variación en los niveles de anticuerpos maternales y la inmunidad trasmitida por los padres a su progenie la cual interfiere con los programas de vacunación.

La mayoría de las reproductoras pesadas son serologicamente positivas al virus de la Infección de la Bursa antes de alcanzar su máxima producción, como resultado de una exposición de campo o a una vacunación con virus vivo. Esta inmunidad le es transmitida efectivamente a la progenie. Sin embargo, conforme a la edad de las gallinas va aumentando, existe una disminución linear en los títulos de los anticuerpos contra el virus.

Teóricamente aumentando por medio de un "Booster" los anticuerpos en las reproductoras, debería ser posible aumentar y prolongar la protección dada a la progenie por medio de los anticuerpos maternales. Pero las vacunas a virus vivo disponibles en el presente han demostrado no ser útiles como "Boosters" en las reproductoras para aumentar los títulos de anticuerpos contra el virus de la Infección de la Bursa. Esto se debe probablemente a que la susceptibilidad disminuye con la edad, se pierden las células susceptibles o por la inmunidad conferida en la primera vacunación. Un producto inactivado administrado por vía subcutánea producirá una respuesta secundaria activa, que resulta en altos títulos específicos tanto en los padres como en la progenie.

Este reporte describe algunas de las experienceias que tenemos de las pruebas de campo con una vacuna inactivada y emulsionada en aceite contra la Infección de la Bursa, para utilizarse en reproductoras.

El propósito de la primera prueba fue establecer la eficacia de la vacuna con virus inactivado para inmunizar reproductoras pesadas. Se utilizaron aves pertenecientes a dos compañías integradas a las que designaremos A y B.

Todas las aves recibieron 0.5 ml de vacuna en la porción media del cuello subcutánea. Se obtuvieron sueros y se les hicieron las pruebas de precipitación en agar gel y suero neutralización. Los resultados se exponen en el cuadro.

Los datos de los cuadros anteriores nos indican que la vacuna es un agente efectivo para inmunizar las reproductoras pesadas. Aún en las aves que tenían inmunidad se nota que ésta aumenta con la vacuna.

Se seleccionaron huevos de las parvadas vacunadas, se incubaron y los pollitos fueron sangrados al primer día de edad y a los sueros así obtenidos se les hicieron las mismas pruebas que al suero de las madres.

Por último se obtuvieron pollitos de las aves vacunadas con la vacuna inactivada y se desafiaron con un virus de campo. Los resultados de esta prueba los tenemos en el cuadro. El cuadro nos muestran los resultados del desafío en pollitos procedentes de madres no vacunadas con la vacuna emulsionada y de pollitos libres de patógenos específicos.

Con estos datos podemos obtener las siguientes conclusiones.

Los títulos de suero neutralización fueron significativamente más altos en las reproductoras vacunadas con la Vacuna Inactivada Emulsionada y su progenie que en las reproductoras no vacunadas y su progenie, hasta la edad que se hizo la prueba.

La progenie de reproductoras pesadas, vacunadas con la Vacuna Inactivada Emulsionada demostró protección cuando fue desafiada con un virus de campo de alta virulencia.

Las parvadas de pollo de engorda procedentes de reproductoras vacunadas se comportan mejor que las parvadas procedentes de reproductoras no vacunadas con la Vacuna Emulsionada.

TODAS LAS PRUEBAS FUERON SUPERVISADAS POR EL DR. JOHN K. ROSENBERGER PROFESOR Y PRESIDENTE DEL DEPTO. DE CIENCIA ANIMAL Y BIOQUIMICA AGRICULA DE LA UNIVERSIDAD DE DELAWARE, NEWARK, DELAWARE 19711.

CUADRO 1. VACUNA VIBF INACTIVADA

Prueba en Reproductoras Pesadas

Sumario de Todas las Parvadas de las Pruebas de Sueroneutralizacion

Parvadas	Clasificacion	Tiempo de Muestreo	Promedio de los Titulos de Sueroneutralizacion
A-1,2,3,4	Controles	Prevac. (24 Sem.)	66.0
	"	Postvac. (30 Sem.)	53.0
	Vacunadas	Prevac. (25 Sem.)	54.4
	"	Postvac. (30 Sem.)	578.6
B-1	Vacunadas	Prevac. (18 Sem.)	66.9
	"	Postvac. (26 Sem.)	263.3
	"	Postvac. (39 Sem.)	282.9
B-2	Vacunadas	Prevac. (18 Sem.)	81.2
	"	Postvac. (23 Sem.)	335.2
	"	Postvac. (38 Sem.)	488.7

CUADRO 2. Efectos en pollitos procedente de reproductoras, con la vacuna, sin vacuna, y SPF despues del Desafio a varios edades.

	Con des de Desafio (15 grupo)			Controles no Desafioda (5 grupo)		
	1 Dia	7 Dias	14 Dias	1 Dia	7 Dias	14 Dias
Con la vacuna emulsionada inactivada						
Peso Corporal	138.9	354.6	450.4	162.0	310.6	399.6
Peso de la Bolsa	0.354	1.03	1.22	0.37	1.05	1.46
Relation Peso Bursal/ Peso Corporal x 1000	2.59	2.85	2.79	2.27	3.41	3.64
Resistance al Desafio	15/15	12/15	9/15	-	-	-
Sin la vacuna emulsionada inactivida						
Peso Corporal	134.4	310.79	457.1	149.3	361.4	435.0
Peso de la Bolsa	0.31	0.8	0.54	0.29	0.99	1.27
Relation Peso Bursal/ Peso Corporal x 1000	2.28	2.5	1.09	1.925	2.73	2.84
Resistance al Desafio	15/15	8/15	1/15	-	-	-
S.P.F.						
Peso Corporal	62.02	93.2	116.46	64.96	119.9	203.2
Peso la Bolsa	0.079	0.1	0.244	0.16	0.43	0.955
Relation Peso Bursal/ Peso Corporal x 1000	1.24	1.09	2.12	2.50	3.52	4.62
Resistance al Desafio	0/15	0/15	1/15	-	-	-

ANALISIS COMPARATIVO DE LA RESPUESTA INMUNOLOGICA INDUCIDA POR TRES VACUNAS COMERCIALES CONTRA LA INFECCION DE LA BOLSA DE FABRICIO

*Bernardo Lozano Dubernard, M.V.Z. y *Armando Antillon Rionda, M.V.Z., M.S., Ph.D. y
**Elsie M. Ruíz Ruíz, M.V.Z.

*Investigación y Desarrollo Industrial, S.A., 7 Norte 356, Tehuacan, Pue. México

**Departamento de Producción Animal: Aves. Facultad de Medicina Veterinaria y Zootecnia. Universidad Nacional Autónoma de México. México 20, D. F.

Una Parvada de 30,000 pollitas de reemplazo White Leghorn fué dividida en tres lotes y vacunada a los 15 dias de edad en el agua de bebida con tres distintas vacunas comerciales contra la IBF, con el objeto de medir su patogenicidad e inmunogenicidad.

El grado de patogenicidad fué medido a través de la observación clínica, exámen-post mortem, pesos de bolsas de Fabricio y timos y estudio histológico de las bolsas de Fabricio. La inmunogenicidad se valoró en base a pruebas de inmunodifusión en agar -- (IDA), virus suero neutralización (VSN) y por la resistencia mostrada al desafío.

Los resultados demonstraron cierto grado de variación en la patogenicidad de las-vacunas. Además, fueron buenas inductoras de anticuerpos neutralizantes y pobres productoras de anticuerpos precipitantes. En la prueba del desafío, se encontraron diversos grados de protección conferido por las vacunas.

Se discuten brevemente las características de un plan do control contra la IBF.

COMPARATIVE ANALYSIS OF THE IMMUNE RESPONSE INDUCED AGAINST INFECTIOUS BURSAL DISEASE

A Flock of 30,000 White Leghorn replacement birds was divided into three groups. Each group was vaccinated at 15 days of age via the drinking water with three different IBDV-commercial vaccines.

The degree of pathogenicity was measured by clinical observation, post mortem-examination, the weights of bursae and thymuses, and histologic study of the bursa-of Fabricius. The rate of immunity was measured by the AGP-test, virus neutralization (VN) and challenge tests.

Some degree of variation in the pathogenicity of the vaccines was observed. A high rate of neutralizing antibodies and a poor production of precipitins was also noted. All three vaccines conferred good protection in the challenge test.

A vaccination control program is briefly discussed.

INTRODUCCION

La Infección de la Bolsa de Fabricio (IBF), es una enfermedad viral en la cual se - destruyen las células linfáticas de la bolsa de Fabricio, timo, bazo y otros agregados - linfáticos (6), produciendo daños severos al sistema inmunológico (4). Los estudios serológicos indican que se encuentra muy difundida en las principales areas avícolas del mundo (8), y una vez que se presenta en una explotación avícola, es muy difícil erradicarla (10), por lo que la única práctica viable para controlarla, es por métodos de vacunación (6).

Es deseable que una cepa vacunal no cause daño en la bolsa de Fabricio, aunque se administre a pollitos de un dia de un dia de edad sin anticuerpos maternos; no debe reducir la habilidad de los pollos para responder al estímulo de otras vacunaciones y, aunque sea aplicada a pollitos de corta edad, les debe inducir una sólida y contínua protección (2).

Actualmente se han hecho intentos para obtener cepas vacunales efectivas, sin embargo se reportan diferencias en cuanto a su patogenicidad e inmunogenicidad (9). En México se están utilizando vacunas contra la IBF a base de las cepas Lukert y PBG-98 principal-- mente, bajo diferentes nombres comerciales. Sin embargo, no existen trabajos controlados sobre sus comportamientos a nivel de campo.

Para establecer un calendario de vacunación contra la IBF, es necesario conocer el estado inmunológico de las parvadas, de lo contrario, la protección esperada de las vacunas será muy irregular. En México, algunos veterinarios y avicultores vacunan contra ésta enfermedad en forma rutinaria a las dos semanas de edad; por ello en el presente expemento se trató de replicar las mismas condiciones del campo.

El objetivo del presente experimento consistió en analizar la patogenicidad e inmunogenicidad de tres vacunas comerciales contra la Infección de la Bolsa de Fabricio, bajo condiciones de campo.

MATERIAL Y METODO

El experimento se llevó a cabo durante 8 semanas en una granja comercial de pollitas de reemplazo de crianza en piso, ubicada en el valle de Tehuacan, Puebla. Una parvada de 30,000 pollitas White Leghorn, fué dividida en tres lotes de 10,000 aves cada uno; los lotes se denominaron A, B y C, aplicando a cada lote una vacuna comercial contra la IBF, de marca diferente; además, se dejó un lote testigo no vacunado de 100 gallitos, los cuales fueron alojados en unidades Horsfall-Bauer de aislamiento, desde el primer dia de edad.

El calendario de vacunación fué similar para los tres lotes experimentales; el lote testigo no vacunado, no recibió ningún tipo de vacunación. El lote A fué vacunado con la cepa Lukert; el lote B, se vacunó con la cepa PBG-98, y al lote C, se le aplicó la cepa Lukert. Las aves fueron vacunadas a los quince dias de edad. Las vacunas, previa titulación en embrión de pollo, fueron aplicadas en el agua de bebida para medir y comparar su patogenicidad e inmunogenicidad.

Se procedió a medir la patogenicidad de los virus vacunales en las aves, separando 4 gallitos de cada lote, antes de la vacunación y durante los siete dias postvacunales; se anotaron los signos clínicos, se pesaron las aves y finalmente fueron sacrificadas. Se anotaron los cambios a la necropsia, se obtuvo el timo y la bolsa de Fabricio los que fueron pesados y finalmente, la bolsa fue fijada en formol para su análisis histológico, haciendo una descripción de las lesiones siguiendo los lineamientos descritos por Winterfield y Thacker (9).

La inmunidad conferida por las cepas vacunales, se midió en base a pruebas serológicas y por la resistencia mostrada al desafío. Las pruebas serológicas realizadas, consistieron en inmunodifusión en agar (IDA), y virus suero neutralización (VSN), para lo cual se tomaron muestras de suero los dias 15, 30, 45 y 60 de edad; los títulos de virus suero neutralización menores de 1 log. 10 se consideraron negativos. El desafío se llevó a cabo para los cuatro lotes, a las 6 semanas de edad, en las unidades de aislamiento, con una cepa patógena de IBF, cuyo título era de $10^{6.7}$ DIE 50%/ml; los animales fueron observados clínicamente todos los dias, sacrificandose 3 aves de cada grupo los dias 1, 2, 3, 5, 9 y 16 post desafío; se registró el peso corporal, anotándose los cambios a la necropsia; se obtuvo la bolsa de Fabricio y el timo, los cuales fueron pesados y, finalmente la bolsa fue fijada en formol para su análisis histológico. Además, con la información de peso corporal, peso de timo y bolsa de Fabricio, se realizaron análisis de regresión y con los títulos de neutralización viral, se realizó un análisis de varianza aleatorio (7).

RESULTADOS

Al evaluar la patogenicidad de las cepas vacunales, no se observaron signos clínicos ni lesiones a la necropsia en ninguno de los cuatro lotes experimentales. En el análisis de regresión, de la proporción de la bolsa de Fabricio en cuanto al peso corporal; solamente se encontraron diferencias significativas ($P < 0.05$), cuando se comparó el lote testigo con los lotes A y C.

En el análisis histológico de la bolsa de Fabricio, en el lote testigo no hubo alteraciones patológicas durante los 7 dias post vacunación. En el lote A, se observaron lesiones que iban de mínimas a leves, durante la mayoría de los 7 dias post vacunación en el 64% de las bolsas. En el lote B, las lesiones observadas fueron mínimas en el 17% de las bolsas, y en el lote C se observaron lesiones mínimas en el 42% de las bolsas. Lo anterior indica que la vacuna A poseía un mayor grado de patogenicidad a la bolsa de Fabricio cuando se comparó con las vacunas B y C.

Al analizar la inmunogenicidad de las vacunas por la prueba de IDA, la presencia de sueros positivos a anticuerpos precipitantes fué mínima, no existiendo entre los lotes A B y C diferencia alguna. En las pruebas de VSN se obtuvo la media geométrica de los títulos de virus neutralizado durante todo el experimento, siendo de 1.56 para el lote A, de 1.89 para el lote B y de 2.10 para el lote C. Estadísticamente, sólo se encontró diferencia significativa ($P < 0.05$), cuando se compararon los títulos entre los lotes A y C.

Después de la prueba de desafío, en el lote testigo no vacunado, se encontraron signos clínicos de IBF en más del 70% de los animales del dia 3 al 5 post desafío, desapareciendo éstos a partir del dia 9 post desafío. En los lotes vacunados, la morbilidad varió entre un 35 a 45%, sin embargo, los signos clínicos tuvieron menor intensidad. A la necropsia, en el lote testigo desafiado, se observaron lesiones características de IBF -- del dia 3 al 5 post desafío, desapareciendo del noveno dia post desafío en adelante, con excepción de la esperada atrofia de las bolsas de Fabricio. En los tres lotes vacunados, se encontraron ligeras hemorragias en musculos, timos y grasa coronaria, además de bolsas de Fabricio aumentadas o disminuídas; sin embargo, éstos hallazgos no fueron contundentes para hacer un diagnótico positivo de IBF, teniéndose que recurrir al exámen histológico.

En el análisis de regresión del porcentaje de la bolsa de Fabricio, en relación al peso corporal, se encontró una diferencia altamente significativa ($P < 0.01$) al comparar el lote testigo, con los tres lotes vacunados; al comparar entre sí los tres lotes, solamente se encontró una diferencia significativa cuando se compararon el lote A con el C.

En el análisis histológico de la bolsa de Fabricio de los animales desafiados, se encontraron lesiones características en los cuatro lotes experimentales. En el lote testigo desafiado, más del 70% resultaron positivos histológicamente a la infección. En el lote A, aproximadamente el 55% de las bolsas resultaron positivas; en el lote B, un 28% fueron positivas, y en el lote C, un 40 por ciento mostraron lesiones de IBF.

DISCUSION Y CONCLUSIONES

El lote testigo no vacunado fue sangrado a los 45 dias de edad, para comprobar que las aves no estuvieron en contacto con el virus de la IBF. Al hacer el análisis histológico de las bolsas de Fabricio de los tres lotes vacunados, se encontró cierto grado de patogenicidad cuyo rango iba de mínimo a leve; éstos resultados indican que las vacunas utilizadas en el experimento son poco patógenas para la bolsa de Fabricio, pero de ninguna manera apatógenas.

Información reciente indica que las parvadas libres de anticuerpos maternos al ser vacunadas contra la IBF, adquieren una sólida protección contra los virus patógenos (1). Sin embargo, a nivel comercial, dificilmente se encuentra una parvada libre de anticuerpos, ya que un elevado porcentaje de ellas han estado en contacto con virus vacunales o de campo (3). Por ello, el éxito de un programa de vacunación en edad temprana, en parvadas con inmunidad materna, es limitado, ya que el virus vacunal es fácilmente neutralizado por los anticuerpos maternos (8).

Haciendo un análisis histopatológico comparativo del porcentaje de animales que resultaron positivos a la infección, se observa que los animales del lote B, resisitieron mejor al desafío, sin embargo, éstos resultados no son determinantes, ya que se sacrificaron únicamente tres animales por dia, encontrándose en ocasiones una bolsa normal o bolsas atrofiadas; lo anterior es indicativo de que las vacunas no mostraron un grado de protección adecuado. Esto podría ser explicado en base a una inmunidad materna muy heterogénea, ya que en el caso de bolsas normales, probablemente se trató de animales con muy baja o nula inmunidad materna, habiendo quedado protegidos por la vacuna; caso contrario, las bolsas que mostraron lesiones de IBF o atrofia, probablemente provinieron de aves con sólida inmunidad, por lo cual, al ser neutralizado el virus vacunal por los anticuerpos maternos, las aves quedaron desprotegidas inmunológicamente. De lo anterior se desprende la importancia de tener parvadas con inmunidad materna homogénea.

La vacuna A, fue la que mayor grado de patogenicidad mostró, la que indujo niveles más pobres de anticuerpos neutralizantes y además, fue la que menos protegió durante la prueba de desafío. La vacuna B, junto con la C, fueron las menos patógenas, y las que mejor título de anticuerpos neutralizantes alcanzaron.

Para establecer un calendario de vacunación contra la IBF, sería recomendable que se contara en forma rutinaria con una prueba de laboratorio, como la VSN, para conocer el estado inmunológico de la parvada, y además, que éstas tuvieran títulos altos y homogéneos de anticuerpos maternos que las protegieran durante las primeras cuatro semanas de vida, cuando puede producirse inmunosupresión o inmunodepresión. Posteriormente, podría utilizarce una cepa vacunal de mediana patogenicidad para estimular al sistema inmunocompetente y proteger así, contra una posible infección que causara mortandad y reducción temporal de peso, con las consiguientes pérdidas económicas (5).

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IMMUNOGENICITY AND PATHOGENICITY OF DIFFERENT STRAINS OF INFECTIOUS BURSAL DISEASE VIRUS APPLIED AS VACCINE

S.A. Naqi, D.V.M., Ph.D.
Department of Veterinary Microbiology and Parasitology
Texas A&M University
College Station, Texas 77843

Three infectious bursal disease virus strains, designed as BV, BV-M and LKT which are commonly applied as vaccines, were evaluated in chicks that were specific-pathogen free and free of antibodies to infectious bursal disease virus (IBDV). For comparison, a pathogenic field isolate of the IBDV (FV) was also inoculated into 1-day-old and 3-week-old chicks.

At 5 weeks old, the mean virus-neutralizing (VN) antibody titers of the 3 vaccinated groups did not differ significantly ($P < 0.05$). At 7 weeks old the BV-induced VN titers were significantly higher ($P < 0.05$) than the LKT and the BV-M-induced titers. Differences remained similarly significant ($P < 0.05$) at 9 weeks old. Precipitating antibodies were consistently demonstrable in the serums of BV-vaccinated chickens at 5, 7, and 9 weeks old by the agar-gel precipitin (AGP) test, whereas many serums from the BV-M- and LKT-vaccinated birds had no detectable AGP reaction at 9 weeks old. Field virus exposure at 1 day or three weeks old resulted in high VN titers as well as persistent AGP reaction.

The vaccine virus persisted in the bursa of Fabricius until birds were 9 weeks old in groups receiving LKT and BV vaccines but not in the group given the BV-M vaccine.

Upon challenge with FV at 9 weeks old, the vaccinated birds were protected against bursal atrophy.

None of the vaccine viruses, when used according to the manufacturers' recommendations, or the FV, when given at 3 weeks old, caused significant ($P < 0.05$) immunosuppression.

INMUNOGENICIDAD Y PATOGENICIDAD DE DIFERENTES CEPAS DEL VIRUS DE LA INFECCION DE LA BOLSA DE FABRICIO USADAS COMO VACUNAS.

Tres cepas del virus de la infección de la bolsa de Fabricio (VIBF) designadas BV, BV-M y LKT, comunmente usadas como vacuna, fueron evaluadas en pollitos libres de patógenos específicos y libres de anticuerpos contra -- VIBF. Para comparar se inoculó un aislamiento de campo virulento (FV) en pollitos de 1 dia y de 3 semanas de edad.

A las 5 semanas de edad el promedio del título de anticuerpos virus-neutralizantes (VN) no fue significativamente ($P<0.05$) diferente entre los 3 grupos. A las 7 semanas de edad BV indujo títulos que fueron significativamente ($P<0.05$) mas elevados que los inducidos por BV-M o LKT. Las diferencias fueron similares a las 9 semanas de edad, los anticuerpos precipitantes se encontraron en forma constante a las 5, 7 y 9 semanas de edad en los sueros de aves vacunadas con BV, mientras que muchos sueros de aves vacunadas con BV-M o LKT no tenian anticuerpos precipitantes a las 9 semanas de edad, la exposición al virus de campo al dia o 3 dias de edad dió como resultado elevados títulos de anticuerpos VN y una reacción precipitante persistente.

El virus vacunal persistió en la bolsa de Fabricio hasta que los pollos cumplieron 9 semanas en los grupos que recibieron LKT o BV, pero no en los vacunados con BV-M.

Las aves vacunadas al ser desafiadas con FV a las 9 semanas de edad, -- fueron protegidas de la atrofia bursal.

Ninguno de los virus vacunales, usados de acuerdo a las instrucciones del producto, o el FV administrado a las 3 semanas de edad, causó una inmunodepresión significativa ($P< 0.05$)

Traducción: Cortesía del Dr. Benjamín Lucio Martínez

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Three commercially available infectious bursal disease (IBD) vaccines (BV, BV-M and LKT), were evaluated in White Leghorn-type chicks that were free of antibodies to infectious bursal disease virus (IBDV). The vaccines were used according to the manufacturers suggested protocal. For comparison, a pathogenic field isolate (FV) of IBDV was also inoculated into 1-day-old and 3-week-old chicks. At 7 weeks old all birds in each treatment were vaccinated with Newcastle disease (ND) vaccine to measure any immunosuppression affected by the IBDV strains. At 9 weeks old 10 of 20 birds from each treatment group were isolated and challenged with FV. Five days later, all birds were necropsied. The bursa of Fabricius (BF) from individuals were identified, weighed and subjected to immunofluorescence (IF) studies.

Table 1 shows virus-neutralizing antibody (VN) titer and the results of agar gel precipitin tests at 5,7 and 9 weeks of age.

As reported earlier (1), the more virulent strains (FV and BV) produced higher and more persistent levels of antibody than the less invasine strains (BV-M and LKT). This was particularly obvious in case of AGP test results. A lack of correlation between VN titers and AGP reactions was also observed.

Comparison of unchallenged and challenged birds is presented in table 2.

Table 1. Geometric mean virus-neutralizing antibody titers (GMT-VN) and agar-gel precipitin (AGP) reactions of chickens to 3 vaccine strains and a field strain of infectious bursal disease virus.[A]

Treatment	5 weeks old		7 weeks old		9 weeks old	
	GMT-VN	AGP	GMT-VN	AGP	GMT-VN	AGP
BV	4424.0b	11[B]/11[C]	25380.0a	20/20	10200.0a	20/20
BV-M	3611.0b	11/11	2352.0c	20/20	1122.0d	16/20
LKT	7130.0b	10/12	6152.0b	20/20	2580.0c	2/18
FV-1DA	50950.0a	11/11	46330.0a	19/19	21450.0ab	18/18
FV-3WK	14050.0ab	11/11	47190.0a	19/19	47740.0a	19/19
Control	2.6c	0/18	0.00	0/18	1.5e	0/18

[A]Values followed by different letters differ significantly at $P < 0.05$.

[B]Number of AGP-positive birds. [C]Number of birds tested

The comparison of challenged and unchallenged birds is presented in Table 2.

Table 2. Body-weight changes (BWC), bursal weights (Br.W), and immunofluorescence (IF) of bursa in unchallenged chickens and chickens challenged with infectious bursal disease virus.[A]

Treatment	Unchallenged			Challenged		
	ΔBWC[B] (g)	Br.W(% of body wt. in g)	IF	ΔBWC (g)	Br.W(% of body wt. in g)	IF
BV	64.2a ±13.1[C]	0.23b ±0.04	5/5	5.0bc ±6.9	0.17b ±0.02	9/10
BV-M	- 8.2c ± 3.1	0.44a ±0.03	0/5	17.2b ± 5.1	0.41a ±0.01	1/9
LKT	75.0a ±12.0	0.46a ±0.06	2/5	58.0a ± 6.3	0.40a ±0.03	2/10
FV-1DA	25.0b ± 7.8	0.03c ±0.00	0/5	- 1.4bc ±14.5	0.05 ±0.00	1/9
FV-3Wk	57.2a ±13.0	0.07c ±0.01	2/5	46.0a ± 7.7	0.11bc ±0.06	8/10
Control	60.8a 12.7	0.49a ±0.04	0/5	-16.5c ± 9.4	0.33a ±0.03	10/10

[A]Values followed by different letters differ significantly at $P < 0.05$.

[B]Δ = body weight change in 5-day period.

[C]Standard error of the mean.

Loss of weight in unchallenged BV-M vaccinated birds was attributed to factors other than the experimental treatments. Challenge with the FV did not result in mortality. Therefore, viral pathogenicity and protection against challenge was measured on the basis of atrophy of the BF and reduced weight again following challenge. Table 2 shows that the vaccinated birds were protected against bursal atrophy. The vaccine virus persisted in the BF until birds were 9 weeks old in groups receiving LKT and BV vaccine but not in the group given the BV-M vaccine.

None of the vaccine viruses, when used according to the manufacturers' recommendations or the FV, when given at 3 weeks old, caused significant immunosuppression (Table 3).

Table 3. Newcastle disease virus (NDV) hemagglutination-inhibition (HI) antibody titers in chickens vaccinated with infectious bursal disease virus, exposed to a field strain, and controls.[A]

		No. of birds with HI antibody titers 2 wk following NDV vaccination								
Treatment	No. of birds	0	10	20	40	80	160	320	640	GMT
BV	19	-	3	3	4	4	2	3	-	53.55a
BV-M	20	-	4	8	4	4	-	-	-	24.92a
LKT	18	3	-	3	5	3	4	-	-	29.44a
FV-1DA	18	11	1	-	4	-	1	-	1	4.89b
FV-3WK	19	3	1	3	3	6	2	1	-	26.50a
Control	18	1	1	2	5	5	4	-	-	46.10a

[A]Values followed by different letters differ significantly at $P < 0.05$.

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COMPARACION CLINICA Y SEROLOGICA ENTRE UNA PARVADA DE POLLONAS DE REEMPLAZO VACUNADAS CONTRA LA IBF Y OTRA INFECTADA NATURALMENTE

Elsie Ruíz R., M.V.Z. y Armando Antillon R., M.V.Z., M.S., Ph.D.

Investigación y Desarrollo Industrial, S.A.
7 Norte 356
Tehuacan, Pue., México

Departamento de Producción Animal:Aves
Facultad de Medicina Veterinaria y Zootecnia
Universidad Nacional Autónoma de México
México 20, D. F. México.

En un experimento que comprendió las etapas de crianza y desarrollo, se analizaron la mortandad y desechos, consumo de alimento y respuesta inmunológica contra la ENC, entre una parvada vacunada contra la IBF y otra infectada en forma natural.

No hubo diferencias significativas en la mortandad y desecho, consumo de alimento acumulado y los títulos IH contra la ENC. En el lote infectado naturalmente, se notó una respuesta más uniforme en la presencia de anticuerpos precipitantes - contra la IBF.

A COMPARISON OF CLINICAL AND IMMUNE RESPONSES OF AN IBDV-VACCINATED FLOCK AND A NATURALLY INFECTED FLOCK

An experiment was carried out to analyze mortality and culling rates, feed - consumption and immunologic response to NDV, between a flock of replacement pullets vaccinated against IBDV and a naturally infected flock.

No significant differences in the accumulated feed consumption, mortality and culling rates and HI titers were observed during the 20-week period. The response to the AGP-test against IBDV was more uniform in the naturally infected flock than in the vaccinated group.

INTRODUCCION

La Infección de la Bolsa de Fabricio (IBF) es una enfermedad causada por virus la cual afecta principalmente aves jóvenes produciendo necrosis de órganos linfáticos como la bolsa de Fabricio, el bazo, el timo y las placas linfáticas. Se le conoce en nuestro país desde hace varios años (3), habiendo sido confirmada su elevada incidencia a través de encuestas serológicas en parvadas de reproductoras y aves comerciales (6).

Actualmente, y debido al gran número de parvadas que han estado en contacto con el virus, su presentación clínica pasa muchas veces desapercibida, sin embargo, mediante pruebas serológicas y estudio histopatológico se ha demostrado que las aves sufren la infección en edad temprana.

Cuando se presenta la enfermedad en aves jóvenes, produce una marcada depresión de las células B del sistema retículo endotelial (SRE), precursoras de los anticuerpos. - Las secuelas que deja la IBF se manifiestan en forma de graves repercusiones económicas ya que hay reducción temporal del crecimiento, despigmentación parcial, aumento en la susceptibilidad a numerosas enfermedades (4,9,12,16), y disminución en la respuesta inmunológica humoral ante la presencia de antígenos vacunales (7,13). Además, la mortandad por causas inespecíficas se eleva (1).

Se ha establecido que los títulos de la prueba de IH contra la enfermedad de Newcastle (ENC), disminuyen notablemente debido a la IBF, siendo dicha disminución directamente proporcional a la edad de las aves (15). Se ha visto que los pollos inoculados -

con el virus de la IBF al dia de edad, presentan una respuesta serológica significativamente inferior (15), mientras que al infectar pollos a los 21 dias no se inhibe la respuesta de anticuerpos (14). Pollonas bursectomizadas a los 14 dias de edad presentan respuestas serológicas normales, e inclusive a los 12 dias de edad, la formación de anticuerpos neutralizantes es muy similar a la que presentan las aves controles.

Se ha demostrado desde hace tiempo en el valle de Tehuacan, la presencia de la IBF a traves de pruebas serológicas e histológicas. Sin embargo, no fué sino recientemente que se decidió el uso de vacunas en gran escala, como medida tendiente a eliminar o disminuir las secuelas que se presentan durante la crianza, el desarrollo y la postura, en parvadas que sufren la infección en edad temprana.

Por ello, el objetivo del presente experimento fue hacer un estudio comparativo, durante las etapas de crianza y desarrollo, del comportamiento clínico patológico y la respuesta inmunológica contra la ENC, entre una parvada de pollas de reemplazo vacunada contra la IBF, y la otra parvada infectada en forma natural en edad temprana.

MATERIAL Y METODOS

Para el presente experimento se utilizaron dos granjas comerciales* de pollonas de reemplazo criadas en piso, localizadas en el valle de Tehuacan. La granja A con un total de 40,420 pollitas divididas de la siguiente manera: 13,222 Babcock B-300, 20,934 Babcock B-380 y 6,064 Hi Sex. En la granja B se alojaron 124,084 pollitas Babcock B-300. Tanto las medidas de manejo como el alimento fueron similares para ambas granjas.

En los dos lotes experimentales se llevó a cabo el mismo programa de vacunación, excepción hecha de la vacuna contra la IBF** que fué aplicada únicamente a las aves de la granja B; la vacuna, de marca comercial, tuvo un título de $10^{5.5}$ DLE/50% por dosis, aplicandose por vía subcutánea al primer dia de edad, y en el agua a los 25 dias.

El experimento tuvo una duración de 20 semanas, durante las cuales se llevaron registros del consumo de alimento, mortandad y desechos semanarios, junto con la observación clínica de las parvadas. En ambos grupos, y durante todo el desarrollo experimental, se tomaron al azar 25 muestras de sangre con intervalos aproximados de 10 dias cada uno; se obtuvo el suero corriendose pruebas de IH contra la ENC, utilizando 10 unidades hemoaglutinantes, y según el método de Beard y Wilkes (2). Con los mismos sueros se llevaron a cabo pruebas de inmunodifusión en agar (IDA) usando el método de Hirai y Shimakura (8), para detectar anticuerpos contra el virus de la IBF, cuyo antígeno fué preparado a partir de un aislamiento de campo.

Las aves de la granja A, no vacunada contra la IBF, fueron revisadas constantemente durante las primeras semanas de vida, para conocer por medio del exámen a la necropsia, aislamiento del virus y estudio histopatológico, el momento de infección natural; para el aislamiento del virus se utilizaron embriones de pollo libres de anticuerpos específicos (ALPES)***.

RESULTADOS

Las pruebas de laboratorio indicaron que hubo infeccion natural de IBF a los 35 dias.

El análisis de mortandad y eliminación por retraso corporal de la granja A tuvo los siguientes resultados: las aves B-380 tuvieron el menor porcentaje con un 3.7, mientras que la Hi Sex tuvo un 4.25 y la B-300 un 6.53 por ciento. Al hacer el análisis estadístico se encontró una diferencia significativa ($P < 0.01$) de la B-300 con la Hi Sex y la B-380. Sin embargo, al revisar la mortandad y desecho durante las primeras cuatro semanas de edad, o sea antes del brote de IBF, se obtuvieron los siguientes resultados: la B-380 tuvo un 1.93%, lo cual indica que el 1.1% fué posterior a la infección; la Hi Sex tuvo un 1.33% y un 2.92% fue después de las cuatro semanas. Finalmente, la B-300 tuvo elevada mortandad en las primeras cuatro semanas (4.23), y posteriormente un 2.19. Como puede observarse, el porcentaje más elevado de mortandad y desecho posterior al brote de IBF, correspondió a las aves Hi Sex seguida por la B-300.

Los resultados de eliminación y mortandad de la Granja B a las 20 semanas fueron del 3.3%; al comparar éstos resultados con los de la parvada B-300 de la Granja A, se encontró una diferencia altamente significativa ($P < 0.01$), ya que ésta última tuvo un porcentaje del 6.5. La parvada vacunada contra la IBF tuvo un 1.26% durante las primeras cuatro semanas de vida, y un 2.09% en el periodo restante. Al comparar éstos resultados con los de la estirpe B-300 de la Granja A, no se observaron diferencias significativas.

*Avícola Tehuacan
** Laboratorios Serva, S. A.
***ALPES- Aves Libres de Patógenos Específicos, S.A., Tehuacan, Pue. México

No se encontró diferencia significativa (P>0.05) en el consumo de alimento, entre las aves Hi Sex y B-300 de la Granja A. Tampoco hubo diferencia entre las aves B-300 de ambos grupos.

Al hacer el análisis estadístico de los resultados de las pruebas de IH, entre las aves B-380 y B-300 de la Granja A, se notó que los resultados no eran significativos. Las pruebas de inmunodifusión en agar para detectar anticuerpos contra el virus de la IBF, indicaron una baja progresiva en el % de muestras positivas, los cuales desaparecieron alrededor de los 20 dias para reaparecer a los 40 dias, manteniéndose elevados los niveles de anticuerpos durante todo el experimento.

DISCUSION Y CONCLUSIONES

Los resultados del exámen virológico e histopatológico demostraron la presencia del virus de la IBF en las aves de la Granja A, en la cual se había presentado la infección en parvadas anteriores, lo cual sugiere su naturaleza enzootica.

En las aves del lote A, se notó durante las primeras dos semanas de edad, una rápida disminución en la curva de anticuerpos maternos contra la IBF, hasta desaparecer finalmente al principio de la tercera semana de vida. Esta baja se explica en base a la curva catabólica de los anticuerpos maternos, que señala su vida media (Lukert, 1977). Una vez ocurrida la infección natural, se presentó un rápido aumento en el porcentaje de anticuerpos precipitantes contra la IBF, los cuales perduraron a niveles elevados durante todo el desarrollo experimental. Esto fue debido a que el virus de campo produjo lesiones en la bolsa de Fabricio y que probablemente tenía propiedades inmunogénicas bastante buenas.

Las aves del grupo vacunado mostraron anticuerpos precipitantes contra el virus de la IBF durante las etapas de crianza y desarrollo, sin embargo el porcentaje de sueros positivos fue mas irregular que en el lote infectado en forma natural. Esto puede explicarse porqué nunca se hizo el muestreo serológico de las mismas aves, ya que los anticuerpos precipitantes, igual que los neutralizantes, varían considerablemente de ave a ave (Weisman y Hitchner, 1978). También puede deberse a que la cepa vacunal de IBF utilizada en éste experimento, produce altos niveles de anticuerpos neutralizantes y bajos niveles de anticuerpos precipitantes (10).

El análisis de la mortandad y eliminación por retraso corporal entre las tres estirpes de aves de la Granja A, no vacunada-infectada en forma natural, señala que las aves B-380 tuvieron el porcentaje global más bajo de los grupos experimentales. Esto también fue notado en el periodo comprendido entre la cuarta y vigésima semanas de edad. Aunque no puede confirmarse, estos resultados hacen suponer que existieron diferentes comportamientos entre aves de distintas estirpes.

Al hacer el análisis de la mortandad y desecho durante las 20 semanas, entre las estirpes B-300 de las Granjas A y B, se observó un mayor porcentaje en la parvada infectada naturalmente (6.5%), que en la parvada vacunada (3.3%). Sin embargo, al revisar la mortandad comprendida entre la cuarta y vigésima semanas, no hubo diferencias.

No hubo ninguna diferencia en el consumo de alimento entre las estirpes ligeras del grupo infectado naturalmente, ni entre las aves B-300 de las Granjas A y B. Tampoco se observaron diferencias en los títulos de anticuerpos IH entre las diferentes estirpes de la Granja A, ni entre las dos B-300 de ambos grupos. Es posible que los resultados anteriormente señalados, se hayan debido a que el brote de IBF se presentó alrededor de la quinta semana de edad, en la cual aparentemente ya no hay inmunodepresión (Faragher, 1974; Rosenberger et al., 1976; Rinaldi, 1966).

Por lo anteriormente señalado, se concluye que al comparar un lote de aves vacunadas contra la IBF los dias 1 y 25 de edad, contra otro lote de aves que poseian también una moderada inmunidad materna contra la IBF, y se infectan en forma natural con éste virus a la quinta semana de edad, no se altera la respuesta inmunológica contra la ENC, medida por la prueba de IH; tampoco influye en el consumo de alimento, ni en los promedios de mortandad y desecho. Finalmente, se observa una respuesta más uniforme a la presencia de anticuerpos precipitantes contra el virus de la IBF en las aves infectadas en forma natural, que entre las aves vacunadas.

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EVALUACION DE LA SUSCEPTIBILIDAD DE AVES WHITE LEGHORN AL VIRUS DE LA INFECCION DE LA BOLSA DE FABRICIO (IBF)

R. McMillan, A. Ardans y B. Reynolds

RESUMEN:

En un experimento de campo en pollas White Leghorn fue estudiada la presencia y el efecto de la IBF, en el desarrollo de la bolsa de Fabricio. Se hicieron los siguientes estudios

-Pruebas de inmunofluorescencia en muestras congeladas de bolsas de Febricio.
-Análisis histopatológicos de bolsas de Fabricioñ pruebas de inmunodifusión en agar para detectar anticuerpos contra la IBF.
-Respuesta inmunológicañ vacunación contra ENC, desafío contra ENC, serología contra ENC por medio de pruebas de IH.
-Aislamiento del virus de la IBF.

Se utilizaron dos tipos de aves: pollas comerciales procedentes de reproductoras que habían sido vacunadas durante la etapa de crianza contra la IBF y pollitas SPF libres de anticuerpos contra la IBF. El estudio abarcó el crecimiento y desarrollo de la bolsa de Fabricio en ambos lotes experimentales. Para evaluar la inmunidad pasiva de las pollonas, se utilizó como cepa de desafío, un virus de IBF previamente titulados en embriones de pollo. Los pollitos de ambos grupos fueron desafiados contra la IBF a los 4 y 21 días de edad. Así mismo, a las 3-1'2 semanas de edad, fueron vacunados contra la ENC con la cepa B_1, y desafiados a las 5-1'2 semanas con la cepa Texas GB. Los resultados fueron los siguientes:

1. Cuando se utiliza en el momento adecuado, la prueba de anticuerpos fluorescentes puede ser un buen axiliar para detectar la IBF.

2. El efecto inmunológico causado por la IBF en pollonas durante la fase de vrecimiento es bastante complejo. Tanto las aves desafiadas con IBF a lost 4 y 21 días, como aquellas expuestas en forma natural en el campo, tuvieron una buena protección al desafío contra la cepa GB de enfermedad de Newcastle, dos semanas después de haber sido vacunadas con la cepa B_1. Las aves del grupo control no vacunado mostraron signos clínicos y murieron poco después del desafío.

Traducciómñ Cortesía del Dr. Armando Antillón Rionda

ASSESSING SUSCEPTIBILITY OF WHITE LEGHORN CHICKS TO INFECTIOUS BURSAL DISEASE VIRUS.

SUMMARY:

In a multifaceted field study the presence and effect of IBDV on the developing Bursa of Fabricius in White Leghorn pullets was studied. The evaluating parameters included:

-Application of fluorescence antibody techniques to frozen tissue sections of the Bursa of Fabricius.
-H and E sections of Bursa of Fabricus; IBDV AGP serology.
-Immunological Response Study: NDV vaccination, NDV challenge, NDV serology (H.I.)
-Virus isolation

Two sources of chicks were used in this investigation. Commercial chicks from hens which had been vaccinated during their growing period with IBDV vaccine strain, and SPAFAS chicks from hens certified free from IBDV. The study follows the growth and development of the bursa in the field as well as in experimentally-housed pullets. A laboratory strain of IBDV was titrated in embryos from each source of chicks and then used in a challenge study to evaluate passive protection in the young pullets. Chicks which had been challenged with IBDV at 4 days and 21 days as well as chicks from the field were vaccinated with Newcastle disease virus B_1 vaccination strain at 3-1/2 weeks of age and then challenged with Newcastle disease virus GB strain at 5-1/2 weeks of age. The results of the study indicate that:

1. Fluorescent antibody techniques can be an effective means of detecting IBDV infection if applied at the proper time.

2. The immunological effects of IBDV on growing pullets is complex. Birds which had undergone early (4 days) and late (21 days) laboratory challenge with IBDV as well as birds naturally exposed in the field were protected against NDV-GB challenge 2 weeks after one water vaccination with NDV-B_1 vaccine strain. Whereas unvaccinated controls developed clinical signs and died shortly after challenge.

ASSESSING SUSCEPTIBILITY OF WHITE LEGHORN CHICKS TO INFECTIOUS BURSAL DISEASE VIRUS

Richard McMillan, D.V.M., M.P.V.M., Alex Ardans, D.V.M., Ph.D., Bruce Reynolds
Veterinary Medical Teaching Hospital, School of Veterinary Medicine
University of California, Davis, CA 95616

Introduction

In the fall of 1979 a joint project was undertaken between the Veterinary Medical Teaching Hospital at U.C.Davis and a commercial table egg producer located in the central valley of California.

A number of field observations which had been made during previous months while working closely with this commercial producer's brooding, growing and laying operation had stimulated the study. Included were: (1) Serological monitoring on the brooder ranch strongly indicated the presence of a field strain of Infectious Bursal Disease Virus (IBDV). (2) Clinical disease syndromes were observed in the young growing replacement pullets similar to those described as a sequelae to early IBDV infection. (3) Surveys of antibody response to Newcastle Disease vaccination indicated that pullets at 20 to 24 weeks of age did not have the H.I. titers which would be expected considering the vaccination program used; the range was too great with many low titers.

Trial Design

A. Purpose

1. Evaluate passive protection of chicks to IBDV at 4 days, 11 days, 25 days, and 39 days of age.
2. Evaluate the effects of early IBDV challenge on subsequent vaccination and challenge with Newcastle Disease Virus.
3. Determine the presence and effect of an IBDV field strain (if present) at the commercial brooding facility.

B. Materials and Methods

Egg and Chick Source - Two sources of chicks were used: (1) commercial chicks from hens which had been vaccinated for IBDV at 10 days (1/2 dose) and again at 14 weeks (2X dose) with Bursa-Vac(R), the "hottest" live vaccine licensed, in the drinking water; (2) Specific Pathogen-Free chicks from hens certified free of IBDV. None of the chicks in the study were actively vaccinated for IBDV. Eggs from each of these sources were used also in the embryo susceptibility and virus titration portions of the experiment.

Eggs from the commercial hens were hatched in a commercial hatchery in the San Joaquin Valley and at 1-day of age the majority of chicks were placed on the commercial brood ranch (CF). Approximately 200 were taken directly from the hatchery to an isolated brooding facility at UCD (CC). The SPF (SC) eggs were hatched at the UCD Avian Science hatchery under strict isolation and were placed in another isolated brooding facility at UCD.

Embryo Susceptibility - Eggs from the SPF hens and from the two commercial flocks which supplied the commercial chicks in the study were used to titrate the IBD virus and evaluate embryo susceptibility. This virus originally came to UCD in 1975 from Dr. John Rosenburger of the Universtiy of Delaware. It had since been passed two times in young chickens by eye drop inoculation and subsequent harvesting of the Bursa of Fabricius.

In the Virus Titration/Embryo Susceptibility study, 7-day-old embryonating eggs were inoculated by the yolk sac route.

IBDV Challenge - In the IBDV virus challenge study, commercial chicks and SPF chicks at UCD were challenged with 0.1 ml of a 1:100 dilution of the IBDV virus by eye drop inoculation at 4 days, 11 days, 25 days, and 39 days of age. Three days post-challenge these birds as well as unchallenged controls and commercial field birds from the brooder ranch were assembled, sacrificed after bleeding, and evaluated as to clinical signs, body weight, bursa weight, gross lesions, bursal histology, and serology using indirect fluorescent antibody techniques with frozen sections of the bursas and the AGP technique with IBDV antigen.

NDV Immunity Evlauation - Birds were vaccinated with NDV-B1 strain in the water at 4-weeks of age, and two weeks later these birds as well as unvaccinated controls were challenged with NDV-GB strain.

Immunity was evaluated by observing birds for clinical signs, and testing for antibodies to both NDV and IBDV at 4 weeks old (prevaccination), 6 weeks old (prechallenge-

postvaccination), and at 9 weeks old (postchallenge). Hemagglutination Inhibition (HI) was used to determine NDV titers, and the IBDV AGP test was used to detect the presence of antibodies to IBDV.

Experimental Groupings - In this paper the groups of primary interest are described below:

1. Commercial Field (CF) - These birds were the main target of the investigation. They were compared with the experimentally reared birds in order to highlight as much as possible what was going on at the brood ranch as far as the parameters we were observing.

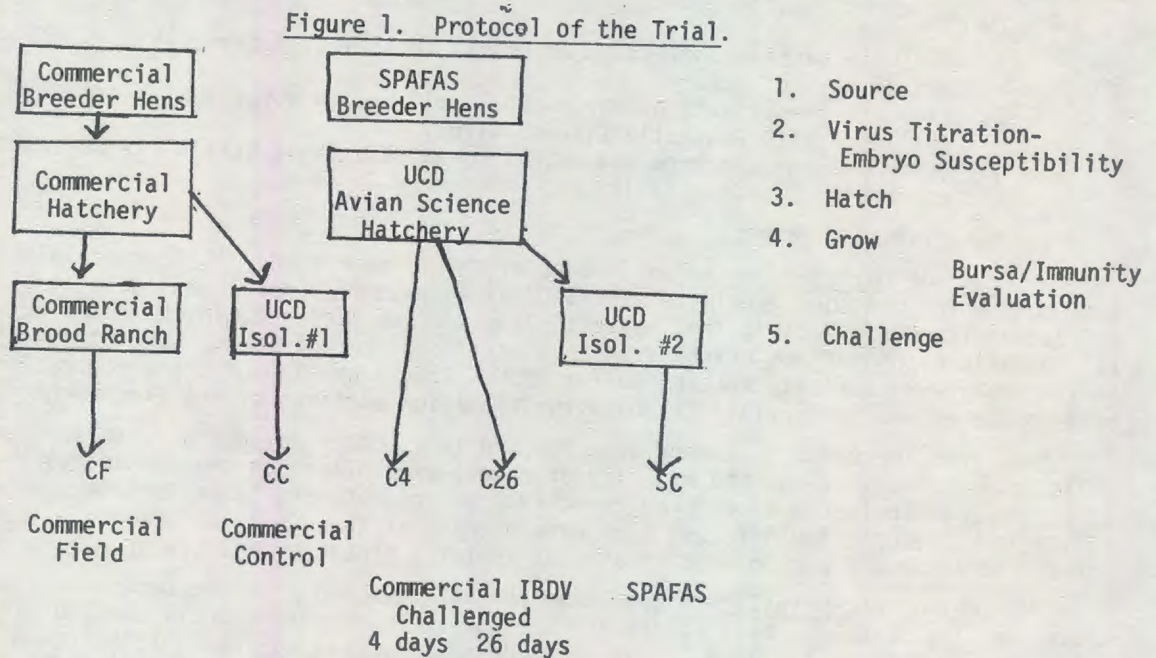
2. Commercial Control (CC) - These birds were from the identical source as the CF birds. They differed in that they were grown in UCD battery brooders and, most importantly, in that they were not exposed at any time to an IBD virus.

3. Commercial IBDV Challenged - These birds are the same as CC birds except that they were challenged at 4 days (C4) or 26 days (C26) of age with IBDV.

4. SPAFAS Control (SC) - These birds represent the totally susceptible population and are used as susceptible controls in the various experiments. They also were reared in battery brooders.

Figure 1 schematically outlines the basic trial design and experimental groupings.

Figure 1. Protocol of the Trial.



Results and Discussion

Table 1 outlines the results of the embryo susceptibility study and established the virus dilution which was used in the IBDV challenge portion of the trial. The relatively low titers could be due to the IBDV strain not being particularly well adapted to eggs. The comparison of LD_{50} titers between the SPF eggs and the commercial eggs (5814-H and 5836-H) indicate a level of protective antibodies present in the yolk sac of the commercial eggs.

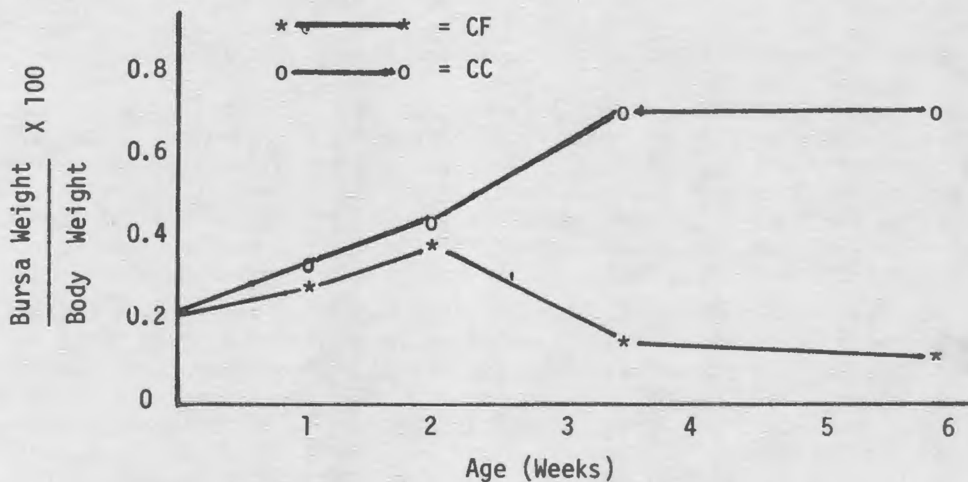
Table 1. Embryo Susceptibility - LD_{50} Determination

Egg Source	Virus Dilution	Dead/Total	LD_{50} Titer
SPAFAS	10^{-1}	8/8	
	10^{-2}	4/8	-2.23
	10^{-3}	2/8	
Com'l Flock #5814	10^{-1}	6/8	
	10^{-2}	1/8	-1.56
	10^{-3}	2/8	
#5836	10^{-1}	7/7	
	10^{-2}	1/7	-1.67
	10^{-3}	1/6	

The effects on body weight and bursa weight are shown for two of the experimental groups in Figure 2. The bursa weight/body weight ratios of the CF and CC groups are depicted graphically for the six-week period. The dramatic effect on the CF (field) birds corresponds to the observed reduction in size of their bursas after two weeks of age.

Figure 2

Bursa Weight/Body Weight Relationship - Commercial Field (CF) versus Commercial Controls (CC) -- IBDV Study



The results of assessing passive immunity by IBDV challenge are summarized in Table 2. Five birds from each group were evaluated at each time.

Table 2. Data Summary - IBDV Field and Experimental Challenge (Bursa Effects)

Age Weeks	Group	Gross Bursal Lesions	Histology	Fluorescent Antibody Positive/Total
	CF	None	Normal	0/5
	CC	None	Normal	0/5
1	C4*	None	Normal	1/5
	S4*	None	Degeneration, necrosis	5/5
	SC	None	Normal	0/5
	CF	None	Normal	0/5
	CC	None	Normal	0/5
2	C11*	None	Normal	1/5
	S11*	Edema, hemorrhage	Degeneration, necrosis	5/5
	SC	None	Normal	0/5
	CF	Pale, small	Interfol. fibroplasia	4/5
	CC	None	Normal	0/5
4	C26*	Edema, hemorrhage	Degeneration, necrosis	3/5
	S26*	Edema, hemorrhage	Degeneration, necrosis	5/5
	SC	None	Normal	0/5
	CF	Small	Fibroplasia, epithelia folding	0/5
	CC	None	Normal	0/5
6	C39*	Edema, hemorrhage	Degeneration, necrosis	3/5
	S39*	Edema, hemorrhage	Degeneration, necrosis	5/5
	SC	None	Normal	0/5

*Number represents age that group was challenged with IBDV.

This data shows that the commercial chicks had passive immunity which moderated the effects of challenge with IBDV as compared with suceptible SPF chicks. However, based on FA test, infection was not prevented. Passive immunity did appear to prevent microscopic damage to the bursas of the comemrcial chicks challenged at UCD up to 14 days of age. Thereafter these birds became increasingly susceptible; these data also suggest that between 2 and 4 weeks of age the commercial field birds at the brooding ranch (CF) became infected and experienced damage to the Bursae of Fabricius by a field IBDV strain.

Immunity Evaluation - Using the NDV described above, an attempt was made to assess the immunological effects of laboratory and of field challenge by IBDV. The results are outlined in Table 3. The commercial birds CC, CF, C4, C26 represent birds which were either unchallenged, field challenged, or experimentally challenged at 4 or 26 days of age with IBDV. SPF (SC) birds were included as controls. The table shows that under the conditions of this trial the IBDV infection had no detectable effect on the resistance to subsequent NDV challenge and that this resistance was determined by whether the group was vaccinated or unvaccinated for NDV.

Immunological competence is a complex matter and it appears that in this case birds challenged with IBDV with only a limited amount of passive protection were later capable of developing adequate immunity to respond to a single vaccination for NDV and resist subsequent challenge 2 weeks or 6 weeks or 2 months later by a virulent NDV strain.

Table 3. Immunological Response Study - NDV Challenge Data Summary

20 Birds per Group I.D./No.	IBDV Challenge	NDV Vacc.	NDV Serology GMT Prevacc.	NDV Serology GMT Postchall. Postvacc.	NDV Serology GMT Postchall.	Clinical Signs
CF	Field	+	0.6	2.7	4.5	None
CC	None	+	0.5	4.9	3.8	None
CC	None	-	0.5	0.7	8.0	CNS, Anorexia, Death
C4	4-day	+	0.3	4.2	3.7	None
C26	26-day	+	0.4	3.5	3.8	None
SC	None	-	0.0	0.0	-	CNS, Anorexia, Death

Vaccination at 3-1/2 weeks of age; challenge at 6 weeks of age.

SIGNIFICANCE OF BURSAL SIZE SURVEY

Douglas R. Kuney, Staff Research Associate, Arthur A. Bickford,
Extension Veterinarian, and Donald D. Bell, Farm Advisor,
University of California, Riverside

In order to determine the normal growth pattern of the bursa of Fabricius in growing single-comb White Leghorn pullets, ten flocks representing five commercial strains were sampled. Body weight, bursal weight, bursal diameter and agar gel precipitin (AGP) reactions to infectious bursal disease virus (IBDV) were recorded throughout the grow period.

Although somewhat variable, the bursal growth pattern, as measured by the diameter method, is characterized by a rapid growth phase from 1 to about 6 weeks of age, followed by an equally rapid regression bottoming out between 8 and 12 weeks. A second growth phase begins at about 12 weeks, peaking between 16 and 20 weeks of age. Measurements beyond 20 weeks indicate a second regression. First peak diameter averages ranged between 1.40 cm and 1.94 cm. Second peak diameters ranged between 0.98 cm and 1.81 cm. Variability in diameters maximized around the two peaks.

Bursal diameter and bursal weight cubed were highly correlated at all ages. Bursal diameters correlated significantly with body weight through 4 weeks of age after which this relationship weakened with age. Diameter patterns from live and already dead (from daily mortality) birds were quite similar; although bursae from dead birds were displaced below those from live birds on the average of 0.19 cm in diameter.

Of the flocks tested for IBDV exposure (using the AGP method), none were vaccinated for IBD; however, all yielded positive reactions on the AGP tests at the time of the first regression phase.

Although data from this study are preliminary only, a consistent pattern of bursal growth among flocks exists. Diameter measurements of the bursa of Fabricius can be made in lieu of weight measurements, thus enabling bursal size determinations at the ranch level. If bursal function is related to bursal size at early ages (1 to 4 weeks), this method holds promise in determining early damage to the immune system.

EL SIGNIFICADO DEL ESTUDIO DEL TAMAÑO DE LA BOLSA DE FABRICIO

Para determinar el modelo normal de crecimiento de la bolsa de Fabricio en los pollos de cresta singular, White Leghorn, diez parvadas que representan 5 clases comerciales se probaron. El peso del cuerpo y de la bolsa, el diametro de la bols, y las reacciones del precipitin de agar gel a IBF se notaron durante todo el período de crecimiento.

Aunque un poco variable, el modelo de crecimiento de la bolsa usando el método del diametro, se caracteriza per una fase de crecimiento rápido desde 1-6 semanas de edad, seguido por una regresión iqualmente rápida que nivela para aterrizar entre 8-12 semanas. Una fase segunda de crecimiento empieza a aproximadamente a 12 semanas, llegando en una cima entre 16-20 semanas de edad. Las medidas además de 20 semanas indican una regresión segunda. Los diametros promedios de la segunda cima variaron entre 0.98 cm y 1.81 cm. La variabilidad en los diametros llegó a su máximo acerca de los dos cimas.

El diametro de la bolsa y el peso de la bolsa cubicado se correlacionaron sumamente en todas las edades. Los diametros de la bolsa correlacionaron significamente con el peso del cuerpo hasta la cuarta semana de edad, después de que esta relación se hizo débil con tiempo. Los modelos de diametro de los pollos vivos y los pollos ya muertos (de la mortalidad diaria) fueron bastante semejantesñ aunque la bolsa de los pollos muertos fueron desplazados abajo la de los pollas vivos por término medio de 0.19 cm en diametro.

De las parvadas probaron para exposición a IBF, ninguna fue vacunada contra IBF; sin embargo, todas producieron reacciones positivas a las pruebas de precipitin de agar gel durante la primera fase de regresión.

Aunque los datos de este estudio son sólo preliminarios un modelo consistente de crecimiento de la bolsa existe entre las parvadas. Se puede hacer las medidas de diametro de la bolsa de Fabricio en vez de las medidas de peso, y así permite la determinación del tomaño de la bolsa al nivel de la granja. Si la función de la bolsa relaciona al tomaño de la bolsa en las edades tempranas (1-4 semanas de edad) este método tiene posibilidades respeto a la determinación temprano del daño al sistema inmuno.

SIGNIFICANCE OF BURSAL SIZE SURVEY

Douglas R. Kuney, Staff Research Associate, Arthur A. Bickford,
Extension Veterinarian, and Donald D. Bell, Farm Advisor,
University of California, Riverside, CA 92521

Introduction

The role of the bursa of Fabricius in the development of the humoral antibody-forming capacity of the chick was demonstrated by Glick, 1956 (1). About 6 years later (1962) the contagious virus causing Gumboro disease, which infects, damages and causes atrophy in the bursa of Fabricius, was isolated. Since then there has been concern about the effects of Gumboro or infectious bursal disease (IBD) on the immune system of poultry. The importance of the bursa of Fabricius in the development of immuno-competent B-lymphocytes has been clearly shown by several researchers since these early discoveries.

Glick, 1956 (2), characterized bursal growth and regression in White Leghorn chickens between 2 days and 10.5 weeks of age. He reported that most rapid growth of the bursa occurs before 4 weeks of age. Bursal regression occurred between the 6th and 7th week of age. Jolly, 1913 and 1914 (3,4), reported bursal regression between 16 and 20 weeks of age.

It was felt that if the bursa of Fabricius had a definite pattern of development in the growing chicken, bursal damage might be detectable by making size measurements. This method would be particularly useful to the ranch manager who is trying to detect early damage to the immune system because of IBD or some other agent affecting the bursa of Fabricius (such as aflatoxin).

Experimental methods

The managers of five commercial pullet-growing firms were asked to make sample diameter measurements of the bursae in 10 pullets each week, starting at 1 week of age and continuing for 20 weeks. Five pullets were to be normal appearing and live, while five pullets were to be from that day's mortality. Managers made these measurements by placing a plastic template with a series of holes ranging from 0.32 cm to 2.54 cm in diameter over the intact but exposed bursa. The hole which most closely approximated the diameter of the bursa was recorded. Five additional commercial flocks were sampled by researchers at the University of California at 2, 4, 6, 8, 12, 16, 21, and 24 weeks of age. The university researchers recorded body weight, bursal diameter, bursal weight, and agar gel precipitin (AGP) reactions to IBD virus antigen in each of the 10 birds. Diameter measurements were made by the same method used by ranch managers.

Results

Bursal development measured by the University of California researchers (Table 1) is characterized by a rapid growth phase between 2 and 6 weeks of age, followed by an equally rapid regression between 6 and 8 weeks of age. A second unexpected growth phase occurred between 12 and 16 weeks, followed by a more gradual regression to 24 weeks. Disappearance of the bursa was not observed by 24 weeks of age. First peak diameters averaged between 1.40 cm and 1.94 cm. Second peak diameters averaged between 0.98 cm and 1.81 cm. Variability in both bursal weight and diameter increased with bursal size. The growth patterns of bursae recorded by ranch managers were similar, although the variability was greater.

Table 1. Development of the Bursa of Fabricius

Age (wks)	Diameter (cm)	Weight (g)	Body Weight (g)
2	1.09	0.43	100
4	1.42	1.12	207
6	1.72	1.97	377
8	1.31	0.74	529
12	1.30	0.70	894
16	1.68	2.03	1118
21	1.62	1.75	1342
24	1.27	0.64	1546

Diameter patterns from live and already dead (daily mortality) birds were very similar; although bursae from dead birds were displaced below those from live birds on the average of 0.19 cm in diameter (Table 2).

Table 2. Diameter of Bursae From Live and Dead Birds

Age (wks)	Live (cm)	Dead (cm)	Age (wks)	Live (cm)	Dead (cm)
2	1.02	0.84	12	1.40	1.27
4	1.56	1.17	14	1.37	1.24
6	1.46	1.31	16	1.44	1.32
8	1.41	1.24	18	1.50	1.31
10	1.40	1.30	20	1.25	1.10

Bursal weight and diameter were highly correlated through all ages (2 through 24 weeks of age) as shown in Table 3. The best linear correlation occurred when the bursa weight was raised approximately to the third power. This relationship is consistent with the spherical shape of the bursa.

Table 3. Bursal Size and Relationship

Age (wks)	Size (cm)	Weight (g)	R_1*	R_3*
2	1.09	0.43	0.96	0.97
4	1.42	1.12	0.97	0.98
6	1.72	1.97	0.97	0.98
8	1.31	0.74	0.95	0.96
12	1.30	0.70	0.91	0.98
16	1.68	2.03	0.97	0.97
21	1.62	1.76	0.95	0.95
24	1.27	0.64	0.75	0.93

Avg. R_1 = .91
R_3 = .97

*R_1 -- Size x $(wt)^1$
*R_3 -- Size x $(wt)^3$

The bursal weight and diameter relationship did not change with age as indicated by the similarity between slopes at different ages (Table 4).

Table 4. Effect of Age on Bursal Weight to Bursal Size Relationship

Age (wks)	Slope b	Signif*	Age (wks)	Slope b	Signif*
2	$.745 \times 10^{-2}$	n.s.	12	$.803 \times 10^{-2}$	n.s.
4	$.943 \times 10^{-2}$		16	$.996 \times 10^{-2}$	
6	$.974 \times 10^{-2}$		21	$.863 \times 10^{-2}$	
8	$.709 \times 10^{-2}$		24	$.103 \times 10^{-2}$	

*F Test to $P \geq 0.10$.

As expected, body weight was not well correlated with bursal diameter when averaged over all ages (R = 0.17), since the growth curve for body weight is almost linear while that of the bursa is more sigmoidal in shape (see Table 5). These two measurements, however, were significantly correlated at 2 and 4 weeks of age as indicated by the high regression coefficients. After 4 weeks of age the correlation between body weight and bursal size deteriorated.

Of the 5 flocks sampled by the university researchers, none was vaccinated for IBD. All 5 flocks yielded 90 percent or more positive reactions to the AGP tests at 6 weeks of age but were 10 percent or less negative at 4 weeks, thus an indication of a field exposure to the IBD virus. In each case the timing of detected antibody to IBD corresponded with a regression in the development curve of the bursa. Maternal antibody was detected by the AGP tests in only one of the flocks.

Table 5. Bursal Size and Bodyweight Relationship

Age (wks)	Size (cm)	Body Wt (g)	R	Age (wks)	Size (cm)	Body Wt (g)	R
2	1.09	100	0.76	12	1.30	894	0.09
4	1.42	207	0.75	16	1.68	1118	0.03
6	1.72	377	0.51	21	1.62	1342	-0.38
8	1.31	529	0.30	24	1.27	1546	-0.39

Since both body weight and age had significant effects on bursal development, a multiple regression analysis was performed on the data collected by the university. The predicted bursal diameters are given in Table 6 for pullets having the listed body weights. Any interpretations from this table, however, should be done with caution since a relatively small amount of data was used in preparing these predicted values.

Table 6. Predicted Bursal Size by Age for Average Body Weight

Age (wks)	Avg body wt (g)	Predicted bursal size (cm)	Age (wks)	Avg body wt (g)	Predicted bursal size (cm)
2	100	1.08	12	894	1.29
4	207	1.47	16	1118	1.66
6	377	1.60	21	1342	1.64
8	529	1.39	24	1546	1.27

Conclusion

Although the information from this study is considered preliminary only, there appears to be a consistent pattern of development in the bursa between 1 and 24 weeks of age. The bursal weights measured in this study correlate well with those measured by Glick, 1956 (2). However, Glick did not make measurements beyond 7 weeks of age for female White Leghorns. The results of this study show an interesting and unexpected second growth phase between 16 and 21 weeks of age.

Since the regression occurring at about 6 weeks of age corresponded with the detection of antibody to the IBD virus, it is possible these flocks suffered a mild infection which did not destroy the generative epithelium of the bursae. The second growth phase described earlier may merely be a continuation of development following the infection. If this explanation is valid, the birds studied by Glick in 1956 (2) may also have been infected by a similar virus, thus giving additional credibility to the findings of Jolly (3). It becomes obvious from these findings that continued research is necessary for the determination of normal bursal development.

As shown by the data, bursal diameter is well correlated with bursal weight, and so diameter measurements in the field could be made instead of weight measurements. If either deviation from expected bursal diameter or actual bursal weight can be shown to be correlated with bursal function, this technique of surveillance may prove to be a valuable tool in determining whether early damage to the immune system has occurred.

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ASSAYS OF IMMUNOCOMPETENCE IN INFECTIOUS BURSAL DISEASE (IBD)

V. Sivanandan, D.V.M., M.S., Ph.D. and S.K. Maheswaran, D.V.M., Ph.D.
Department of Veterinary Pathobiology
College of Veterinary Medicine
University of Minnesota
St. Paul, Minnesota 55108

Two groups of chickens were injected with a field isolate of IBD virus at 1 day (within 6 hours of hatch) and at 3 weeks of age, by intra-ocular route. (1) A lymphocyte stimulation assay (whole blood technique) using phytohemagglutinin (PHA) and concanavalin A (Con A) as mitogens was performed. The results indicated a bimodal pattern with a minimum inhibition between 3-4 weeks post-infection (p.i) and a maximum inhibition at 6 weeks p.i. (2) A one-way mixed lymphocyte reaction (MLR) assay was done using mitomycin-C treated cells as stimulator cells and lymphocytes from the experimental birds as the responder cells. The results showed a significant difference in the response of chickens infected at 1 day to that at 3 weeks of age. An attempt will be made to correlate the effect of IBD virus on the cellular differentiation to the functional immunocompetence of the chickens.

LOS ENSAYES DE INMUNOCOMPETENCIA DE LA INFECCIÓN DE LA BOLSA DE FABRICIO (IBF)

V. Sivanandan, Department of Veterinary Pathobiology, College of Veterinary Medicine, University of Minnesota, St. Paul, Minnesota 55108

Se inyectaron dos grupos de pollos con un aislado del campo de IBF a un día de edad (dentro de 6 horas de empollar) y a 3 semanas por medio intra-ocular. (1) Se hizo un ensaye de estimulación limfocita ((la técnica usando la sangre total) que usa fitohemaglutinin (Fha) y concanavalian A (Con A) como mitogenos. Los resultados indicaron un modelo bimodal con una inhibición mínima entre 3-4 semanas después de la infección y una inhibición máxima a 6 semanas después de la infección. (2) Un ensaye con una reacción mezclada de un camino de limfocita se hizo usando las células tratadas con el mitomicin C como las células estimuladoras y las limfocitas de pollos experimentales como las células respondedoras. Los resultados enseñaron una diferencia significativa con respeto a las respuesta de los pollos infectados a un día en contraste con la de los pollos infectados a 3 semanas. Un esfuerzo será hecho para correlacionar el efecto del virus IBF en la diferenciación de las células a la inmunocompetencia funcional de los pollos.

Introduction

The peripheral blood lymphocytes of many species have been shown to undergo blast transformation when activated by non-specific phytomitogens such as phytohemagglutinin (PHA), Concanavalin A (Con A), pokeweed mitogen (PWM) and endotoxin. The *in vitro* lymphocyte stimulation assay using purified lymphocytes has gained importance as a widely accepted assay for testing lymphocyte immune functions with applications in the studies of immunocompetence. Nevertheless, in recent years several lymphocyte transformation studies using whole blood (non-separated) rather than lymphocyte-enriched preparation have been reported. Several workers have shown that PHA and Con A responsiveness was a property of thymus-derived lymphocytes (T-cells) in chickens. An experiment was designed to utilize the microculture system to measure chicken lymphocyte mitogen-induced blastogenesis, using whole blood technique in virus infected and control birds.

A one-way mixed lymphocyte reaction (MLR) where mitomycin-C or X-ray was used to block the DNA synthesis of one of the allogeneic cell populations, thereby permitting accurate evaluation of the responsiveness of either cell population has been reported. Stimulation or non-stimulation in the one-way MLR, reflects the antigenic disparity between responding and stimulating cell populations. There was evidence to support the idea that MLR responder cells were T-cells and the stimulator cells were B cells. Based on these findings, an experiment was designed by which a one-way MLR was performed using mitomycin-C treated cells as stimulator cells obtained from chickens which were genetically different strains and lymphocytes from birds that were infected with IBD virus and control chickens as the responder cells.

Experimental Design

Specific pathogen free eggs were hatched at our department facility, and one group of chickens were infected with a field isolate of IBD virus by intraocular route within 6 hours of hatch, while another at 3 weeks of age. A third group served as uninfected controls. Each group were housed in separate isolation units.

Lymphocyte Stimulation Assay

A microculture system using the whole blood technique was performed. Phytohemagglutinin (PHA-P) and Concanavalin A (Con A) were used as mitogens. ^{125}I-2 deoxyuridine (^{125}I-udR) was used as the labeling agent. Cultures were then harvested on glass filter paper and counted on a gamma counter.

Results

The results of our study were expressed as percent inhibition (PI):

$$PI = \frac{\text{Mean counts per minute (cpm) of control} - \text{mean cpm experimental}}{\text{Mean cpm control}} \times 100$$

A comparative study made on the percentage inhibition of PHA and Con A stimulated peripheral blood lymphocytes of both 1 day and 3 week old IBDV-infected chickens, showed a bimodal pattern, with a minimum inhibition between 3-4 weeks post-infection (p.i.) and a maximum inhibition of 6 weeks p.i.

One-way Mixed Lymphocyte Reaction (MLR) Assay

Isolated peripheral blood lymphocytes of chickens were used for the one-way MLR assay. The stimulator cells were treated with mitomycin-C which blocked DNA synthesis. The responder cells were obtained from the experimental birds. In control cultures, mitomycin-C treated autologous cells were substituted for mitomycin-C treated allogeneic cells. ^{3}H-thymidine was used as the labeling agent. Samples were then counted in a liquid scintillation spectrometer.

Results

Calculation of stimulation index (SI) -

$$S.I. = \frac{\text{Mean counts per minute of mixed cultures}}{\text{Mean counts per minute of the corresponding controls}}$$

The mean stimulation index of each group (IBDV-infected at 1 day and 3 weeks of age, uninfected controls) were compared at 6, 8 and 10 weeks of age. The results showed that chickens infected at 1 day and 3 weeks of age had a significantly lowered MLR response than uninfected control chickens. The significance was greater at 6 weeks of age than at 10 weeks of age.

An attempt will be made to correlate the findings of this study to the absolute numbers of peripheral blood T and B cells (reported at 28th WPDC, 1979). Effort will be made to summarize the overall effect of IBDV on the immunocompetence of chickens infected at 1 day and 3 weeks of age.

STUDIES ON INFECTIOUS BURSAL DISEASE VIRUSES OF CHICKENS, TURKEYS AND DUCKS

J. B. McFerran and M. S. McNulty
Veterinary Research Laboratories, Stormont, Belfast, BT4 3SD,
N. Ireland

Viral diagnosis of IBD can be made by (a) identification of the virus by direct EM examination of bursal smears, (b) serological testing using the SN or indirect FA tests, (c) virus isolation. Most of our isolates were made in chick embryo fibroblast cultures. Usually 2-5 blind passages were required before a consistent cpe was seen. However virus growth could be detected in early passages by immunofluorescence. Some strains could only be isolated in birds or embryonated eggs.

We have made a number of isolates of IBD virus from chickens, turkeys and ducks. These isolates and viruses from other laboratories were compared serologically. They were classified into 2 serotypes using the virus neutralisation test. The majority of isolates belonged to serotype 1, which included the duck virus and chicken isolates from Europe and the USA. However there was considerable antigenic variation within this group. Serotype 2 included 2 isolates from turkeys and one from chickens. These viruses were closely related.

If protection in vivo mirrors virus neutralisation then more than one vaccine will be required. Furthermore, even within serotype 1, one vaccine may not give adequate protection. However if cross neutralisation does not reflect cross protection, then serotype 2 viruses might be useful for vaccinating day old birds with high levels of maternal antibody to serotype 1 viruses.

ESTUDIOS SOBRE EL VIRUS DE LA INFECCION DE LA BOLSA DE FABRICIO EN GALLINAS, PAVOS Y PATOS.

El diagnóstico viral de la infección de la bolsa de Fabricio (IBF) se puede realizar por: (a) identificación del virus por el examen directo de - impresiones bursales en el microscopio electrónico: (b) pruebas serológicas mediante virus neutralización o fluorescencia indirecta, (c) aislamiento del virus.

La mayor parte de nuestros aislamientos fueron hechos en fibroblastos de embrión de pollo. Generalmente se requirieron 2-5 pases ciegos antes de que aparecieran lesiones consistentes. Sin embargo, el crecimiento del virus podría ser detectado en los primeros pases con inmunofluorescencia. Algunas cepas pudieron ser aisladas sólo en aves o en huevos embrionados.

Hemos realizado un cierto número de aislamientos del virus de IBF (VI-BF) de gallinas, de pavos y de patos. Estos aislamientos y virus de otros laboratorios se compararon serologicamente y se les clasificó en 2 serotipos basados en la prueba de virus neutralización. La mayoría de los aislamientos pertenecen al serotipo 1, en el que se incluyen el virus de los patos y los aislamientos de gallinas de Europa y los EUA. Sin embargo, hubo una considerable variación antigénica aun dentro de este grupo. El serotipo 2 incluye 2 aislamientos de pavos y uno de gallinas, los que están estrechamente relacionados.

Si la protección in vivo semeja a la virus neutralización. Será necesaria más de una vacuna dentro del serotipo 1, una sola vacuna no será suficiente protección. Sin embargo, si la neutralización cruzada no se refleja en protección cruzada el serotipo 2 puede ser de utilidad para vacunar aves de un día de edad que tengan niveles elevados de anticuerpos maternos contra el serotipo 1.

Traducción: cortesía del Dr. Benjamín Lucio Martínez

STUDIES ON INFECTIOUS BURSAL DISEASE VIRUSES ISOLATED FROM CHICKENS, DUCKS AND TURKEYS

J.B. McFerran, Ph.D. B.Sc. M.R.C.V.S. and
M.S. McNulty, Ph.D. M.V.B. M.R.C.V.S.
Department of Agriculture, Northern Ireland,
Veterinary Research Laboratories, Stormont,
Belfast, BT4 3SD, Northern Ireland

During the last 5 years in Northern Ireland, there has been an evolution of infectious bursal disease (IBD). Initially neither antibody nor bursal lesions were seen. Antibody was then detected, followed by the development of bursal lesions. Disease was subclinical at first, but recently severe outbreaks of disease associated with the immunosuppressive effects of IBD have been seen.

Virus isolation attempts in chick embryo fibroblast (CEF) cell cultures were surprisingly successful. With most isolates poor or no cytopathic effects were seen following initial inoculation, but immunofluorescent staining (IF) showed a few positive cells. After 2-3 blind passages, cpe was produced. When isolation attempts were made from birds with severe bursal lesions, CEF inoculation was less successful. Although some fluorescing cells were seen on initial passage, these decreased and disappeared on further passage, and cpe was not seen. These virulent strains were isolated by inoculating young chicks ocularly. To aid in diagnosis and in the selection of specimens suitable for bird inoculation, bursal smears were examined by electron microscopy (EM) (3). This technique proved most useful. More recently we have compared the sensitivity of EM and IF examination of bursal impression smears. Provided high quality antiserum is used, the IF technique is at least as sensitive as the EM.

Isolates were classified as being IBD primarily on their morphology. If they grew in cell culture the group antigen was demonstrated using immunofluorescence. If they did not grow in cell culture, bursas collected 3 days after infection was used as antigen in the double immunodiffusion test.

Antiserums were prepared against a number of isolates and then the relationship studied by serum neutralisation tests in CEF cells. It was possible to divide the isolates into 2 distinct serological groups (4).

Group I contained a vaccine virus (Intervet), a virulent virus from the USA (2), a cell culture adapted virus from the USA (Lasher), a duck isolate made in Northern Ireland during investigations on EDS, and 5 chicken isolates made in Northern Ireland. When the cross neutralisation results of Group I viruses were analysed using the formula of Archetti and Horsfall (1) it was found that these isolates do not form a homogenous group. Thus whilst the vaccine virus and the duck isolate were virtually identical, the US Lasher strain showed considerable antigenic drift from the British vaccine strain ($R = 3.3$) and some Northern Ireland isolates ($R = 2.8$ to 5.7). The virulent American strain was however closely related to 2 local isolates.

Group 2 contained two isolates made from turkeys and one from a fowl (4, 5). These viruses are antigenically closely related.

It is not yet known if cross protection mirrors cross neutralisation. If it does, then at least two vaccines will be required. If it does not, then a type 2 vaccine might be used to vaccinate young chicks in the face of high levels of maternal antibody to type 1 virus.

The origins of these IBD viruses within Northern Ireland remain interesting. Initially we were concerned that it might have been illegal use of vaccine. The difference in antigenicity refute this. Following the realisation that IBD and infectious pancreatic necrosis (IPN) virus of

trout were morphologically identical, it was postulated that these viruses were identical and fish meal might be the source. However although biochemical studies showed a close relationship, differences did exist and furthermore they are not serologically related, not even sharing a group antigen (6).

The fact that IBD has a segmented RNA genome suggests that ample scope exists for genetic change, as happens in other viruses - eg Influenza and Orbiviruses, with similar configuration.

Acknowledgements

We wish to thank Mr W. Baxendale and Dr H. Lasher for supplying us with their strains of viruses.

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DIAGNOSIS OF RESPIRATORY DISEASES IN POULTRY

H. John Barnes, D.V.M., Ph.D.
Veterinary Medical Research Institute
Iowa State University
Ames, Iowa, 50011

Introduction

Collectively, respiratory diseases are currently one of the most important disease problems faced by poultry producers today. Economic loss results from increased mortality, increased condemnations, decreased productivity and increased costs required to manage and treat affected flocks. Perhaps more importantly, respiratory diseases serve as a barrier to the introduction of more labor and energy efficient technology.

Respiratory diseases have increased in importance because of changing production methods where birds are reared in confinement in increasingly larger flocks, multi-age flocks are present on the farm, maximum population densities are used, and there is minimum clean-up between flocks. Nowadays respiratory disease outbreaks can rarely be attributed to a single infectious agent. Rather they result from the interaction of a multiplicity of factors involving infectious agents, environment and the host. It is incumbent upon the disease investigator to identify as many factors as possible -- both primary and predisposing -- that might be involved in a respiratory disease outbreak so that an appropriate preventative program can be developed and instituted.

In this presentation an approach to the diagnosis of respiratory diseases in poultry will be presented. Since the procedure was developed during investigations into the causes of respiratory diseases of turkeys, this avian species will be used as a model.

Clinical Examination

Select a representative cross section of birds from the flock. Six is usually a sufficient number of live birds with 4-6 additional dead birds which are still in good condition. Live birds should represent the spectrum of the disease process. Conduct a careful clinical examination. Determine if signs indicate a disease process primarily involving the upper respiratory tract (rhinitis, conjunctivitis, sinusitis), trachea (rales, dyspnea) or lower respiratory tract (cyanosis, dyspnea). Weigh the birds and compare with expected standards. Collect blood for serum and prepare a thin blood film.

Necropsy

Conduct a complete necropsy examination and note all lesions. Aseptically collect swabs for bacteriology from affected portions of the respiratory tract and collect tissues for virus isolation. Place samples of affected tissues in 10% buffered neutral formalin for histopathology. Make impression smears of lesions. Remove the bursa of Fabricius and weigh it. Calculate bursal weight as a percent of body weight. Compare values with standard values. Weights of adrenal glands may also be helpful as a general indicator of a pre-existing "stress" condition. Collect composite fecal sample.

Microbiology

Culture swabs and tissues on appropriate media, in chicken embryos or in tissue cultures. Examine serums for antibodies to respiratory disease agents. Obtain a set of 10-12 convalescent serums about 4 weeks later for retesting.

Clinical Pathology

Evaluate blood smear for approximate numbers of white cells and percentage of different types. Note any abnormalities in erythrocytes and look for parasites. Rapid dip stains reduce time required to prepare blood smears.

Do a fecal floatation. Examine for parasite ova and cysts.

Stain and examine cytological preparations. Cytology has been found useful in rapidly differentiating bacterial infections and confirming suspected mycotic infections. Using this technique a highly accurate diagnosis can often be made in less than an hour.

Histopathology

Examine and evaluate histologic sections. Routine use of Giemsa stain has been found to be particularly useful for interpreting respiratory tract pathology. The stain is easily done and infectious agents -- bacteria, fungi and mycoplasma -- are readily visualized.

Farm Visit

Although it is not always possible, a visit to the affected flock may explain many apparent mysteries seen in the laboratory. Note especially ventilation and litter conditions.

Other Procedures

In selected cases rapid electron micropsy processing techniques have been used on tracheal mucosa to confirm mycoplasmal infections. Direct EM has been used to identify respiratory adenovirus infections but has been of limited value in diagnosis of other respiratory viral diseases. A variety of fluorescent antibody techniques have been reported by other investigators which need to be evaluated.

Conclusion

This procedure will provide an excellent assessment of the avian respiratory tract. It provides for an evaluation of the host, environment and identification of known (and occasionally previously unknown) infectious agents; the 3 main components in avian respiratory disease. This information will provide a basis for developing a respiratory disease prevention, control and treatment program on the farm.

DIAGNOSTICO DE ENFERMEDADES RESPIRATORIAS EN AVES DE CORRAL

Introducción

Colectivamente, las enfermedades respiratorias son comúnmente una de las más importantes enfermedades que afrontan los avicultores actualmente. Perdidas económicas son el resultado del incremento en mortalidad, incremento en confiscación de canales, reducción en productividad e incremento de costos requeridos para el control y tratamiento de parvadas afectadas. Posiblemente lo más importante de las enfermedades respiratorias es que sirven como una barrera a la introducción de tecnología energetica eficiente y mano de obra.

Las enfermedades respiratorias han incrementado su importancia debido al cambio en los metodos de producción en los cuales las aves crecen en largas parvadas en confinamiento, diferentes edades existen en la granja, máxima población es usada, y un minimo de aseado existe entre parvadas. Hoy día los brotes de enfermedades respiratorias pueden ser raramente atribuidas a un solo agente infeccioso, más bién son el resultado de la combinación de diferentes factores los cuales incluyen agentes infecciosos, medio ambiente y el huésped. Es la obligación del investigador de la enfermedad, la identificación del mayor número de factores posibles -- ambos primarios y predisponentes -- que puedan estar implicados en un brote de enfermedad respiratoria para que con ello se puedan elaborar e instituir los programas preventivos apropiados.

En esta presentación será expuesto un acercamiento al diagnóstico de las enfermedades respiratorias de las aves de corral. Debido a que este procedimiento fué desarrollado durante una investigación de las enfermedades respiratorias en guajolotes, esta especie de aves será usada como modelo.

Examinación Clinica

Seleccione una sección representativa de aves en la parvada. Seis animales es normalmente un número adecuado más 4-6 aves muertas que estén en buenas condiciónes para el examen de postmuerte. Los animales vivos deben ser representativos del problema. Efectue una cuidadosa examinición clinica. Determine si los signos clínicos indican que la enfermedad es primariamente del aparato respiratorio alto (rinitis, conjuntivitis, sinusitis), traquea (ronquido, disnea) ó del tracto respiratorio bajo (cianosis , disnea). Pese los animales y compare con pesos normales. Colecte sangre para obtener suero y prepare un fino frotis sanguineo.

Necropsia

Efectue un completo examen de necropsia y anote todas las lesiónes. Asepticamente colecte muestras para bacteriología de las áreas afectadas del tracto respiratorio y colecte tejidos para aislamiento de virus. Las muestras de las áreas afectadas para su estudio histopatólogico deberán ser sumergidas en solución buffer de formalina al 10%. Efectue impresiones en laminillas de las áreas afectadas. Separe la bolsa de Fabricio y pesela. Calcule el peso de la bolsa como porcentaje del peso del ave. Compare sus valores con valores standard. El peso de las glándulas adrenales pueden ayudar como un indicador general de condiciónes pre existentes de estres. Colecte materia fecal.

Microbiología

Cultive las muestras y tejidos en medios apropiados, en embrión de pollo ó en cultivo de tejidos. Examine si los sueros contienen anticuerpos contra los agentes causantes de las enfermedades respiratorias. Obtenga una muestra de 10-12 sueros de animales convalecientes a las 4 semanas para una nueva evaluación.

Pathología Clinica

Evalue el frotis sanguineo por el número de leucocitos y porcentaje de los diferentes tipos. Anote cualquier anormalidad en eritrocitos y busque parásitos., Tinsiones rapidas de inmersión reduce el tiempo requerido para la preparacion de frotis sanguineo.

Efectue examen fecal de flotación. Examine la presencia de huevos y cistes de parasitos. Examine y tiña preparaciones citológicas. Citología ha auxiliado en la rapida diferensiación de infecciónes bacterianas y en la confirmación de supuestas enfermedades fungales. Usando esta técnica un diagnóstico exacto puede ser establecido en menos de una hora.

Histopatología

Examine y evalue las reacciones histológicas. El uso rutinario de la tinción de Giemsa es auxiliar en la interpretación patológica del tracto respiratorio. Esta tinción es facilmente efectuada y los agentes infecciosos -- bacteria, hongos y micoplasma son rapidamente visualizados.

Otros Procedimientos

En casos seleccionados, técnicas rápidas de microscopía electrónica han sido empleados en mucosa traqueal en la confirmación de infecciones producidas por micoplasma. Microscopía electrónica directa ha sido empleada en la identificación de infecciones respiratorias producidas por adenovirus sin embargo ha sido de poco valor en el diagnóstico de estas enfermedades respiratorias virales. Diferentes técnicas de anticuerpos fluorescentes han sido reportadas por varios autores las cuales necesitan ser evaluadas.

Conclusión

Este procedimiento proporciona una excelente evaluación del tracto respiratorio en aves. Proporciona una evaluación del huésped, medio ambiente y la identificación de conocidos (y ocasionalmente desconocidos) agentes infecciosos que son los principales componentes en las enfermedades respiratorias de las aves. Esta información proporciona las bases para el desarrollo de programas preventivos, de control y tratamiento en la granja.

(Translation courtesy of Dr. Roberto Alva-Valdes)

Monitoring Programs for Disease Control, by A.A. Bickford, V.M.D., University of Missouri, Columbia, Missouri 65211.

INTRODUCTION

The verb "monitor" is used in a variety of ways and certainly means different things to different people. As it applies to poultry health programs it too frequently is given a very narrow meaning with specific reference to measurement of serum antibodies after the administration of vaccines. It would seem more prudent to use the more general definition - that is "to check or adjust the quality of a program" and to apply it in its broadest sense in periodically checking health management programs.

In a sense the chicken or turkey is the ultimate monitor of the quality of management programs. They will show evidence of poor programs in a variety of ways including unthriftiness, poor growth, mortality, depressed egg production, etc. Obviously, then, a lot can be learned by monitoring the health status of the birds. However, in some instances it's too late to adjust management programs once birds become affected. In these situations management factors need to be directly monitored. In any operation there are two major sources of health problems - 1) the birds and 2) the environment. Both are critical in growing and maintaining a healthy flock and neither can be ignored in monitoring efforts. This presentation addresses some of the major concerns in maintaining flock health and means of monitoring disease control programs.

DISEASE CONTROL PROGRAMS - THE ESSENTIALS

While this topic has been covered many times in previous meetings some elements deserve repeating. All good poultry management programs start with healthy birds whether day-old chicks or poults or started pullets. Unhealthy or uneven birds are not likely to perform well even in the best of environmental circumstances. It is absolutely essential, to develop a stringent set of guidelines for evaluating the quality of birds prior to delivery. The next critical item in management is maintenance of a well controlled environment for the birds. This encompasses housing, ventillation, lighting, feeding, water supply and security management. Finally, specific measures for disease prevention and control must be thought through and tailored to the needs of the specific operation. Included in this category are effective sanitation and hygiene programs, readily accessible veterinary and nutritional expertise, well-planned vaccination and preventive medication programs, access to reliable laboratories for various monitoring tests, and workable contingency plans for dealing with any serious disease outbreak.

MONITORING - FOR WHAT AND WHEN

Monitoring simply refers to periodic checks of your management and disease control programs. It should be interpreted as a systematic evaluation of all programs to see if they are achieving the high standards intended. Monitoring is an approach to identifying weaknesses in programs before they cause costly disease outbreaks. Monitoring is too often thought of as the application of very sophisticated analytical or serologic techniques when in fact, it is done at a variety of levels in every successful poultry operation. Some managers monitor at a "seat of the pants" or "eyeballing" level by extremely close observation of bird health and management practices. They see to it that mechanical equipment is working, that air and water quality is maintained, that vaccines are properly handled, and that birds are uniform, growing well and "looking good". Other managers combine the "eyeballing" level of monitoring with some well-selected analytical procedures such as periodic nutrient analysis of feeds, mycotoxin analysis of feed, analysis of air movement in houses, chemical and microbial analyses of drinking water, analysis of antibody levels following vaccination, post-mortem evaluation of sick, cull of dead birds, etc. The top managers have, by experience, identified those components of their programs with the greatest potential for weakness and breakdown - they also know which operational components are strong and relatively worry-free. Furthermore, they often know when weaknesses will show up, that is when weather, movement of birds, labor problems, etc. will most seriously challenge their programs. These managers apply strict and scheduled monitoring procedures of programs that are most subject to failure or that are too variable to count on and just enough periodic monitoring of strong programs to "keep everyone honest".

Finally, monitoring is meaningless unless there is a committment to quickly correct the deficiencies that may be revealed. If indicated adjustments are made monitoring can be an extremely cost effective management tool.

ESTUDIOS SOBRE LA TRANSMISSION DE LA CEPA F DEL MICOPLASMA GALLISEPTICUM

S. H. Kleven, D.V.M., Ph.D., Department of Avian Medicine, University of Georgia
Athens, Georgia

La cepa F del Micoplasma gallisepticum ha sido usada como vacuna en varias áreas de los Estados Unidos para evitar pérdidas por bajas de postura en granjas comerciales destinadas a producción de huevos.

Pruebas de campo han sugerido que la cepa F es de bajo poder de transmisión y por consiguiente puede ser usada como una ayuda en el control y erradicación de la micoplasmosis en granjas de postura con diferentes edades.

Pollas tipo leghorn libres de Micoplasma fueron vacunadas a las 18 semanas de edad por la vía ocular con la cepa F del Micoplasma gallisepticum (MG). A diferentes intervalos entre las 0 y 27 semanas después de la vacunación, 10 aves vacunadas fueron colocadas en un corral del galpón junto con 10 aves no vacunadas (controles), midiéndose el nivel de anticuerpos contra MG.

Muestras de la tráquea para cultivos y aislamiento de MG fueron obtenidos con intervalos regulares a lo largo de las 49 semanas del experimento. Tambien 5 grupos de pollos de engorde fueron levantados en el mismo galpón durante las primeras 8 semanas.

Se obtuvieron los siguientes resultados : 1. La cepa F fué fácilmente transmitida a las aves en contacto hasta las 6 semanas después de la vacunación y más lentamente después. 2. El Micoplasma no fué transmitido en aves que estaban separadas por un corral desocupado o por un corredor o pasillo. 3. Muchas aves vacunadas o infectadas por contacto fueron portadoras del organismo en la tráquea durante las 49 semanas que duró el experimento. 4. De los 5 grupos de pollos de engorde, 4 permanecieron sin infección, mientras que el grupo que se colocó adyacente a las aves vacunadas se infectó approximadamente a la 7a. semana de edad.

STUDIES ON THE TRANSMISSION OF THE F STRAIN OF MYCOPLASMA GALLISEPTICUM

S. H. Kleven, D.V.M., Ph.D., Department of Avian Medicine, University of Georgia
Athens, Georgia

The F strain of Mycoplasma gallisepticum has been used as a vaccine in several areas of the U. S. to prevent egg production losses on MG-infected, multiple-age commercial egg farms. Field experiences have suggested that the F strain is poorly transmitted, and therefore may be used as an aid in the eradication of multiple-age laying farms.

Mycoplasma-free leghorn pullets were vaccinated by eye drop with the F strain of MG at 18 weeks of age. At various time intervals from 0 to 27 weeks post vaccination, 10 vaccinates were placed in a pen with 10 controls, and the antibody response to MG was measured. Tracheal cultures for MG were obtained at intervals throughout the 49 week trial. Also, 5 groups of broilers were reared in the same house during the first 8 weeks.

The following results were obtained: 1. The strain was readily transmitted to pen males up to 6 weeks post vaccination, and more slowly thereafter. 2. The organism was not transmitted to birds which were separated by an empty pen or aisle. 3. Many vaccinates and infected contacts carried the organism in the trachea throughout the 49 week trial. 4. Of 5 groups of broilers, 4 remained uninfected, while 1 group (housed adjacent to vaccinates from 1 day of age) became infected at about 7 weeks of age.

PATHOGENICITY OF TWO STRAINS OF MYCOPLASMA GALLISEPTCIUM IN BROILERS. Rafael Rodriguez and S. H. Kleven, College of Veterinary Medicine, Poultry Disease Research Center, University of Georgia, Athens, Georgia 30605

ABSTRACT

Strains F and R of Mycoplasma gallisepticum (MG) were compared for their relative pathogenicity in terms of inducing airsacculitis and antibody production to MG.

Chickens exposed to the R strain had significantly higher air sac lesion incidence ($P < 0.05$) and greater severity of airsacculitis than did chicks which were exposed to the F strain. Chickens simultaneously vaccinated with Newcastle disease - infectious bronchitis vaccine and exposed to MG had more severe lesions than chickens exposed to mycoplasma alone.

Chickens exposed to the F strain had significantly lower gemometric mean hemagglutination-inhibition antibody titers to MG than did chicks exposed to the R strain.

RESUMEN

Los grupos de pollos comerciales de engorde que fueron expuestos a un aerosol con cepa F del Mycoplasma gallisepticum (MG) mostraron diferencia significativa ($P < 0.005$) en la evaluación de las lesiones de los sacos aéreos en relación con los grupos de pollos que recibieron el aerosol con la cepa R del MG.

Los grupos de pollos infectados por la cepa F del MG, obtuvieron diferencia significativa en los niveles de anticuerpos del MG por la prueba de inhibición de la hemaglutinación (IH) comparadas con los pollos infectados con la cepa R del MG.

El grupo de pollos vacunado contra la enfermedad de New Castle y Bronquitis Infecciosa (ENC-BI), mostró título mayor de anticuerpos contra ENC que los grupos de pollos infectados con la cepa F y R del MG.

La cepa F del MG, de baja virulencia (menos de 21 pase) puede ser utilizada como cepa vacunal en pollos commerciales de engorde para evitar descartes por aerosaculitis ocasionadas por el MG, aprovechando su buen poder antigénico y grado de protección una vez se prueba su estabilidad.

Se demonstró la facilidad y rapidez del aislamiento del MG de muestras tomadas a partir de la tráquea con relación a las tomadas de los sacos aéreos.

Introducción

La Mycoplasmosis aviar es todavía una de las enfermedades que más daños económicos ocasiona a la avicultura en el mundo, por cuanto la presencia de los mycoplasmas patógenos aviares Mycoplasma gallisepticum (MG), Mycoplasma synoviae (MS), Mycoplasma meleagridis (MM), y posiblemente el Mycoplasma gallinarum (3) en las aves, produce aumento de la morbilidad, mortalidad, condenación o descarte en los mataderos por la aerosaculitis, peligro para la salud pública por la asociación en ocasiones con bacterias patógenas para el hombre tales como Staphilococcus aureus, Escherihcia coli, etc., afectando además la conversión alimenticia de la parvada.

El presente estudio fué adelantado con el fin de obtener una mejor información sobre el comportamiento de 2 cepas (R y F) del Mycoplasma gallisepticum y su posible uso en la elaboración de una vacuna para controlar esta enfermedad en los pollos de engorde y en las aves de postura.

Los objectivos del trabajo fueron:

1) Evaluar las lesiones en los sacos aéreos del ave producidos por cada una de las cepas del MG, usdas en el experimento.

2) Detectar la presencia del organismo mediante aislamiento a partir tráquea y sacos aéreos y su identificación por medio de la técnica de inmunofluorescencia.

3) Detectar el nivel de anticuerpos mediante las pruebas de aglutinación en placa e inhibición de la hemaglutinación (IH).

4) Establecer una correlación con los niveles de anticuerpos para la enfermedad de New Castle.

Materiales y métodos

Infección con las cepas R y F del Mycoplasma Gallisepticum. La cepa R (aislada por Richey, 1961, Poultry Disease Research Center, Athens, Georgia [P.D.R.C.]) fué suministrada por el Dr. H. W. Yoder, Jr., Southeast Poultry Research Laboratory, Athens, Georgia. La cepa F, fue facilitada por el Dr. Gentry, Pennsylvania State University.

Las cepas fueron cultivadas en el medio Frey modificado. Los pollos fueron infectados mediante un aerosol, por 5 minutos, con el cultivo de estas cepas de MG en caldo nutritivo de 24 horas. El aparato usado fue un nebulizador Vaponefrin standard (Vaponefrin Company, Edison, N. J.).

Aislamiento e identificasión de Mycoplasmas. Se usaron hisopos estériles para tomar las muestras de la tráquea y los sacos aéreos y luego se sumergieron y agitaron varias veces en caldo de Mycoplasma al cual se le había agregado previamente rojo fenol para facilitar la lectura de la reacción y crecimiento del Mycoplasma, luego se pasó al Agar de Mycoplasma. Se incubaron at 37° c., y ambiente húmedo por 4-5 días; observándolos diariamente para ver el crecimiento de colonias de mycoplasmas. Posteriormente se usó la técnica de inmunofluorescencia para su identificación.

Procedimientos serológicos. A todas las muestras de suero se les hizo la prueba rápida de placa y de inhibidión de la hemaglutinación.

Evaluación de la lesión de los sacos aéreos. Todos los pollos fueron necropsiados y examinados para observar las lesiones de los sacos aéreos, teniendo en cuenta el criterio establecido por Kleven, et al.

Diseño experimental. Ciento veinte pollos comerciales de engorde de 1 día de edad provenientes de una incubadora local y libres de MS, y MG, fueron usados en el experimento. Cuatro grupos de 25 y 1 grupo de 20 aves de 1 día de edad fueron colocados en 5 unidades de aislamiento Horsfall Bauer, que poseen aire filtrado y presión positiva.

No. Aves	No. Grupo	Tratamiento
25	1	Recibieron cepa "F" virulenta por aerosol (5 pase 4 x 10^8 cfu/ml)
25	2	Recibieron cepa "F" virulenta por aerosol más la vacuna ocular contra New Castle y Bronquitis Infecciosa (ENC-BI)
25	3	Recibieron cepa "R" virulenta por aerosol (18 pase 3 x 10^6 cfu/ml)
25	4	Recibieron cepa "R" virulenta por aerosol más ENC/BI, ocular.
20	5	Recibieron únicamente vacuna de ENC-BI, vía ocular.

- A los 14 días de edad se les aplicó el respectivo tratamiento.
- Muestras de sangre tomadas por punción en el corazón, hisopos con muestras de la tráquea y sacos aéreos fueron tomados de 5 aves de los grupos No. 1, 2, 3, 4; y 4 aves del grupos No. 5 a los 5, 12, 18, 23 y 30 días después del tratamiento. Al mismo tiempo la evaluación de lesiones de los sacos aéreos fue practicada.

Estudio estadístico de los datos obtenidos se realizó al final del experimento.

Resultados

En las tablas 1 y 2, se muestra el número de aislamiento del Mycoplasma en las cinco oportunidades en que se tomaron muestras de la tráquea y sacos aéreos de los grupos de aves del experimento.

Se confirma una vez más lo demonstrado por otros investigadores, en cuanto a la facilidad y rapidez para hacer aislamientos de la tráquea con relación a los sacos aéreos y la diferencia notada en el mayor aislamiento del organismo de los sacos aéreos en los grupos que reciberon el aerosol con la cepa R sola y/o con la vacuna de ENC-BI con relación a los que recibieron la cepa F sola y/o con la misma vacuna.

Los resultados de presencia de anticuerpos detectados por las pruebas de aglutinación en placa e inhibición de la hemaglutinación, se muestran en las tablas 3 y 4 para MG, y tabla 5, para ENC.

En la tabla 4, se observa la diferencia significativa ($P < 0.005$) entra la cepa R sola y/o combinada con la vacuna de ENC-BI y la Cepa F en las mismas condiciones.

Los resultados del grupo que recibió la vacuna de ENC, presentó títulos más altos que los grupos de pollos que recibieron el aerosol con las dos cepas del MG, más la vacuna contra ENC-BI.

Se notó reducción de anticuerpos contra la ENC en los grupos de aves infectadas con la cepa R sola y/o asociada con la vacuna ENC-BI en relación con los grupos de pollos infectados con la cepa F sola y/o combinada con la vacuna ENC-BI.

La evaluación de lesiones de los sacos aéreos se muestra en la tabla 6. Se observó diferencia significativa ($P < 0.005$) entre los grupos infectados con las cepas F y F+ENC-BI y comparados con los lotes infectados con la cepa R y R+ENC-BI.

Discusión

Teniendo en cuenta los resultados se puede concluír que la Cepa F del MG sola y/o combinada con la vacuna de New Castle y Bronquitis Infecciosa no produce mayores lesiones en los sacos aéreos o su nivel es bajo, no detectable macroscópicamente al momento en que los pollos comerciales de engorde están listos para su sacrificio y salida al mercado.

Igualmente se establece que es más fácil y rápido el aislamiento del organismo de la tráquea en general con relación a los sacos aéreos, especialmente en los primeros días de vida del pollito para así poder establecer el diagnóstico y aislamiento del organismo a nivel de incubador o galpón de cría.

Por los resultados del presente estudio se pudo establecer que aves infectadas con cepas patógenas o virulentas de MG, presenta títulos de anticuerpos IH contra ENC menores que aves no infectadas contra Mycoplasma.

Una vez se establezca su establidad la cepa F en pases bajos (menos de 21) se puede utilizar como cepa vacunal contra la enfermedad denominada Mycoplasmosis aviar por MG en pollos comerciales de engorde, ya que posee buen poder antigénico y capacidad de protección.

AN ECONOMIC ANALYSIS OF *MYCOPLASMA GALLISEPTICUM* CONTROL IN LAYER CHICKENS

T. E. Carpenter, Ph.D., M.S., K. F. Miller, B.S., R. F. Gentry, D.V.M., Ph.D., M.A., L. D. Schwartz, D.V.M., M.S., and E. T. Mallinson, V.M.D.
Department of Epidemiology and Preventive Medicine
School of Veterinary Medicine
University of California at Davis
Davis, California 95616

Abstract

An analysis of the impact of *Mycoplasma gallisepticum* (MG) infection and controlled exposure with Conn. F-strain on egg production by commercial layer hens was performed. Production data were evaluated from 106 flocks in Pennsylvania over a 2 year period.

The study showed that the 45-week egg production of layers naturally infected with MG was 18 eggs/hen housed, or 8%, less than that of noninfected layers. This decrease was reduced to 10 eggs/hen housed, or 4.5%, when layers received a controlled exposure with Conn. F-strain prior to egg production.

An economic analysis was performed to determine the level of risk of infection with MG necessary for the controlled exposure program to be recommended. This level of risk was determined to be 60%. Therefore the program would be economically beneficial to a multiple-age laying operation having a history of MG infection.

ANÁLISIS ECÓNOMICO DEL (IMPACTO) CONTROL DEL MYCOPLASMA GALLISEPTICUM EN GALLINAS PONEDORAS

Resúmen

Un análisis del efecto que sobre la producción de huevos en gallinas ponedoras produce la infección y la exposición controlada con Mycoplasma gallisepticum Cepa Conn.-F fué relizado. Datos de producción fueron evaluados en 106 lotes ubicados en Pensilvania durante un lapso de 2 años.

El estudio muestra que ponedoras naturalmente infectadas con MG tienen una producción inferior en 18 huevos, (8%) en comparación con ponedoras no infectadas, para un período de postura de 45 semanas de duración. Esta disminución alcanza a solo 10 huevos (4.5%) cuando las ponedoras reciben una "exposición controlada" con Cepa Conn.-F antes de iniciar su período de postura.

Un análisis económico fue llavado a cabo a fin de determinar la magnitud de la probabilidad o riesgo de infección por MG para que un programa de exposición controlada sea recomendado. Dicha probabilidad fue determinada en 60%. Sin embargo el programa puede ser economicamente beneficioso, cuando en un plantel con antecedentes de infección con MG existen ponedoras de diferentes edades.

Introduction

Economic losses resulting from downgrading, reduced feed and egg production efficiency, and medication costs make Mycoplasma gallisepticum (MG) infection one of the costliest disease problems confronting the poultry industry (4).

An evaluation of production data from several commercial layer flocks of chickens was performed to quantify the impact of MG infection on egg production. In addition, the value of controlled exposure, or vaccination, using Conn. F-strain MG was estimated.

Given this information, the technique of decision theory (3) may be applied so that each individual poultryman may select the most economically optimal action, concerning MG control, given his flock's risk of exposure to MG.

Methods and Materials

Production data collected over a two year period from 106 commercial layer flocks of chickens located in Pennsylvania were analyzed. These flocks were comprised of one of five strains of layers.

Data from these flocks were separated according to flock exposure: not exposed [MG(-)]; controlled exposure with Conn. F-strain [MG(inoc)]; and natural exposure [MG(+)].

Flocks were eligible for vaccination with Conn. F-strain only when they consisted of replacement pullets entering a ranch having a confirmed MG problem. Confirmation was by a positive blood test and/or culturing of MG from infected birds already on the ranch.

Administration of the vaccine was either as a spray or in the drinking water. The time of administration was typically performed shortly after the birds were placed on the laying ranch and prior to egg production.

Three factors of the vaccination procedure were evaluated with respect to impact on egg production. These factors were: 1) method of application, 2) time of application and 3) relative affect on the various strains of layers.

The relative impact of MG infection on the different strains of chickens was also analyzed. Analysis of the data was performed using the analysis of variance technique (2).

Decision theory was used to interpret the results of the analysis as it affected the poultryman. The Hurwicz criterion (Fabrycky, 1974) was selected to determine the level of risk of a flock becoming infected with MG before vaccination would be recommended as the economically optimal alternative.

Results and Discussion

There were significant differences ($p<0.05$) in volume of egg production among each of the three exposure groups: MG(-) = 224 eggs/hen housed; MG(inoc) = 214 eggs/hen housed; and MG(+) = 206 eggs/hen housed. Compared with a MG(-) flock, the loss in egg production associated with vaccination was 10 eggs/hen housed, or 4.5%, and the loss associated with natural exposure, without vaccination, 18 eggs/hen housed, or 8.0%.

No significant difference in egg production was found to be associated with either the method or time of vaccination. Similarly, there was not a significant difference in egg production associated with either vaccination or natural exposure with MG among the various strains of chickens.

The above results may be used to assist the poultryman in deciding whether or not he should vaccinate using Conn. F-strain. However, before these alternatives may be evaluated, the following assumptions must be made:

1. The price of an egg is 4¢.
2. The cost of vaccination with Conn. F-strain is 3.5¢/bird.
3. The net benefits per hen housed for each MG situation are as follows:
   a. MG(+) = 0.0¢
   b. MG(inoc) = 8 eggs at 4¢/egg less 3.5¢ for vaccination = 28.5¢
   c. MG(-) = 18 eggs at 4¢/egg = 72¢

Given these assumptions, the individual poultryman may determine whether he should vaccinate a flock of replacement layers with Conn. F-strain or attempt to maintain the flock MG(-) without vaccination. To make this decision, it is necessary that he determine the break even point (BEP), or the level of risk his flock has of becoming infected with MG. At a level above the BEP he should choose to vaccinate and below the BEP he should not vaccinate.

Let P(MG(+)) = the probability of a flock becoming infected with MG
and P(MG(-)) = the probability of a flock not becoming infected with MG.

By multiplying the monetary values of the 3 possible situations (MG(-), MG(inoc), MG(+)) times their respective probabilities of occurring, the expected monetary values for each situation may be determined. From these values, the BEP may be calculated.

The BEP is calculated by solving the following:

$$P(MG(+)) \times (0.0¢) + P(MG(-)) \times (72¢) = 28.5¢,$$

where 28.5¢ = the net benefit, compared with MG(+), of inoculating a layer with Conn. F-strain.

$$P(MG(+)) \times (0¢) + P(MG(-)) \times (72¢) = 28.5¢$$
$$P(MG(-)) \times (72¢) = 28.5¢$$
$$P(MG(-)) = 28.5¢/72$$
$$P(MG(-)) = 0.40$$

Since $P(MG(-)) = 1 - P(MG(+))$, the equation may be rewritten as

$$P(MG(+)) = 1 - 0.40 = 0.60.$$

Therefore the BEP = 0.60. Thus if the risk of a replacement flock becoming infected with MG is greater than 60% the poultryman should vaccinate his flock. If it is less than 60% he should attempt to maintain a MG(-) without vaccination.

An example of where a 60% or greater risk of MG flock infection might occur is in a multiple-age laying operation having a history of MG infection. For such an operation the vaccination program is recommended.

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EYE NOTCH SYNDROME

Charles W. Howe, D.V.M.
Hy-Line International
Dallas Center, Iowa

This presentation is mainly a call for help in diagnosis for a rather unusual condition noted in several flocks over the past few years.....

Approximately two years ago I was travelling in the eastern U.S., and being questioned about a peculiar eye lesion that was being observed, primarily in New York State. I must admit I thought this was a peculiar situation about which I knew nothing.

I finally had a chance to observe this condition in two or three flocks back there. It was being called "Eye Notch Syndrome" by people back there, and since I can think of no better name to give this condition, I have continued to use this term.

The flocks I saw were of a competitive strain and needless to say, I went away saying, "thank goodness we are not bothered by that condition".

I returned to Iowa feeling pretty good but then I visited a commercial farm a short distance from DesMoines and saw the very same eye pathology that I had seen back east.

This farm has six identical houses. They grow their own pullets on the farm but separated from the layers. They are in flat deck cates and on an adequate nutrition and vaccination program. The only consistent problem they have is mites. They are Mg-free and have not had any particular problems with the exception of consistently lower than expected peak production by two or three percent. Total egg production during the laying period is on standard or above. They have from time to time been bothered by T_2 toxin lesions in the mouth.

Pullets are grown separately with standard faccination program for Pox, Newcastle, Bronchitis and A.E. Grow-out is usually over 96%, and no eye lesions are seen in the growing birds.

They are moved to the layer houses at 19 to 20 weeks and at 22 weeks the eye pathology becomes evident. The slides show the progression of the pathology. These slides were taken by Dr. Barnes, who also took a sample back to Iowa State University. Despite an extensive work-up of the case, they failed to find any etiologic reason for the condition but did get a nice set of photos.

There appears to be a definite sequence to the condition as evidenced by the slides. First, a "roughing" or blue appearance around the eye, then swelling with acute lesions on the eyelid. This is followed by a scabbing over and then loss of tissue leaving a notch or a flap. The lesion is not unlike that one might see in a torn eyelid from a physical force such as barbwire, protruding nails, etc., in large animals.

There may be one or several lesions of varying degrees. In the case I am most familiar with a very high percentage of the birds show the condition.

While the possibility that this may be a physical injury does exist, I believe that some infectious agent is involved and thus am seeking help -- not necessarily because of any severe economic effect, but more as a curiosity.

In the last few flocks housed, we have noted less lesions. Originally we had planned to use Erythromycin on these flocks as that has been used in the East; however, the feed mill supplying this particular flock did not have the necessary permits to use this drug.

We eventually used 100 grams Tetracycline on the flocks on an intermittent basis. When the T_2 toxin lesions became severe, we switched to a product containing a zinc proteinate (ZinPro) at the rate of two pounds per ton of feed. After this the lesions occurred less frequently.

From comments I have heard from others, this condition appears to be observed more frequently; whether this is due to people looking for it or an acutal increase in number is entirely speculative.

As a matter of fact, whether this is economically significant or not may be questioned; however, in the flocks I am speaking of, we have seen an increase in early production as the incidence of eye lesions have decreased.

SYNDROME DEL OJO SEMICERRADO
(Eye Notch Syndrome)

Esta presentación es principalmente un llamado por ayuda en el diagnóstico de una condición bastante rara, que se ha notado en varias parvadas en los ultimos años.

Hace aproximedamente dos años que estaba viajando por Este de los U.S. y me freguntaban acerca de una lesíon particular en el ojo que se estaba observando en las parvadas, principalmente en el estado de Neuva York. Debo admitir que pense que era una situación peculiar de la cual no sabiá nada.

Finalmente tube la oportunidad de observar esta condición en dos o tres parvadas en esos lugares. Se le llamaba "Syndrome del ojo semicerrado" (Eye Notch Syndrome) por la qente de ese Lugar y como yo no pensé en otro mejor nombre que ponerle continue usando el mismo termino que ellos.

Los parvadas que vi eran de lineas competitivas, y sin necesitar decirlo y me regresé diciendo "gracias a dios nosotros no tenemos esta condición."

Regresé a Iowa sintiendome bien, pero luego visité una granja comercial a poca destancia de Des Moines donde observe la misma condición en el ojo a la que había observado en el Este.

Esta granja tiene seis casetas identicas. Ellos producen sus propias pollonas en la granja pero separada de las ponedoras. Las panedoras las tienes en jaula de un solo piso y tienen un programa de nutricion y vacunación muy adecuados. El umico problema que es consistene en la granja son acaros. Las ponedoras estan libres de Mycoplasma y no han tenido ningun problema en particular con la exepcion de una consisten baja (mas bajo de lo ecferado) en la maxima produccion esperada de un dos tres porciento. La producción toal de huevo durante el periodo de posutra esta en el standard o arriba del standard. Ellos han tenido problemas seguido con la toxina T_2 que les da lesiones en la boca de las gallinas.

Las pollonas se crian separadamente con una programa de vacunacion stardard para Viruela, Newcastle, Bronquitis y Encephalitis aviar. Los logros en la cranza de pollonas son sobre el 96% y no se han observado lesiones en los ojos durante el crecimiento de las pollonas.

Las pollonas se cambian a las casetas de postura a las 19-20 semanas de edad y las lesiones del ojo se hacen evidentes a las 22 semans de edad. Estas transparencias fueron tomadas por el Dr. Barnes, quien tabien tomó muestras para analizar en la Universidad Estatal de Iowa. A pesar de un trabajo intensivo que hicieron con las muestras no se encontro ningun agente etiológico que pudiera causar esta condition, pero lograron un buen paquete de fotos sobre este problema.

Parece que esiste una definitiva sequencia del problema como puede observarse en las transfarencias. Primero un "colorete" o una apariencia azul alrededor del ojo despues inflamación con lesiones agudas en el parpado. Esto es seguido por encostramiento y pérdida de tejido dejando una muesca o una aleta. Esta lesion no se parece a la que puede verse en un parpado rasgado por una fuerza física como la producida por alambre, depuas, clavos, etc. en grandes animales.

Puede haber una o varias lesiones en differented gradosñ en el caso que yo conozco más un gran porcentaje de las aves muestran este problema.

La posibilidad que esto pueda deberse auna lesion fisica existe, pero creo que algun agente infeccioso esta involucrado y es por lo que estoy buscando ayuda, no porque esista una sevéra pérdida económica per mas bien por curiosidad.

En las eultimas parvadas encasetadas, nosotros hemos notado menos lesiones. En un principio planeamos usar erytromycina en estas parvadas, como se ha hecho en el este, sin embargo la planta de alimento que nos suple no tiene los permisos para usa esta droga.

Eventualmente usamos 100 gramos de teracycline en estas parvadas como una base intermitente. Y cuando las lesiones de la toxina T_2 se hacen serveras, cambiamos a un producto que contiene proteinato de Zinc (ZinPro) en una cantidad de dos libras por tonelada de alimento. Despues de esto las lesiones ocurren menos frecuentemente.

De commentarios que he escuchado de otra gente, este problema parece que se ha estado observando más frecuentemente, puede ser que ahora la gente este buscando más por este problema o que realmente este aumentando es enteramente especulación.

De hecho se puede poner a duda de que esto sea económicamente significante, sin embargo, en las parvadas en la que yo he estado hablando homos visto un aumento en la produccion de huevo cuando las lesiones en el ojo se disminuyen.

COMPARISON OF TISSUE CULTURE NEUTRALIZATION AND ELISA PROCEDURES FOR INFECTIOUS BRONCHITIS

Z. Garcia, DVM, MPVM, PhD and R. A. Bankowski, DVM, PhD
Department of Epidemiology and Preventive Medicine
School of Veterinary Medicine
University of California
Davis, California 95616

Abstract

Two serological tests, the virus neutralization (VN) in tissue culture using a tissue cell adapted virus and the enzyme linked immunosorbent assay (ELISA) test were compared to detect antibodies against Massachusetts 41 and Connecticut 46 strains of Infectious Bronchitis Virus (IBV).

The VN test was conducted in wells of microplates by the usual procedure. The two strains of IBV were adapted to induce CPE in 24 hours in chicken embryo kidney cells (CEKC) which required 20 serial passages.

The ELISA test was carried out using partially purified virus following ultracentrifugation of each strain of IBV as antigen.

The ELISA test detected higher geometric mean antibody titers (GMT) than the VN test against both strains of IBV. One hundred and four serum samples taken at 1, 3.5, 9.5, 22, 24, 26 weeks of age of a flock of chickens which were vaccinated with the Mass strain three times and the Conn strain of IBV two times during the growing period showed a better antibody response with the Conn 46 than with the Mass 41 IBV strains.

When the chickens were 35 weeks of age a sample of 60 birds were challenged with either the Conn 46 or Mass 41 strains of IBV at the University of California Animal Resources Facilities. The 20 serum samples from each group tested by VN test before challenge and 20 days after challenge showed that the birds in both groups had a marked but a higher anamnestic response with the homologous challenge virus strain than with the heterologous virus strain. The ELISA test showed similar results with the same serum samples when tested with the homologous strain but slight cross reactions were found with the heterologous virus strain.

The serum samples from the unchallenged control group showed no change in GMT with either test or IBV strain.

Resumen

Dos pruebas serologicas, la prueba de suero neutralizacion de virus (NV) en tejido de cultivos usando virus adaptado a cultive de tejidos y la prueba de inmuno adsorcion con enzima conjugada (ELISA) fueron comparadas para la detecion de anticuerpos en contra de las cepas del virus de la Bronquitis Infecciosa, Massachusetts 41 y Connecticut 46.

La prueba de NV se llevo a cabo en microplatos con 24 orificios por el metodo usual. Las dos cepas de Bronquitis Infecciosa fueron adaptadas para producir efecto citopatico (CPE) en 24 horas en celulas embrionarias de rinon de pollo y requirieron 20 pases seriados.

La prueba de ELISA se llevo acabo con virus parcialmente purificado por ultracentrifugacion de cada cepa y asi usado como antigeno.

La prueba de ELISA detecto mayor media geometrica de anticuerpos que la prueba de NV en contra de las dos cepas de Bronquitis Infecciosa.

Ciento cuatro muestras de suero tomadas a la 1, 3.5, 9.5, 22, 24, 26 semanas de edad de una parvada de aves que fueron vacunadas con Mass 41 tres veces y Conn 46 dos veces durante el periodo de cremiciento mostraron mejor respuesta de anticuerpos con la cepa de Conn 46 que con la cepa de Mass 41.

Cuando estas aves tenian 35 semanas de edad, una muestra de 60 aves fueron desafiadas con la cepa de Mass 41 y otra con la cepa de Conn 46 en las instalaciones do la Universidad de California. Veinte muestras de suero do cada grupo fueron probadas antes y 20 dias despues del desafio con las cepas virales, la prueba de NV mostro que las aves de ambos grupos tuvieron una marcada respuesta anamnesica con la cepa viral homologa pero no con la cepa viral heterologa. La prueba de ELISA mostro similares resultados con los mismos sueros cuando fueron probados con la cepa homologa de desafio, pero una ligera reaccion cruzada fue encontrada con la cepa viral heterologa.

Las muestras de suero del grupo control no mostraron cambio en la media geometrica de anticuerpos con las dos pruebas ni con las dos cepas.

MICRONEUTRALIZATION TEST FOR INFECTIOUS LARYNGOTRACHEITIS

G. G. Meza, D.V.M.
Vineland Laboratories Inc.
Vineland, New Jersey 08360. U.S.A.

Abstract

The technique that is described, allows to quantify the circulating antibodies against Infectious Laryngotracheitis. The cytopathic effect, in chicken kidney monolayers, grown in microtiter plates, can be macroscopically seen by fixing the cells with neutral formalin and staining them with crystal violet.

The test is easy to perform, it is more sensitive, uses less space and costs only about one tenth in comparison to the one that uses especific pathogen free chicken embryos.

PRUEBA DE MICRONEUTRALIZACION PARA LARINGOTRAQUEITIS INFECCIOSA.

Resumen

La técnica que se describe permite la cuantificación de los anticuerpos circulantes contra la Laringotraqueitis Infecciosa. El efecto citopatógeno del virus de Laringotraqueitis, en los monoestratos de células de riñon de pollo, cultivados en placas para microtitulación, se hace evidente macroscópicamente mediante la fijación de las células con formalina neutra y su posterior tinción con violeta cristal.

El método es sencillo de realizar, más sensible, utiliza menos espacio y su costo es aproximadamente de solo la décima parte, comparado con la prueba que usa embriones de pollo libres de patógenos específicos.

MICRONEUTRALIZATION TEST FOR INFECTIOUS LARYNGOTRACHEITIS.

Introduction

Infectious Laryngotracheitis (LT) is a disease generally restricted to certain areas, where it is endemic and some times caused by very virulent virus strains. Only in these areas vaccination is permitted, but almost never the immunity level against LT is tested because of its difficulties: the challenge procedure is too risky and the virus neutralization test that could be performed in Specific Pathogen Free (SPF) embryos is too laborious and expensive.

Materials

The technique described uses chicken serum aseptically collected (3) and inactivated at 56° C for 30 minutes, diluted 1/10 in media 199. Infectious Laryngotracheitis virus adapted to grow in chicken kidney cells (CKC). A suspension of CKC: 1 ml of packed cells in 200ml of growth media (media 199, 80%; Tryptose Phosphate Broth, 10%; Fetal Calf Serum, 10%; 100 u per ml of Penicillin, 100 mcg per ml of Streptomycin and 0.25 mcg per ml of Amphotericin B) all obtained from Grand Island Biological Co., Grand Island, New York. When frozen-reconstituted CKC were used, they were processed as previously described (4). Plastic disposable MicroTest II tissue culture plates with lid, 96 flat bottom wells per plate are obtained from Falcon Products, Oxnard California. Micropipettes delivering 0.025 ml and 0.050 ml per drop are obtained from Cooke Engineering Co., Alexandria, Virginia. 10% buffer neutral formalin solution is prepared by mixing 100 ml of Formaldeheide 37%-40%, 4.0 gr. of Sodium Phosphate monobasic, 6.5 gr. of Sodium Phosphate dibasic and 900 ml of deionized or distilled water. Crystal Violet 0.5% solution in deionized or distilled water.

Procedure

Two-fold virus dilutions are prepared in media 199. The unknown serum is diluted 1/10 in media 199. An equal amount from each virus dilution is mixed in a separate sterile test tube with an equal amount of the serum dilution and allowed to react at room temperature (approx. 25°C) for 45 minutes to 1 hour. During this time 1 day old CKC are prepared by decapitation and bleeding of the chickens, removing the kidneys and mincing them into 2mm pieces, trypsinization is carried out for 10 minutes for 2 or 3 times. After each trypsinization the supernatant, with cells in suspension, is collected and the trypsin inactivated by adding 10% of Fetal Calf Serum to the total volume of supernatant. After 3 or 4 washes with media 199, the cells are resuspended in growth media as previously described. After 45 to 60 minutes, 0.050ml of the serum-virus mixture from each dilution are transferred to 5 virus neutralization wells. 0.025 ml of the diferent virus dilutions are transferred to 5 virus titration wells. 0.050 ml of the growth media is inoculated into the growth media control wells. 0.025 ml of the unknown 10% serum dilution is tranferred to the serum toxicity control wells and finally 0.025 ml of the cell suspension is added to all the wells except the growth media control wells.

The plates are incubated for 4 to 5 days (3), fixed with 10% Buffer Neutral Formalin solution for 2 or 3 minutes, which is discarted before staining the cells with 0.5% Crystal Violet solution for 5 minutes, the plates are rinsed with tap water and allowed to dry at room temperature.

Results

Always the normal growth and color of the monolayers are given by the cell control wells, the appearance of the serum toxicity control wells should be the same as the cell controls. The highest dilution with cytopathic effect will give the titer of the virus as well as the titer of the serum. At least a four-fold difference between both titers will indicate that the serum is immune.

Discussion

The test is easy to perform and reliable, uses very little space in comparison to the neutralization test done in SPF embryos and it is inexpensive. This work indicates that microneutralization in CKC is the choice technique to test the immunity level against LT.

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STIMULATION OF INCREASED RESISTANCE TO TURKEY CORYZA BY COLONIZATION WITH TEMPERATURE SENSITIVE MUTANTS OF ALCALIGENES FAECALIS.

Marcus M. Jensen, Ph.D. and Douglas S. Burke, B.S., Dept. of Microbiology, Brigham Young University, Provo, Utah 84602

Temperature sensitive (ts) mutants of Alkaligenes faecalis, that grew slowly at 42°C, but grew well at 30°C, readily colonized the nasal mucosa of turkeys when administered intranasally or in drinking water. No signs of coryza were seen in exposed turkeys and little, if any, colonization of the trachea occurred. Colonization of the nasal mucosa lasted 3 to 4 weeks and induced moderate (1:2 to 1:512) levels of serum antibodies. When challenged after 6 weeks with a virulent strain of A. faecalis, no signs of coryza developed and no colonization with the virulent strain occurred in these turkeys. Control turkeys inoculated with the virulent strain became heavily colonized and showed marked signs of coryza.

Field trials were carried out in which about 100,000 turkeys, ranging in age from 2 to 20 weeks, in 15 flocks, were exposed to a ts mutant by the drinking water route. Five to 6 week old birds receiving heavy doses (over 50 million bacteria per bird) experienced the highest rate of colonization and developed moderate levels of serum antibodies. No depression or other adverse reactions were seen in the vaccinated turkeys. Twenty-four turkeys were removed from one flock 7 weeks after having been vaccinated and were fully resistant to challenges with a virulent strain of A. faecalis.

ESTIMULACION DE UNA RESISTENCIA CRECIENTE CONTRA LA CORIZA DE LOS PAVOS POR COLONIZACION CON MUTANTES SENSIBLES A LA TEMPERATURA DEL ALKALIGENES FAECALIS.

Resúmen.

Mutantes sensibles a la temperatúra (S.T) del Alkalígenes faecalis , que crece lentamente a 42°C, pero que se desarrolla adecuadamente a 30°C, colonizan facilmente la mucosa nasal del pavo cuando se inocula por vía nasal o en el agua de bebida. No se observaron signos de coriza en los pavos expuestos y existió una mínima invasión de la tráquea.
La colonización de la mucosa nasal dura de 3 a 4 semanas e induce niveles moderados (1:2 a 1:512) de anticuerpos séricos. No se observáron síntomas de Coriza ni tampoco de colonización, cuando se hizo un desafío con una cepa virulenta de A. faecalis 6 semanas despúes de la inoculación, en dichos pavos. Pavos testígos que fuéron inoculados con la cepa virulenta, enfermáron severamente mostrando marcados signos de coriza.

Se lleváron a cabo pruebas de campo con 100,000 pavos de 2 a 20 semanas de edad agrupados en 15 parvadas los cuáles fuéron expuestos a la mutante S.T. en el agua de bebida. Los animales recibiéron altas dosis (arriba de 50 millones de bacterias por ave) sufriéron 5 a 6 semanas más tarde el más severo grado de colonización desarrollando niveles moderados de anticuerpos séricos. No se observó depresión u otras reacciones adversas en los pavos vacunados. Se separáron 24 pavos de 1 parvada 7 semanas después de haber sido vacunados - mostrandose totalmente resistentes a los diferentes desafios con la cepa virulenta de A. faecalis.

Traducción: cortesía del Dr. Miguel A. Márquez, R.

A respiratory disease of turkeys, variously referred to as the acute respiratory disease syndrome, rhinotracheitis, or coryza, and hereafter referred to as coryza, has been shown to be caused by the bacterium Alcaligenes faecalis.(1,2)

The objectives of this study were to: (a) develop temperature-sensitive (ts) mutants of A. faecalis that would not grow well at the internal body temperature (41-42 C) of turkeys' (b) determine if these ts mutants would colonize the cooler tissues of the nasal mucosa; (c) determine if this colonization induced immunity against the virulent strains of A. faecalis; and (d) determine if such mutants could be used to vaccinate turkeys in the field.

Materials and Methods

Bacterial Isolates: Two isolates of A. faecalis from turkeys with coryza were used. One designated isolate N, was obtained from Dr. D. G. Simmons, North Carolina State University. The other designated isolate U, was isolated in Utah.

Induction of ts Mutants: The procedure used by Shimizu (3) was followed. Plates containing 15 to 150 colonies were replica plated onto two fresh plates and one plate was incubated at 37° C and the other at 42° C. Colonies that were absent or smaller at 42 C were selected as ts mutants. All mutants were derived from A. faecalis isolate N.

Colonization of Turkeys: In Expt. 1, five ts mutants were selected for colonization studies and were designated 2A and 2P, 1, 2, and 87. Each mutant strain was inoculated intranasally into six 8-day-old Broad White turkeys; each bird received between 10^5 to 10^6 bacteria suspended in 0.1 ml of saline. In addition, six turkeys were inoculated with the virulent isolate N, and 12 turkeys were held as uninoculated controls. Each group of six birds was housed in isolation cages. All bacteria used for inoculations were grown at 30° C for 36 hours.

Swabs were taken from the nasal mucosa and trachea of each turkey just before exposure to the inoculum and at 3 to 4 day intervals over the ensuing six weeks. A. faecalis isolates were identified by standard procedures. Mutant strains were identified by their impaired growth at 42° C. The degree of colonization was reported on a scale of 0 to 4. Signs of infection were reported on a scale of 0 to 4 based on the relative amount of mucus present in the trachea and nasal cavity.

In Expt. 2, 19-day-old turkeys were exposed to ts mutant 87 via drinking water. Four groups of six turkeys each were used. Groups A, B, and C were exposed for 24 hours to water and received doses of 800, 80, and 8 million bacteria per bird, respectively. Group D served as uninoculated controls. Nasal and tracheal swabs were collected at three to four day intervals starting at day seven. Housing and other experimental procedures were the same as used in Expt. 1.

Challenge with Virulent Strains: Forty-two days after exposure to the mutant and virulent strains of A. faecalis, all groups of turkeys from Expt. 1, including the controls, were placed in a common pen and were exposed twice to virulent A. faecalis of isolate N. Following these challenges, nasal and tracheal cultures were taken at weekly intervals for an additional 4 weeks.

Thirty-one days after exposure to mutant 87, three birds from each group of Expt. 2 were placed in pen A and the other three from each group were placed in pen B. The turkeys in pen A were challenged for 24 hours to drinking water that contained the virulent isolate N. Birds in pen B were challenged in a similar manner to the virulent isolate U. Nasal and tracheal swabs were taken at weekly intervals and analyzed for A. faecalis. Starting at about seven days after challenge to the virulent isolates and continuing for about 3 weeks, the turkeys in both pens were inadvertantly deprived of water for four to five hours from mid-morning to mid-afternoon each day during warm weather.

Serologic Tests: Blood samples were collected at 36 days in Expt. 1 and at 28 days in Expt. 2. Serum antibody levels were measured by an agglutination test. Field Trials:

Fresh cultures of mutant strain 87 were used as the vaccine. Fifteen turkey flocks were selected between July 28 and Sept. 8, 1979, that had experienced no known outbreaks of coryza. Base line blood samples and bacterial cultures were collected. The vaccine was administered via drinking water. The length of exposure to the vaccine varied from 4 to 24 hours. Concentrations of vaccine varied from 5 million to 1 billion bacteria per dose, and the ages of turkeys varied from 2.5 to 22 weeks. Eleven flocks received one exposure to the vaccine and 4 flocks were vaccinated twice. At about weekly intervals, nasal and tracheal cultures were examined for the presence of strain 87 or wild strains of A. faecalis. Blood samples were periodically collected.

Challenge of Field Vaccinated Turkeys: Twenty-four 13-week-old turkeys were removed from flock 8 (see table 1) 7 weeks after having been vaccinated and were challenged twice, 12 days apart, to virulent A. faecalis, isolate U. The challenge doses were via drinking water that contained about 10^5 bacteria per ml and was the only water available to the turkeys for 8 hours. Two control groups were used. The first control group consisted of twelve 13-week-old turkeys taken from a flock that reportedly had experienced no coryza; these turkeys were exposed to the same double challenge as the vaccinated turkeys. The second control group consisted of five 6-week-old turkeys that had been raised in isolation; they were exposed to only the second challenge. Nasal and tracheal cultures were collected before challenge and at 3 to 7 day intervals after challenge and were analyzed for the presence of virulent A. faecalis. Serum samples were collected from each group just before their first challenge.

Results

In Expt. 1, definite colonization of the nasal mucosa was produced by all ts mutants except 2A. Birds exposed to the virulent strain were heavily colonized in both the nasal and tracheal mucosa and had extensive mucus on these tissues. Mutants 1 and 2 induced slight signs of coryza. Moderate serum antibody levels were seen after five weeks in the birds that were well colonized. When challenged at 42 and 43 days with the virulent isolate N, those turkeys that had been colonized with ts mutants 1, 2, 2P and 87 showed immunity.

In Expt. 2, the nasal mucosa of each exposed turkey was well colonized by seven days and was cleared of the mutant strain by 21 days. No signs of infection were seen in any of the colonized turkeys. Antibody titers ranging from 1:4 to 1:128 were seen in the exposed turkeys at four weeks. When challenged at 31 days after exposure with either the N or U virulent isolate, the unexposed control turkeys became colonized. During the period of water stress, some colonization by the virulent isolates was seen in all three experimental groups and a slight increase in the amount of mucus was seen. No differences were seen in the responses to the N and the U isolates.

The results of the field vaccination trials are summarized in Table 1. The first series of vaccinations (flocks 1-4) used low doses (5-7 million bacteria per bird) of vaccine. Colonization rates with the vaccine strain was 50% or less. Within 2 to 5 weeks after vaccination, flocks 1 to 4 became colonized with wild strains of A. faecalis; signs of coryza were slight or absent. Flocks 5 to 8 were given vaccine doses of 65 to 90 million bacteria; the 5 and 6-week-old turkeys were 90 to 100% colonized with the vaccine strain, while about 50% of the 13-week-old birds were colonized. The 20-week-old turkeys of flock 9 were mostly refractary to colonization even with the extra large dose of vaccine that was given. No colonization occurred in the 2 1/2-week-old turkeys of flock 10; however, at the time of vaccination, the nasal mucosa of these turkeys was heavily colonized with E. coli.

Flocks 11, 13, 14, and 15 were vaccinated twice.Moderate levels of colonization occurred each time in flocks 11, 13, and 14 and after the second vaccination, most birds had detectable antibody levels. Flock 15 was colonized with both the mutant and wild strains of A. faecalis. Flocks 4 and 12 were possibly breaking with coryza at the time of vaccination and the wild strain was able to out compete or interfer with colonization with the vaccine strain. No depression or other adverse affects were noted as a result of the vaccination.

The field vaccinated turkeys from flock 8 were completely resistant to colonization when exposed to virulent A. faecalis. The field controls were also resistant. Both groups were positive for serum antibodies at the time of the first challenge. The second control group, which had no previous antibodies, became heavily colonized with the virulent strain.

Table 1. Response of turkey flocks in the field to oral administration of coryza vaccine

			First Vaccination		Second Vaccination	
Flock No.	Dosage (Million/bird)	Age (Weeks)	Ratio[a] Colonized	Ratio[a] With Antibodies[c]	Ratio[a] Colonized	Ratio[a] With Antibodies[c]
1	5.3	6	4/8	n		
2	6	12	2/8	n		
3	7	5	2/8	n		
4	6	5	1/8[b]	n		
5	65	13	4/8	4/8 (1:3)		
6	65	5	8/8	8/8 (1:12)		
7	90	6	7/8	7/8 (1:6)		
8	90	6	7/8	8/8 (1:13)		
9	1000	20	1/8	n		
10	180	2.5	0/8	n		
11	180	2.5	5/8	6/8 (1:14)	3/8	5/7 (1:8)
12	250	3.5	0/8[b]	n	n	n
13	50	3.5	3/8	6/8 (1:12)	7/8	8/8 (1:10)
14	50	3.5	3/8	7/8 (1:15)	4/8	5/8 (1:3)
15	100	3.5	5/8[d]	6/8 (1:4)	0/8	6/8 (1:3)

a = No. of birds with positive results/No. of turkeys sampled
n - not determined
b = colonized with wild strain
c = average titer in parenthesis ( ), antibodies measured from 2 to 5 weeks after vaccination
d = 2 were colonized with wild strain

Discussion

Four of the 5 mutants of A. faecalis evaluated in Expt. 1 were able to produce moderate to heavy colonization of the nasal mucosa. Strains 1 and 2 also produced light colonization of the trachea and induced slight clinical signs as measured by the amount of mucus in the nasal cavity. Strains 2P and 87 produced slight, if any, colonization of the trachea and caused no detectable clinical signs of coryza. All birds that became colonized developed antibodies and were resistant to infection when challenged with a virulent strain of A. faecalis. This study measured only serum antibodies, presumably IgG, while protection against this infection, which involves basically the respiratory mucosa, would be expected to result primarily from secretory IgA. While IgA was not measured, it is assumed that antigenic exposure through colonization of the mucosal tissue would stimulate a strong IgA response as well as the measured IgG response.

Strain 87 was selected for further study as it induced immunity in colonized birds, caused no signs of coryza, and was a two-step mutant from the original virulent strain. Expt. 2 demonstrated the feasibility of colonizing turkeys with strain 87 via drinking water. The degree of colonization was not greatly influenced by the number of bacteria present in the water over the range of 10^5 to 10^7 per ml. Those receiving the heavier dose had higher levels of antibodies, but showed no increased protection against challenge with the virulent isolates. No differences were seen in the degree of protection against the N and U isolates of A. faecalis. The induced immunity appeared to be relative, as when the vaccinated birds were subjected to the stress of daily periods of water deprivation, moderate levels of colonization by the virulent isolates occurred and was accompanied by slight signs of infection.

Flocks of turkeys in the field were successfully immunized when vaccinated by the drinking water route with strain 87. Doses of 50 to 90 million bacteria given to 5 to 6-week-old birds gave the best results. Older turkeys were poorly colonized by the vaccine strain and some younger turkeys may have been refractary to the vaccine strain due possibly to heavy colonization with *E. coli*. Turkeys 2.5 to 3.5-weeks-of-age, when vaccinated twice, responded moderately well to both vaccinations.

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UP-DATE ON INFECTIOUS CORYZA

R. Yamamoto, Ph.D.
Department of Epidemiology & Preventive Medicine
School of Veterinary Medicine
University of California
Davis, California 95616

ABSTRACT

Using the rapid plate agglutination test, Page classified Haemophilus paragallinarum into three serological types--A, B, and C. Further studies showed that these types shared common antigens because agglutination test antigens prepared from one serotype could detect antibodies in chickens vaccinated or infected with another seotype. Whether bacterins prepared form one type would protect against another type has been an important and somewhat of a controversial question. Recent studies from Japan fron the laboratory of Dr. Kune have shown that stains of serotypes A and C may possess encapsulated organisms with mucoid, iridescent colonies or nonencapsulated organisms with smooth to rough noniridescent colonies. The former were pathögenic and the latter were nonpathogenic for chickens. Antigenic analysis of the iridescent colonies revealed a type-specific surface antigen (Li for type A and L2 for type C), and three common antigens (L3,H1 and HS) shared by serotypes A and C. Protective immunity was conferred by the type-specific surface antigen which correlated with the production of agglutinins to this antigen. While Kine was unable to characterize his isolates of sertype B, Hinz of Germanu reportes his findings of both mucoid and rough colony forms of this serotype. Bases on this newer information on the antigenic makeup and dissociation potential of H. paragallinarum, the search for highly immunogenic strains for bacerin production should be facilitated.

ACTUALIZACION SOBRE LA CORIZA INFECCIOSA

Utilizando la prueba rápida de aglutinación en placa, Page clasificó *Haemophilus paragallinarum* en tres tipos serológicos: A, B y C. Estudios posteriores demostraron que esos serotipos comparten antígenos comunes debido a que las preparaciones de antigenos de un serotipo para la prueba de aglutinación pueden detectar anticuerpos en aves vacunadas o infectadas con otro serotipo. De lo que resulta una pregunta importante y de controversia¿Podrían las bacterinas preparadas con un serotipo proteger contro otro serotipo?.

Los estudios recientes en Japón realizados en el laboratorio de Dr. Kime - han demostrado que cepas de serotipos A y C pueden poseer organismos encapsulados de colonias mucoides tornasoladas y organismos no encapsulados de - colonias lisas y no tornasoladas. Las primeras colonias resultaron patógenas y las últimas apatógenas para gallinas.

El análisis antigénico de las colonias iridiscentes revelaron un tipo de antígeno específico en la superficie (L1 para tipo A y L2 para tipo C), y 3 antígenos comunes (L3,HL y HS) compartidos por los serotipos A y C.
La inmunidad fue conferida por el antígeno superficial específico del tipo la cual fue paralela a la producción de aglutininas contra este antígeno. Kume no pudo caracterizar sus aislamientos de serotipo B, Hinz de Alemania reportó sus estudios tanto de colonias rugosas como de colonias mucoides del serotipo B. Basada en esta nueva información de la composición antigénica y de potencial de disociación de H. paragallinarum, podría facilitarse la investigación de cepas inmunogénicas para la producción de bacterinas.

Traduccion : Cortesia del Dr. Carlos Lopez Cuello

Using the rapid plate agglutination test, Page (6,7) classified Haemophilus paragallinarum (gallinarum) into three serological types — A, B, and C. Further studies showed that these serotypes possessed common antigen(s) in that antigen prepared from one serotype could detect antibodies (agglutinins) in chickens infected or vaccinated with another serotype (5). Whether bacterin prepared from one serotype would protect against another serotype has been an important and somewhat controversial subject. Page's earlier studies suggested that significant cross protection existed between different serotypes (7), while our studies indicated that protection induced by bacterins was quite mono (serotype) specific (5). To some extent, the study of Rimler and Davis (8) has helped to resolve this question. They showed that chickens which had recovered from infection with one serotype were immune to reinfection with another serotype; on the other hand, inactivated organisms in the form of bacterins tended to confer protection against the homologous serotype only. Thus, inactivated organisms seemed to be lacking in antigen(s), quantitatively and/or qualitatively, which were possessed by living organisms in conferring cross protection between different serotypes.

Several laboratories have been investigating the antigenic (serologic) similarities and differences between serotypes of H. paragallinarum, and the relationship between such antigens to protective immunity (1,2,3,4). The results from these laboratories are essentially similar, but reports from the laboratory of Kume (4) seem most complete and revealing. These workers observed that strains of serotypes A and C when grown on a special agar medium, developed encapsulated organisms with mucoid, iridescent colonies. Such colonies were visualized most readily in cultures incubated not longer than 8 to 10 hours. Within 4 to 6 passages on agar, the mucoid colonies were observed to dissociate to nonencapsulated organisms with smooth to rough noniridescent colonies. The iridescent colony types were pathogenic and the noniridescent ones were nonpathogenic for chickens.

Antigenic analysis of the iridescent colonies from serotypes A and C revealed a type-specific, heat labile-trypsin sensitive surface antigen (designated L1 for serotype A and L2 for serotype C). In addition, three common antigens were shared by the iridescent colonies of serotypes A and C — a heat labile-trypsin sensitive surface antigen (L3), a heat labile-trypsin resistant somatic antigen (HL), and a heat stable-trypsin resistant somatic antigen (HS). The surface antigens, L1, L2, L3, were presumed to be capsular polysaccharide in nature.

Protective immunity was conferred by the type-specific surface antigen (L1 and L2); thus, for example, protection conferred by a bacterin prepared from serotype A was specific for serotype A and was correlated with the detection of agglutinins to the type-specific antigen. While Kume did not observe cross protection between serotypes, I would like to suggest that the surface L3 antigen shared by serotypes A, C and presumably B may be an important immunogen in conferring cross protection between serotypes following natural infection as reported by Rimler and Davis (8). It would be of interest to determine whether antibodies to this antigen is present in chickens exposed to the live organism.

The noniridescent colony types of serotypes A and C possessed only the HL and LS antigens; they lacked the surface antigens, L1, L2, and L3, and thus were nonpathogenic for chickens.

While Kume was unable to characterize his isolates of serotype B because they lacked type-specific L antigens (personal communication), Hinz (1) reported on finding mucoid and rough colony forms of serotype B. He observed that the pathogenic mucoid form of serotype B dissociated in the chicken to the nonpathogenic rough form in the late stages of the disease (17 days). He also observed that the rough variant may dissociate to the mucoid form in the chicken to produce disease.

In summary, it is of interest to note that to this date, no new *H. paragallinarum* serotypes other than that described by Page in 1962 (6,7) have been found. Type A seems to be a common serotype in the United States, Japan and Germany. In addition, based on extensive surveys, serotypes B and C appear to be frequently isolated, respectively, from Germany (1) and from Japan (4). While speculative, there is no reason to believe that serotypes other than A, B and C exist in Mexico and in Central and South America. Finally, it is probable that within a particular serotype, some strains exist that are more immunogenic than others; based on the newer knowledge of antigenic makeup and dissociation potential of *H. paragallinarum*, studies concerning the detection of such strains for bacterin (vaccine) production should be facilitated.

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COMPARATIVE STUDY OF COMERCIAL BACTERINS AVAILABLE IN MEXICO FOR THE CONTROL OF HAEMOPHILUS GALLINARUM.

J.A. Arias, A. Ortiz, J. López M. y F. Galindo.
Department of Veterinary Medicine, ENEP-Cuautitlán
Apartado Postal 130, Cuautitlán Izcalli, Estado de México.

This is a comparative study of the effectivity of different types of bacterins, propagated in different mediums and using different strains in its preparation, two of them were produced in México and one abroad.

The experimental group consisted of 9300 Harco layers, 16 weeks old, this group was divided in four lots, three of them with 3000 hens each one to test the bacterins and the fourth group of 300 hens as non-immunized control. The bacterins were applied to the layers twice with an interval of four weeks between each application using the intramuscular route and at the doses recomended by the fabricant.

Four weeks after the second immunization the birds were challanged with live *H. gallinarum* to compare the protection conferred by the different bacterins in each one of the groups. The protection was better against clinical signs and drop in egg production with a bacterin propagated in broth produced in México; there was not a statistically significant difference in mortality or feed intake.

ESTUDIO COMPARATIVO DE BACTERINAS COMERCIALES DISPONIBLES EN MEXICO PARA EL CONTROL DE HAEMOPHILUS GALLINARUM.

En el presente estudio se compara la efectividad de diferentes tipos de bacterinas, propagadas en diferentes medios y utilizando diferentes cepas para su elaboración; dos producidas en el país y una en el extranjero.

Se utilizó un lote experimental de aproximadamente 9300 aves raza Harco de 16 semanas de edad, estos animales se dividieron en cuatro lotes, tres de tres mil aves cada uno para la aplicación de cada una de las bacterinas en prueba y un cuarto grupo como control sin inmunizar. Las bacterinas se aplicaron por via intramuscular dos inyecciones con intervalo de cuatro semanas entre cada una y a la dosis indicada por el productor.

Cuatro semanas despues de la última inmunización se expusieron las aves a un *H. gallinarum* viable para evaluar la protección conferida por las diferentes bacterinas en cada uno de los lotes. La protección fue mejor contra signos clínicos y baja en la producción de huevo en una bacterina propagada en caldo producida en México, no se encontro diferencia estadísticamente significativa en cuanto a mortalidad o consumo de alimento.

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El uso de bacterinas ha sido uno de los métodos más comunes para el control de la Coriza Infecciosa, éstas han sido preparadas a partir de organismos cultivados en embrión de pollo e inactivadas con formalina; sin embargo trabajos más recientes han demostrado la superioridad de productos desarrollados en caldo, inactivados con mertiolate y con hidroxido de aluminio como adyuvante (1, 3). El tipo de cepa y la concentración final de *H. gallinarum* en las bacterinas han sido problemas para la elaboración de productos comerciales efectivos (4) por lo que es necesario determinar los serotipos prevalentes en las areas donde se piense utilizar bacterinas, asi como asegurar una mínima concentración de 100 millones de organismos por mililitro del producto (1). En el presente estudio se compara en una granja comercial la efectividad de diferentes productos existentes en el mercado nacional en relación a la protección contra: baja en la producción de huevo, consumo de alimento, mortalidad y signos clínicos.

Animales experimentales.- Se trabajó con un lote de 9300 aves raza Harco de 16 semanas de edad, las cuales no presentaron ningun problema respiratorio aparente durante su crianza. Estas aves se dividierón en cuatro lotes

conforme lo descrito con anterioridad.

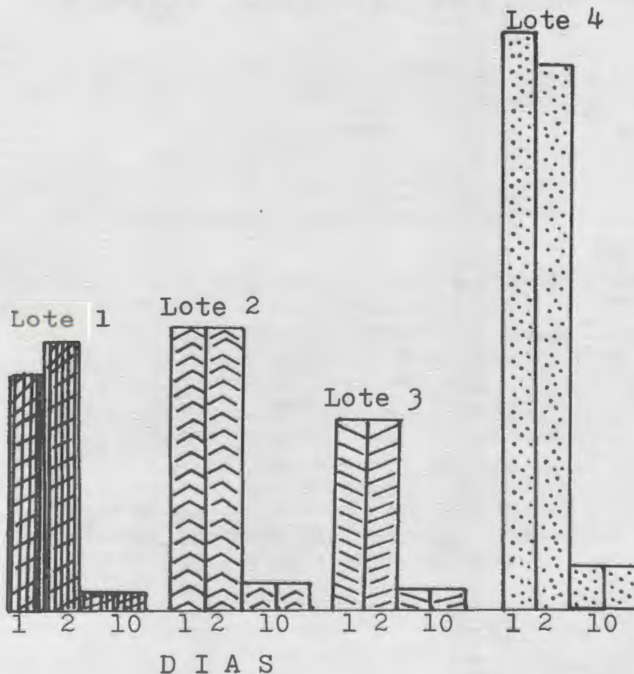
Bacterinas.- Se utilizaron tres tipos de bacterinas:
1) Bacterina propagada en huevo de fabricación nacional, utilizada en la región suroeste de México con excelentes resultados. Las cepas utilizadas para su elaboración son cepas de campo que se aislaron en esa región.
2) Bacterina propagada en caldo a base de las cepas Modesto y W, producida en el extranjero por una firma de prestigio internacional, se han publicado numerosos reportes a cerca de la efectividad de este producto.
3) Bacterina propagada en caldo a base de las cepas Modesto y 17756 de fabricación nacional.

Estas bacterinas se aplicaron vía intramuscular en la pechuga, a las dosis recomendadas por el fabricante a las 16 semanas y se repitió la dosis cuatro semanas despues. Para evaluar la inmunidad conferida por las diferentes bacterinas se llevó a cabo el desafío o exposición controlada, utilizando un inóculo de H. gallinarum vivo y patógeno cuyo título era 10^8 UFC (unidades formadoras de colonias). Despues del desafío y evaluación preliminar se procedio a tratar la parvada con antibioticos vía parenteral y en el agua de bebida.

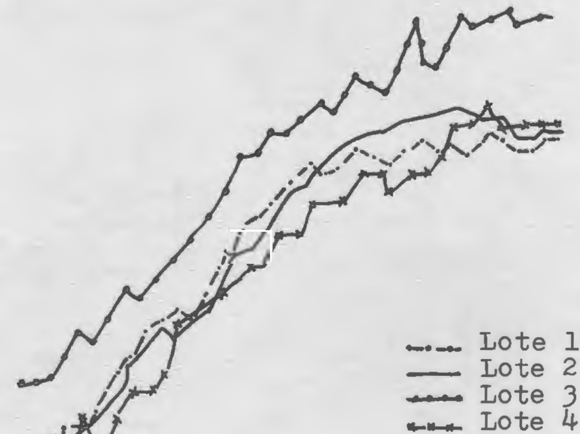
Método de Evaluación.- Se estableció una escala del cero (sin signos clínicos) al tres (inflamación grave y exudado) de acuerdo a la severidad de los signos clínicos (2) y se califico de acuerdo a esta escala un número significativo de aves tomado al azar y se obtuvó un promedio general de afectación (PGA) multiplicando el número de aves afectadas por el número de la escala que les corresponde, luego se sumaron los totales de los cuatro grados de afectación de cada lote y se dividió entre el número total de aves evaluadas por lote. Las diferencias en los PGA fueron estadisticamente significativas para todos los lotes ($P > 0.05$), ver gráfica 1, asi mismo se puede observar en esta gráfica que el lote cuatro (testigo) no solo fue el más afectado, sino que su recuperación fue más lenta aún despues del tratamiento.

Las diferencias en porcentaje de postura diaria fueron significativas durante todo el experimento solo para el lote tres (gráfica 2) y solamente existe diferencia en unos cuantos días despues de la exposición entre el lote control y los lotes uno y dos ($P > 0.05$). No se encontraron diferencias significativas en cuanto a mortalidad y consumo de alimento.

GRAFICA 1
GRADO DE AFECTACION PROMEDIO AL PRIMERO, SEGUNDO Y DECIMO DIA.



GRAFICA 2
PORCENTAJE DE POSTURA DIARIO



El mayor grado de protección conferido por la bacterina propagada en caldo de fabricación nacional puede ser debida a diferencias en el título o espectro antigenico de las cepas utilizadas para su elaboración por lo que consideramos necesario que exista un control oficial para ambos factores; con respecto al producto elaborado en embrión de pollo es importante mencionar que el inoculo para el desafio se obtuvó en el mismo laboratorio donde se produce esta bacterina por lo que pudo haber sido favorecida por un desafio homólogo.

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EXPERIENCES WITH BACTERINS IN THE CONTROL OF INFECTIOUS CORYZA IN SONORA.

L. Guzman M., Rosa Ma. Fabela de G. and C. Garrido M.

Laboratorio de Patología Aviaria.
Asociación de Avicultores del Yaqui A.C.
Sufragio Efectivo 616 Nte.
Cd. Obregón, Sonora.
MEXICO

The rsults of the use of three different bacterins in the control of infectious coryza in light pullet and layers, under field conditions in more than ten million -- birds, during the past 15 years indicate:

a) Pullets of 1 to 15 weeks of age are protected at least 50% against -- symptoms, by the bacterin.

b) In layers, the bacterin does not protect more than 50% against symptoms but the disease becomes more benign and consequently it is easier to - treat it.

c) The egg drop in layers with bacterin always is less than 10% (4-8 % - average), in contrast, the egg drop in layers without the bacterin is of 30-40%.

EXPERIENCIAS CON ALGUNAS BACTERINAS EN EL CONTROL DE LA CORIZA INFECCIOSA EN SONORA.

Se describen las experiencias obtenidas bajo condiciones de campo, con el uso de tres diferentes bacterinas contra la coriza infecciosa en pollas de reemplazo y ponedoras de raza liviana.

En síntesis, los resultados de estos estudios llevados a cabo en los pasados 15 años con más de 10 millones de aves, demuestran que:

a) La bacterina es capaz de proteger pollas menores de 15 semanas de edad en por lo menos 50%, contra síntomas del padecimiento.

b) En gallinas en producción la bacterina, por lo general, no confiere -- una protección mayor del 50% contra síntomas; sin embargo, la enfermedad se hace más benigna y por consiguiente fácil de tratar.

c) La baja de producción de huevo en ponedoras con bacterina no es mayor de 10% (4 a 8% promedio), contra 30 a 40% en aves sin bacterina.

EXPERIENCIAS CON ALGUNAS BACTERINAS EN EL CONTROL DE LA CORIZA INFECCIOSA EN SONORA.*

L.GUZMAN M., ROSA MA. FAVELA DE G., C. GARRIDO M.
Laboratorio de Patología Aviaria
Asociación de Avicultores del Yaqui, A.C.
Sufragio Efectivo 616 Nte.,
Cd. Obregón, Sonora, México.

El Estado de Sonora, localizado en el Noroeste de México posee un clima semidesértico con escasas lluvias, veranos muy calurosos, inviernos benignos y con variaciones en la temperatura durante 24 horas hasta 25°C. En la parte sur del Estado, se localizan tres Valles: El de Guaymas, el del Yaqui y el del Mayo y es esta área en donde se han hecho estos estudios, cuyos resultados daré a conocer.
La población avícola en los tres valles comprende: Progenitoras, Reproductoras, Ponedoras y Pollas Livianas; así como un pequeño porcentaje de pollo de Engorda.
En los últimos 20 años al número total de aves se ha incrementado de 1 a 10 millones, sin embargo,gracias a la buena voluntad de los avicultores así como a la moderna tecnología, los problemas infecciosos que en los años de 1963-1967, eran verdaderamente severos se tienen actualmente bastantes controlados. Entre estos problemas infecciosos la coriza ha ocupado y sigue ocupando uno de los primeros lugares.
El Hemophilus paragallinarum fue aislado por primera vez en nuestro laboratorio en 1964, y en 1965 iniciamos ensayos de campo con bacterinas elaboradas por nosotros. Por supuesto que la composición de estas bacterinas ha cambiado en los pasados 15 años.
Así por ejemplo de 1965 a 1973 la bacterina que producíamos era en embrión de pollo siguiendo la técnica de Page y Clark. Este tipo de bacterina la usamos durante 9 años. En 1974, basándonos en los estudios de Yamamoto y Matsumoto,logramos producir una bacterina en un caldo sintético; ésta bacterina nos proporcionó una mayor protección que la producida en embrión de pollo.
En los últimos 2 años hemos venido haciendo experimentos con una bacterina emulsionada en aceite a la cual hemos incorporado virus de Newcastle con el objeto de inmunizar a las aves contra los dos padecimientos.
Hemos dividido en dos grupos los resultado de la pruebas de campo con estas bacterinas; el primero corresponde a aves en desarrollo hasta las 20 semanas de edad y el segundo, a aves en producción.
En el cuadro No.1 hacemos un resumen de las granjas donde han ocurrido los brotes de coriza en pollas de diferentes edades.
Debo hacer notar que por las condiciones de aislamiento de estas granjas la coriza no ha representado un problema muy serio y que solo en aquellas donde hay reincidencias del problema, el uso de la bacterina se ha hecho a temprana edad (4 semanas).
En las granjas de crianza no se ha presentado la infección, la aplicación de bacterinas por lo general se hace a las 11, 14 y 18 semanas.

CUADRO No. 1

BROTES DE CORIZA EN GRANJAS COMERCIALES

GRANJA		FECHA DEL BROTE	EDAD AL BROTE		% MORBILIDAD
Mónica	39	1977	9.3	semanas	.08
Buáraje	37	1977	9.0	"	2.00
Teresita	25	1977	4.0		1.00
Teresita	27	1977	11.6	"	2.00
Teresita	29	1977	19.0	"	2.00
Milagro	40	1978	10.0	"	0.05
Alamos	40	1978	10.0	"	1.00
Mónica	45	1978	7.0	"	1.00

Como cité antes, la coriza infecciosa en el pasado, produjo sus mayores estragos en las granjas de postura y específicamente en un núcleo de aves que nosotros llamamos Tepeyac-Rancho Grande, en el Valle del Yaqui, donde exis-

*Parte de este trabajo fue dado a conocer en la Conferencia de la APYZAN del 7 de agosto de 1977 en Guaymas, Son.

ten aproximadamente un millón de aves en un área de 5 Km^2 y en donde desde su origen su suscitaron los más serios problemas de Coriza y ERC, por lo tanto fue y sigue siendo nuestro núcleo de aves donde hemos hecho la mayoría de estos estudios. Por lo años de 1967-68 se utilizaba la bacterina en embrión de pollo pero observamos que los resultados obtenidos, no eran lo bastante satisfactorios porque además del uso de la bacterina se decidió hacer la exposición controlada, empleando en este caso el mismo Hemophilus usado en la producción de la bacterina.

Por algún tiempo este sistema dió buenos resultados pero solo en granjas que tenían parvadas de una sola edad; en las granjas con parvadas de diferentes edades no dió los resultados deseados; en otras palabras, la bacterina producida en el embrión de pollo cuando se aplicaba en granjas con parvadas de una sola edad y posteriormente expuestas ya sea por brote natural o artificial con el Hemophilus antigénicamente similar a la de la bacterina, los brotes de coriza eran bastantes benignos y cedían con ligeros tratamientos. También nos dimos cuenta que los brotes de coriza en ponedoras que habían recibido tres dosis de bacterina, la producción de huevo no se afectaba grandemente. Los resultados de los últimos tres años con el empleo de la bacterina de caldo, en la mayoría son muy alentadores como se puede apreciar en el cuadro No. 2 y gráficas.

CUADRO No.2
BROTES DE CORIZA EN GRANJA TEPEYAC

GRANJA	EDAD AL BROTE	FECHA	DIFUSION	% BAJA POSTURA
J-4	28 semanas	Nov. 1979	Lenta	2.8
J-7	29 "	Nov. 1979	Rápida	9.0
J-9	29 "	Nov. 1979	Lenta	6.0
J-11	28 "	Nov. 1979	Lenta	8.5
B-10	34 "	Nov. 1979	Rápida	12.0
B-1	62 "	Dic. 1979	Lenta	5.0
IN-IA	28 "	Mayo 1978	Lenta	2.5
IN-IB	28 "	Mayo 1978	Lenta	2.5

En 1974 en base de los resultados de nuestras pruebas de laboratorio en donde llegamos a observar protección hasta de 100% en exposición con el mismo Hemophilus que empleabamos en la bacterina; nos entusiasmamos tanto que para 1975 iniciamos el siguiente programa a nivel de granja.

1.- Reducir al mínimo el número de edades en cada granja.
2.- Aplicación de 3 dósis de bacterina como mínimo en pollas susceptibles antes de las 20 semanas de edad.
3.- Repetir la dósis cada 3 meses si la enfermedad no hacía su aparición.
4.- Suspender totalmente la exposición controlada que se venía haciendo y por supuesto extremar las medidas de control sanitario.

Desde el año de 1975 hasta la fecha el empleo de este programa en las granjas bajo nuestro control ha dado los siguientes resultados.

Períodos de hasta un año y medio sin un solo brote de coriza infecciosa.

En granjas donde ha aparecido un brote en aves que ha recibido 3 ó 4 dósis de bacterina el padecimiento ha sido muy benigno sin tener complicaciones y en algunos casos ni siquiera ha sido necesario el tratamiento a base de antibióticos. En los casos donde ha ocurrido un brote en aves en plena producción ésta no ha sido disminuida más de 12% aún en parvadas con 90% de producción (ver gráficas).

Si bien es cierto que no hemos logrado erradicar el problema aún en parvadas donde se han empleado hasta 5 dosis de bacterina, creemos haber dado un gran paso en el control de la coriza ya que en la actualidad ésta ha dejado de ser el problema que antaño causaba bajas de 30 o 40% en la producción durante varias semanas.

Estamos conscientes de que hace falta una mayor investigación para el mejoramiento de las bacterinas, tanto en su proceso de elaboración como en la selección de cepas de Hemophilus que sean altamente antigénicas.

LIVING WITH INFECTIOUS CORYZA

Gregg J. Cutler, D.V.M., M.P.V.M.
Camarillo, CA 93010

ABSTRACT

In March, 1979, Infectious Coryza was discovered in one house of 85,000 birds at Egg City. Within 60 days all forty houses comprising over 2.5 million birds had undergone the disease.

Initially, depopulation of the infected index flock was considered. The rapid spread of the disease made this method of control impractical.

The effect on each flock varied with age and *Mycoplasma* status. Young, Mg-free birds suffered little egg production loss. Older birds suffered moderate loss. Those birds going through Mg infection suffered most severe egg production loss (up to 40% decrease).

The Egg City program for control of coryza consists of producing coryza, *Mycoplasma gallisepticum*, and *M. synoviae*-free pullets which are well immunized against the major viral diseases. These birds are exposed to virulent *H. gallinarum* organisms by aerosol at the time they are housed at Egg City (20 weeks of age). Very good egg production is obtained at relatively low protection cost.

RESUMEN -- VIVIENDO CON LA CORIZA INFECCIOSA

En marzo de 1979, se descubrió que había Coriza Infecciosa en una Caseta con 85,000 aves de postura en "Egg City". En un periódo de 60 días, en todas las cuarenta casetas que forman porte de este complejo avicola con un total de más de 2.5 millones de aves, se habían contaminado con la enfermedad.

Inicialmente se consideró la posible eliminación de la parvada afectada, pero la rapída diseminacion de la enfermedad hizo que este método resultara imprắctico.

Los efectos de la enfermedad en cada una de las parvada, fue en aumento de acuerdo como la edad y el estado de Mycoplasma aumeutaban. Parvades jovenes libres de Mycoplasma, fueron las menos afectados en cuanto a la baja de proudccín de huevo. Parvadas mas viejas tubieron moderadas pérdidas. Y aquellas parvadas que estaban con infeccion de Mycoplasma fueron las que sufrieron una más severa baja en la produccion de huevo (bajas hasta de un 40%).

El programa de "Egg City" para el control de Coriza consiste primeramente en la produccion de pollonas libres de coriza, Mycoplasma gallisepticum. Y Mycoplasma synoviae, e immunizadas contra la mayoria de las enfermedades virales. Una vez que estas pollonas son encasetadas en "Egg City" (a las 20 semanas de edad), se exponen a una cepa viruleuta de Haemophillus gallinarum usando el método por aerosol.

May buena produccion de huevo es obtenida a un costo de produccion relativamento bajo.

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The Egg City Ranch in Moorpark, California, was free of infectious coryza for nearly eight years. In March, 1979, coryza was discovered in one house of 85,000 birds. Within 60 days all forty houses comprising over 2.5 million birds had undergone infectious coryza.

The suspected index case was observed in the corner of one house on the main level of Egg City on March 15, 1979. There are 12 flocks on this level separated only by roadways approximately 9 meters wide. Workers had been in the index house making repairs on each of the previous several days. This was done after the same crew had unloaded pullets (20 weeks old) on each morning. The infected house was boarded up and plans were made to depopulate the flock.

The next morning (March 16, 1979) coryza was observed in the house receiving pullets which were unloaded by the crew working in the suspected index case house. The two houses are separated by about 100 meters. Coryza was also diagnosed in an adjacent house and in a house 500 m distant from the other houses. At this time depopulation efforts were abandoned.

A telephone call on March 16th was made to the pullet-growing facility 200 km to the north. There was no report of any respiratory disease.

Three days later (March 19th) coryza was seen in three additional houses at the Egg City Ranch. There was also a call from the pullet-growing ranch indicating that there was a respiratory disease present. Later that day it was diagnosed as infectous coryza.

It is suspected that the index case might have been in the pullet house. Birds infected at the grow ranch were brought to Egg City and then transmitted to what was originally thought of as the index house. An alternate mechanism of introduction may have been directly into the suspected index house and then transmitted back to the growing ranch via pullet-moving equipment.

The effect of infectious coryza on each flock varied widely depending on age and *Mycoplasma gallisepticum* status. Young, Mg-free flocks suffered respiratory disease but very little in the way of lost egg production.

Flocks in the process of turning Mg-positive suggered the most severe loss of egg production. Drops of 40% were the rule in this group.

Flocks late in the pullet cycle suffered moderate losses in egg production (approximately 15%).

Older force-molted flocks suffered production losses similar to late pullet flocks. Early force molted flocks had delayed egg production of three to seven days but no decrease in production.

Antibiotics, increased protein, and increased vitamins-minerals were tried in varying combinations. Erythromycin shoed significant benefit but only at higher than legal levels. Increased protein and vitamins-minerals treated birds did better than controls but not at statistically-significant levels.

The Egg City program for coryza control consists of producing replacement pullets free of coryza, Mg and Ms and vaccinated against Marek's disease, Newcastle disease, bronchitis, Pox, and laryngotracheitis. These pullets are then exposed at approximately 20 weeks of age to virulent *Haemophilus gallinarum* when delivered to Moorpark. The Haemophilus is cultured in embryonating eggs, diluted with phosphate buffered saline and applied by aerosol. This program produces good immunity, very good egg production, and at relatively low cost.

EGG DROP FROM CORYZA SUPERIMPOSED ON M.S.?

J. Butterweck, D.V.M., M.S. and E. Kerr, B.S.
Poultry Antigen Laboratories
1512 East "D" Street
Ontario, California 91764

A large layer ranch in Southern California experienced a progressive, slow spreading upper respiratory syndrome with a concurrent 10-15% drop in egg production over a 6 month period. A preliminary diagnosis of coryza was made based upon wide spread coryza problems at that time, this ranch's past problems with coryza, and isolating Hemophilus gallinirum from a few affected birds.

However, upon further veterinary investigation it became apparent that M.S. was likely, the primary agent based upon (1) epidemiological nature of the syndrome, (2) flock going M.S. positive, (3) isolation of M.S. and no other pathogens, (4) response and lack of response to specific antibiotic treatments, (5) experimental challange studies and (6) history of never having a break with this particular coryza vaccine when properly used.

¿ BAJA DE POSTURA POR CORIZA INFECCIOSA SUPERPUESTA CON M. s.?

En una granja de ponedoras localizada en California del Sur se notó un problema de vías respiratorias altas, de difusión lenta el cual produjo una baja del 10-15 % de la postura en un lapso de 6 meses. El diagnóstico preliminar fue de Coriza Infecciosa, basados en la amplia difusión de la enfermedad en la región, los antecedentes de coriza en parvadas anteriores y el aislamiento de Haemophilus gallinarum de aves enfermas.

Sin embargo, al investigar en forma más profunda este problema, se hizo aparente que Mycoplasma synoviae (M.s.) fue probablemente el agente causal primario. Estos datos se apoyaron en las siguientes observaciones: (1) la naturaleza epidemiológica del problema, (2) la respuesta positiva de la parvada en forma paulatina a la aglutinación contra M.s., (3) respuesta a terapia antibiótica específica, (5) pruebas experimentales de desafío (6) ausencia de signos clínicos a la reacción post vacunal contra coriza.

Traducción: cortesía del Dr. Armando Antillón Rionda

INTRODUCTION OF PROBLEM

In 1979, a 200,000 Southern California layer ranch that had been depopulated (all in-all out) experienced an upper respiratory disease syndrome with a concurrent 15% drop in egg production spreading through the open housing slowly. A preliminary diagnosis of coryza was made based upon a wide spread epidemic of coryza in Southern California at that time, this ranch's past history of coryza problems, and an isolation of Hemophilus gallinirum from a few of the sick birds. The birds had been vaccinated with an attenuated modified live coryza vaccine by water when the birds were on the pullet ranch at 20 weeks of age. Three weeks later they were moved to the lay ranch and blood samples were tested and were positive for coryza.

Upon thorough investigation it became apparent that Hemophilus gallinirum(H.G.) was not the primary agent and that Mycoplasma synoviae (M.S.) was likely the primary infectious agent. M.S., as a primary cause of a disease syndrome, is becoming recognized in increasing frequency. (1), (2).

FIELD INVESTIGATION WITH LABORATORY DIAGNOSTIC AIDS

1. Epidemiological Nature of Syndrome. The syndrome spread slowly. It took over 3 months to spread through the open housing units. In many cages not all the birds were affected at the same time. As a result, egg production in each housing unit dropped 10 to 15% that lasted over 6 weeks. Uncomplicated coryza spreads rapidly resulting in a pronouced egg drop. Mycoplasma infections spread slowly with a moderate production drop as occured in this syndrome.

2. Symptoms. Etiological agents that cause upper respiratory diseases are not easily distinguishable based upon symptoms and post mortems. In this syndrome there were wet noses and sneezing (rhinitis and sinusitis), coughing and wheezing (trachealitis) and dry matter on the eyelid (bilaterial conjunctivitis and harder gland infection). Based upon post mortems the pathogensis appear to progress from a rhinitis and conjunctivitis to deeper structures of the respiratory system. As secondary agents became involved the incidence of cull birds with air saculitis increased. Some birds had swollen hocks and foot pads (synovitis).

3. Serology. Sister flocks raised on different ranches remained M.G. and M.S. seronegative. This problem flock gradually went M.S. positive and the serological conversions correlated well with the spread of the syndrome. M.S. rapid plate tests, in one section of the open-house unit, went from 8/23 (33%) to 15/20 (75%) positives within a 3 week period during the observed spread of the syndrome. The few M.G. positive samples were all M.S. positive which was interpreted as a cross reaction. H.I. to Newcastle disease were 1:640 and were interpreted as the flock was exposed to a field strain of Newcastle disease virus since there had been no booster Newcastle vaccinations within 8 weeks. Rapid plate tests for coryza were consistently positive from 23 weeks of age throughout the lay period. This was interpreted that the birds were and remained immunized to coryza.

4. Isolation of infectious agents. Innoculation of 40,10 day old embryonated eggs with exudate from the respiratory system of 5 infected birds resulted in isolation of M.S. from two of five layers. The chick embryos had swollen feet and hocks and thickened membrane with pearl-like plaques. On repeated attempts isolations of Staphyloccus and Hemophilus was made from a small number of birds. Since these bacteriological isdates were from less than 5% of the samples these were considered secondary agents and of no significance in the primary disease syndrome. Isolation of viruses and bacteria pathogens is common in healthy immunized livestock and poultry. Thus isolation of Hemophilus-gallinirum was probably made from immunized birds.

5. Experimental Challenges. Two sets of challenge experiments were performed. In the first, two cages, each with 20 twelve week chickens were vaccinated with Poultry Antigen Laboratories (P.A.L.) attenuated modified live coryza vaccine. Two weeks later they were caged with 5 twelve week non-vaccinated controls and 5 of the ranches' layers that had recently developed wet snotty noses. None of the vaccinated or controls developed wet snotty noses within one week.

In the second experiment, the yolk suspension of Hemophilus gallinirum ranch isolate was innoculated into the noses of 20 twelve week chickens that were vaccinated with P.A.L.s live coryza vaccine two weeks before and 5 non-vaccinated controls. One out of 20 and 4 out of 5 of the respective birds developed a wet nose within 24 hours and 4 days later they were all symptomless, indicating the Hemophilus gallinirum was mildly pathogenic.

6. Antibiotic treatments. Attempts were made to control this syndrome with antibiotics in the feed for 2 week periods. Erythromycin at 185 grams/ton, Tylosine at 50 grams/ton and Oxytetracycline at 200 grams/ton were used but only the Oxytetracycline resulted in a noticeable improvement in egg production. Erythromycin is recognized to control H.G.; Tylosine, M.G.; and Oxytetracycline, M.S.(3) This response would again point the finger at M.S.

7. Previous performance of vaccines. This flock had been vaccinated with P.A.L. modified attenuated live coryza vaccine by water route at 20 weeks of age and where used, this has controlled all field challenges of H.G. in California and Venezuela for over a year. Since this is a non-spreading vaccine, use of antibiotics two days before and seven days after post vaccination, and failure to neutralize the water chlorine with skim milk could all have a factor in reducing the effectiveness of the immunization program. Additional rapid plate agglutination tests for coryza were performed at 30 weeks of age. Twenty-eight of 29 were positive irregardless of disease syndrome status.

CONCLUSIONS

As with most field disease investigations, one is never sure he has all the facts. Further investigation and laboratory testing with proper interpretation might produce a more definitive answer. With this investigation we felt there was no doubt that coryza was not the cause of the syndrome. This was largely based upon what appeared to be an adaquate vaccination program thereby immunization with a highly effective proven program. Further tests did not show there should be any reason to doubt this assumption and suggest that M.S. was a likely cause of this syndrome.

Presently, in California, M.S. is being incriminated in more disease syndromes. We are presently working on a M.S. vaccine as an alternative to M.S. control.

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ESPIROQUETOSIS AVIARIA: COMENTARIOS GENERALES Y DATOS PRELIMINARES SOBRE SU REPRODUCCION EXPERIMENTAL EN PERIQUITOS AUSTRALIANOS.

RESUMEN: A.J. DaMassa, y H.E. Adler.

Se discute la espiroquetosis en una forma global, así como los problemas - comunmente encontrados en su diagnóstico. También se señalan los factores - ecológicos y biológicos que aseguran la supervivencia de Borrelia anserina en la naturaleza.
Los resultados obtenidos demuestran la susceptibilidad de los periquitos -- australianos (Melopsitaccus undulatus) a la infección por B. anserina. Hubo mortandad en cuatro de seis aves inoculadas con una combinación de B. anserina y la vacuna contra la enfermedad de Newcastle tipo B_1.
Así mismo, se demostró masiva espiroquetemia. Otras dos aves, inoculadas - en forma similar y tratadas con penicilina, mostraron lenta recuperación. - Finalmente, dos periquitos fueron infectados en forma directa con B.anserina al parasitarlos con garrapatas Argas (P) sanchezi. Ambas aves sufrieron la enfermedad recuperándose posteriormente.

Traducción: Cortesía del Dr. Armando Antillón R.

AVIAN SPIROCHETOSIS: GENERAL COMMENTS AND PRELIMINARY DATA ON THE EXPERIMENTAL DISEASE IN BUDGERIGARS.

SUMMARY:

The importance of avian spirochetosis on a global basis, together with the problems normally encountered in the diagnosis of the disease is discussed. Ecological, as well as biological factors ensuring the successful maintenance of B. anserina in nature is outlined.

Preliminary data is presented which conclusively demonstrates the susceptibility of budgerigars (Melopsittacus undulatus) to infection with B. anserina. Four out of six birds inoculated intranasally with a combination of B. anserina and B_1 type Newcastle disease virus died. A massive spirochetemia was demonstrated. Two budgerigars similarly exposed and showing similar clinical signs were treated with penicillin and slowly recovered. An additional two budgerigars were exposed to B. anserina by allowing single, infected A. (P.) sanchezi ticks to feed on them. Both developed spirochetosis, presented a similar disease pattern but recovered.

AVIAN SPIROCHETOSIS: GENERAL COMMENTS AND PRELIMINARY DATA ON THE EXPERIMENTAL DISEASE IN BUDGERIGARS

A. J. DaMassa, Ph.D. and H. E. Adler, D.V.M., Ph.D.
Department of Epidemiology and Preventive Medicine
School of Veterinary Medicine
University of California
Davis, California 95616

Avian spirochetosis, caused by Borrelia anserina has been known to affect many species of birds since its original description from geese in 1891 (14). In the Americas the disease has been described primarily from western U.S.A. from epizootics in chickens (1,5,13), turkeys (7,8,10,12), and mongolian pheasants (11).

Borrelia anserina is normally introduced into a susceptible bird population by certain Argasid or "soft" ticks belonging to the genus Argas. The outbreaks in U.S.A. have been initiated primarily by the feeding of Argas (P.) sanchezi (5). This tick species, together with A. (P.) radiatus, is the predominant poultry Argas in western U.S.A.; both also occur in Mexico (9). Argas ticks are true biological vectors of B. anserina but the spirochete may also be transmitted by certain other arthropods mechanically and only for a limited period of time (3).

On a worldwide basis avian spirochetosis remains as an important disease of domestic poultry. A considerable number of scientific notes are published each year by scientists from India, U.A.R., Bulgaria, U.S.S.R. and Israel, among others. In Northern Nigeria the condition is second only to Newcastle Disease as an important disease of poultry (15).

Regardless of the species of bird diagnosis normally depends on the demonstration of the organism in the blood. Diagnostic reagents are not available except in specialized laboratories in areas where the disease is prevalent. However, even these may be rendered useless because of the presence of diverse immunological and serological types of *B. anserina* (3). An additional compounding difficulty may arise in some species of birds because a spirochetemia may no longer be present by the time that clinical symptoms are observed (2).

Borrelia anserina is eminently adapted for survival in nature. Among the factors ensuring the survival of the spirochete are 1) ticks which normally transmit *B. anserina* may infest both domestic and wild birds, thereby assuring widespread dissemination of the arthropod, as well as providing opportunities to infect diverse bird species, 2) a single tick may remain infective for at least 4 years and has the capability to infect a new host at each feeding (3), 3) a single infected tick has been shown to infect many different species of birds. These are; chicken, turkey, White Pekin Duck, Japanese quail (*Coturnix coturnix japonica*), mongolian pheasants (*Phasianus colchicus mongolinus*), Chinese pheasants (*Phasianus colchicus torquatus*), Chuckar partridges (*Alectoris gracea*), and budgerigars (*Melopsittacus undulatus*)(2), 4) in nature, since *Argas* ticks are closely associated with the nest, infection of the young bird assures a more pronounced spirochetemia, for a longer period of time. This is so by virtue of the young birds immature immunological capacity. Thus the infection of the tick is thereby facilitated, 5) even if the degree of spirochetemia is low, *Argas* ticks appear able to concentrate the organism, thereby allowing transmission at a later time, particularly after 1-2 ecdyses (2), 6) female ticks have been shown capable of transmitting *B. anserina* transovarially for at least 45 months (3), and 7) the spirochetes within an infected tick are unaffected when the tick feeds on an immune bird, or on one whose bloodstream contains high levels of penicillin, or a combination of both (3).

Preliminary Note: Experimental Spirochetosis in Budgerigars.

Recently, we have witnessed a severe form of spirochetosis in a limited number of budgerigars exposed to *B. anserina* either by, 1) an intranasal instillation or 2) by allowing single, infected *A. (P.) sanchezi* ticks to feed on susceptible birds. The preliminary findings were:

1. Six adult budgerigars, both male and female, received an intranasal exposure to *B. anserina* mixed with a Newcastle Disease (B_1 type) vaccine. The total volume introduced was about 1 drop, delivered from a 22 gauge needle. Within six days all birds displayed clinical signs of depression, ruffled feathers and were somnolent and tremulous. Additionally, the dropping were watery, stringy, and bilious green in color. Blood smears revealed a low level spirochetemia on the sixth day post exposure which increased in number until too numerous to count four days later. Six days after the onset of symptoms 3 parakeets could no longer remain perched and one could no longer stand (i.e. moribund). These four died within 6 days after onset of symptoms. Two birds which received 10,000 units of penicillin G potassium intramuscularly on the first day following onset of clinical signs recovered fully. At autopsy the most prominent lesion was in the liver, which had multiple, round, well defined punctiform grey necrotic spots (1-2 mm diameter) distributed throughout the entire liver surface. Spleens were about 2-times normal size and only one out of four contained foci of infarction.

2. Two adult budgerigars were exposed to *B. anserina* by allowing a single, infected *A. (P.) sanchezi* tick to feed on each bird. The procedure used to expose birds to infected ticks is described elsewhere (3). The onset of clinical symptoms, watery, green droppings, spirochetemia, and the duration of the disease process was generally the same as the intranasally exposed budgerigars. However, both birds survived and appeared to recover faster than those exposed to *B. anserina* and Newcastle Disease. A period of about 3 weeks was required after the disappearance of a spirochetemia before signs of depression, general unthriftiness, and watery-green droppings disappeared.

Discussion

Insofar as we know this is the first report to establish that psittacine birds commonly referred to as budgerigars (*Melopsittacus undulatus*) are highly susceptible to infection with *B. anserina*. The only other psittacine known to be susceptible is the grey parrot, *Psittacus erithacus*. In this later case only a single parrot was involved which died of the disease (6).

As indicated previously the dual infection, i.e. with *B. anserina* and Newcastle Disease appeared to produce a more explosive disease process, although the number of birds involved preclude a precise analysis. This aspect needs to be studied in greater detail, which if true could explain losses due to *B. anserina* in adult birds (particularly chickens), since it is well established that older birds are increasingly more difficult to infect.

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GANGRENOUS DERMATITIS AND OTHER SKIN PROBLEMS IN CHICKENS

A. A. Bickford, V.M.D., Ph.D.
Department of Veterinary Pathology
University of Missouri
Columbia, Missouri 65211

Disorders of the skin are notoriously varied and difficult to characterize and classify in all species and the chicken is no exception. While this presentation will emphasize the condition known as gangrenous dermatitis (gangrenous cellulitis clostridial dermatomyositis) it is also intended to provide a brief overview of other skin conditions and to suggest a categorization of integumenary diseases based on pathogenetic features.

The skin is the first line of defense against many injurious agents. Because of the feather coat in birds the skin over most of the body is protected to some degree and both the epidermis and dermis is relatively thin and delicate compared to the skin of most mammals. In unfeathered areas (comb, wattles, facial skin, shanks and feet), however, the skin is much thicker with a remarkably increased degree of epidermal keratinization.

In general the surface keratinization, the close-knit cell layers of the epidermis, and the dermal collagen collectively present a very effective barrier to surface injury and invasion of surface pathogens. However, it must be remembered that the epidermal cells are subject to constant turnover - surface cells are sloughed or abraded away and are constantly replaced by new cells generated through mitotic activity in the basal cells of the stratum germinativum. Probably this factor, more than any other, ties the health of the integument to a healthy metabolic state in the bird. Any impairment of metabolism which disrupts normal mitotic activity will be reflected sooner or later in epidermal disease. Many nutritional deficiencies, stress, intoxications and endocrine disorders are implicated in this type of integumentary disease. Other metabolic disorders, however, influence the production of normal keratin, the integrity of the circulatory system, the uptake of nutrients by the cells of the integument or the status of collagen fibers in the dermis and subcutis, all of which are important factors in the maintenance of healthy skin. Finally, one should keep in mind that the integument is breached regularly by microorganisms of various types which enter through fissures, cuts, lacerations, etc. or which have the potential for independent invasion. Protection against these microorganisms is dependent on an effective inflammatory response and a healthy immune system. Defects in either will likely result in a more fulminant skin disease and may permit a serious septicemic infection.

With the background it is logical to establish several categories of skin diseases and to specify several disease examples as follows:

I. Uncomplicated physical damage to healthy skin

- picking injury (cannibalism)
- wire cuts and other mechanical injury
- thermal injury (burns, frostbite, etc.)
- caustic chemical injury

II. Uncomplicated infections of healthy skin

- superficial staphylococcal infection (wounds or injections site)
- classical fowl pox
- favus
- external parasites (pediculosis, acariasis)

III. Skin diseases reflecting a metabolic problem

- nutritional dermatitis (pantothenic acid, biotin, etc.)
- exudative diathesis (Vitamin E - selenium deficiency)
- xanthomatosis
- scabby hip syndrome
- anemia and hemorrhage
- ergotism

IV. Complex skin diseases reflecting infectious agents superimposed on underlying metabolic or circulatory defects or impaired inflammatory or immune responses

- -"infectious anemia - hemorrhagic syndrome"
- -fulminating bacterial or fungal infections
- -gangrenous dermatitis
- -vesicular dermatitis - staphylococcal infection

V. Neoplastic skin diseases

- -Marek's disease
- -squamous cell carcinoma

DERMATITIS GANGRENOSA Y OTROS PROBLEMAS DE LA PIEL EN POLLOS

Los problemas de la piel son notoriamente variados y difícil de caracterizar y clasificarlos en todas las especies, y los pollos no son una exepcion. Esta presentacion va a enfatizar más el problema conocido como dermatitis gangrenosa (celulitis gangrenosa, dermatomiositis clostridial) pero tambien intenta dar una ligera revisada a otros problemas de la piel y sugerir una categorizacion de las enfermedades integunentarias basados en caracterisiticas patogéneticas.

La piel es la primera linea de defensa contra muchos agentes injuriantes. Debido a la cubierta de plumas de las aves, la piel de la mayor parte del cuerpo esta protegida hasta cierto gradoñ pro tanto la dermis y epidermis es relativamente delgada y delicada comparada con la piel de la mayoria de los mamiferos. En las areas no cubiertas por plumas (cresta, barbillas, la piel de la cara, y las patas), la piel es mas gruesa y con un aumento muy notorio en el grado de keratinizacion epidermal.

En general la supervicie keratinizada, el fino tejido celular de las capas de la epidermis y el colageno derma todos juntos presentan una barrera muy efectiva para proteger la superficie contra lesiones e invasion de patogenos. Sin embargo, debe tenerse en cuenta que las celulas epidermales estan sujetas a una constante renovacion-las celulas superficiales se descaman y son reemplazadas por nuevas celulas generadas por la actividad mitótica de las celulas basales del estrato germinativo. Probablemente este factor, mas que coalquier otro, relaciona la salud del integumento con unsano estado metabólico en las aves. Cualquier altercaccion del metabolismo que interumpa una normal actividad mitótica va a reflejarse tarde o temprano en una enfermedad epidermal. Muchas deficiencias nutricionales, stres, intoxicaciones y desordenes endocrinos estan implicados en este tipo de enfermedad intergumentaria. Sin embargo, otros desordenes metabolicos tienen influencia en la produccion de una Keratina normal, la integridad del systema circulatorio, la toma de nutrientes por las celulas del integumento o el status de las fibras de colageno en la dermis y subcutisñ todas estos son factores muy importantes en el mantenimiento de una piel saludable. Finalmente, uno debe tener en cuenta que el integumento es violado regularmente por microorganismos de various typos las cuales entran a traves de fisuras, cortes, laceraciones, etc. y que tienen el potencial de invadir. La proteccion contra estos microorganismos es dependiente de una efectiva respuesta inflamatoria y un systema immune muy sano. Defectos en cualquiera de estos resultara en una fulminante enfermedad de la piel y puede conducir a una seria infeccion septicemica.

Con esta base es lógico establecer varias categorias de enfermedades de la piel y especificar varios ejemplos de enfermedades com sigueñ

I. Daño físico, de una piel sana sin complicacciones

- Lesiones por picoteo (canibalismo)
- Cortes por alambres u otras lesiones mecánicas
- Lesiones termales (quemaduras, congelamiento, etc.)
- Lesiones por químicos causticos

II. Infecciones de una piel sana sin complicaciones

- Infeccion superficial staphylococcica (sitio de heridas de heridas o injecciones)

- Clasico viruela aviar
- Favus
- Parasitos externos (acariasis, pediculosis)

III. Enfermedades de la piel que reflejan un problema metabólico

- Dermatitis nutricional (acido pantoténico biotina etc.)
- Diathesis exudativa (vitamina E - Deficiencia de selenio)
- Xanthomatosis
- Syndrome de la cadera costrosa (scabby hip syndrome)
- Hemorragia y anemia
- Ergotismo

IV. Enfermedades de la piel completas, reflejando agentes infecciosos superimpuestos o remarcando defectos metabolicos o circulatorios o alterando las respuestas inflamatoria e immunlogica.

- "Anemia infecciosa-syndrome hemorragico"
- Infecciones fulminantes bacterianas o fungales
- Dermatitis gangrenosa
- Dermatitis vesicular-infeccion staphylococcica

V. Enfermedades neoplasmicas de la piel

- Enfermedad de Marek's
- Carcinoma de las celulas escamosas

PASTY VENT OR VENT GLEET IN TURKEY BREEDER HENS

Arun K. Bahl, D.V.M., Ph.D*
Cuddy Farms, Inc.
P. O. Box 247
Marshville, NC 28103

The term "pasty vent" or "vent gleet" in turkey breeder hens has been suggested for a condition that resembles ulcerative cloacitis in chickens. The etiology of vent gleet has not been definitely determined. A few predisposing factors may have been identified. It occurs in young breeder hens at a proportionally higher rate than in hens in production for some period of time. Breeder hens get listless, unthrifty and show ruffled feathers. Birds with vent gleet show diarrhea-like appearance of posterior extremities and whitish encrustations of the feathers and the skin of the area. Gasping for breath, caseous plugs in the tertiary bronchi, unilateral or bilateral consolidation of lungs either due to CU strain of vaccine or earlier field exposure to Cholera may be one of the predisposing factors. High summer temperatures, high humidity, and dusty barn conditions during growing and breeding period may aggravate the problem.

BLENORRAGIA ANAL EN PAVAS REPRODUCTORAS.

Se presenta en pavas reproductoras teniendo como sinónimo el de "cloaca pastosa", siendo muy similar a la cloacitis ulcerativa de las gallinas. Su etiología no está bien estudiada, aunque se conocen algunos factores predisponentes. Es más común encontrarle en pavas jóvenes que empiezan su postura que en animales de mayor edad. Los animales nuestran apatía, falta de apetito y hay erizamiento de las plumas. Las plumas de la región peri-anal y - abdominal están mojadas con un material de tipo diarreico y hay formación de costras blanquecinas en la piel y plumas. Hay disnea y tapones caseosos en - los bronquios terciarios y hepatización pulmonar, se piensa que un factor -- predisponente pueden ser los brotes de Cólera Aviario en edad temprana, o -- bien la vacunación con la cepa CU de esta enfermedad. El problema puede ser más grave si las aves fueron criadas en época de veranos calurosos, con elevada humedad ambiental y abundante polvo en las casetas.

* Present address: Director of Poultry Health and Veterinary Services, Hudson Foods, Inc., P. O. Box 777, Rogers, AR 72756.

Traducción: Cortesía del Dr. Armando Antillón Rionda.

¿QUE ESTÁ PASANDO CON LA ASCITIS?

Jose Villaseñor M., M.V.Z., M.S.
Norwich Pharmaceuticals, Mexico City, Mexico
and
E. Rivera-Cruz, M.V.Z., M.S.
Consultor, Mexico City, Mexico

RESUMÉ

En años recientes se ha observado en algunos países de América, un aumento de ascitis en polo de engorda. Para este trabajo se realizó una encuesta en varios países del Continente, primcipalmente en México. Las conclusiones a las que se llegó fueron: a) La mayor altura sobre el nivel del mar agrava el problema y b) el invierno y las bajas temperaturas aumentan la incidencia de ascitis.

SUMMARY

In recent years in some countries of America an increase of ascitis in broilers has been noticed. For the present work, a survey on the conditions was done in several countries of the American Continent, particularly Mexico. The conclusions were: a) higher altitudes on sea level, aggravate ascitis and b) winter and low temperatures increase ascitis problems.

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En los últimos años se ha notado en algunos países de nuestro Continente, un aumento en la presentación de casos de ascitis en pollo de engorda. Su relevancia económica se pone de manifiesto al saber que en los lugares donde se diagnostica consistentemente, la mortalidad puede variar entre un 1 y un 30%, siendo generalmente aceptado, como promedio, un 4%.

Para llevar a cabo nuestro trabajo y por la nula investigación que sobre el tema se realiza en Centro y Norteamérica, realizamos una encuesta testimonial en algunos países productores de pollo de engorda de América y una revisión bibliográfica sobre la materia; la suma de estos elementos es lo que se presenta a continuación.

Lo que en este trabajo consideramos como ascitis, se caracteriza por lo siguiente: por lo regular los pollos afectados se encuentran en buen estado de carnes; disnea; plumas erizadas y plumaje opaco; cianosis de la cresta; dificultad para caminar ocasionada por acumulación de líquido en la cavidad abdominal, lo cual puede motivar un andar en posición de pinguino en las aves; hidropericardia en muchos casos; aumento de volumen y congestión del hígado, otras veces este órgano se presenta reducido de tamaño; riñones aumentados de tamaño.

Como causa de ascitis en las aves se han citado, entre otras: a) hipoproteinemias, b) intoxicaciones por sal común, c) intoxicaciones por aflatoxinas, d) intoxicaciones por compuestos clorinados, e) hipoxia ambiental debida a la altitud, f) intoxicaciones por crotalaria, g) intoxicaciones por grasas tóxicas.

El problema de ascitis ha sido observado en algunos países del Continente Americano. En Argentina, Brasil, Venezuela y Panamá no han diagnosticado la enfermedad, según testimonios de Colusi (Argentina)(1), Nakano (Brasil) (2), Fernández (Venezuela)(3) y Matzer (Centroamérica)(4). En Perú la enfermedad sí existe, pero la enorme mayoría del pollo de engorda se produce en la costa, en donde nuestro informante, el Dr. Cotlear(5) dice textualmente: "la enfermedad no constituye un problema de cuidado". En los demás países Centroamericanos y en los Estados Unidos de Norteamérica, si bien la enfermedad ha sido identificada, ésta no representa, a la fecha, una grave amenaza económica para el avicultor. Su diagnóstico es esporádico, según testimonio de Matzer (Centroamérica) y Villegas (EUA)(6).

En Colombia y México, la ascitis sí afecta a un número considerable de pollos de engorda por lo que constituye un problema que merma las utilidades del productor, según testimonio de Rodríguez y Hernández (Colombia)(7) y de

20 personas relacionadas con la industria avícola (México), referidos más adelante.

Por lo anterior y por la facilidad que para los autores significó obtener información en su propio país, de aquí en adelante trataremos el tema con datos obtenidos en México. Nuestros informantes en la República Mexicana fueron una veintena de médicos veterinarios y personas relacionadas con la producción avícola: Antillón, Armando; Balconi, Iván; Castellanos, Julián; Cuadra, Alejandro; Cuitún, Luis; Chávez, Rolando; Dorantes, Jaime; Estudillo, Jesús; Flores, Luis L.;Galván, Gabriel; Gómez, Daniel; Lazo, Luis A.; Mata, Miguel A.; Medina, Susano; Paredes, Francisco; Porter, Guillermo; Rosales, Juan M.; Rodríguez, Luis; Vallarino, David; Zendejas, Juan.

El vasto cuestionario que a ellos presentamos fue elaborado para investigar el perfil de la ascitis en México. Una vez analizadas sus respuestas, éstas fueron condensadas en 10 encabezados. Cabe aclarar que las respuestas a una pregunta fueron de lo más amplias y variadas, lo que ilustra sobre la complejidad de la materia.

Localidad.- Nuestra encuesta se realizó en 18 localidades del país.

Altitud.- En las localidades estudiadas encontramos un rango bastante amplio de altitudes sobre el nivel del mar: 2 a 2680 mts.

Temperatura.- Mínima -4°C; máxima 42°C.

Edad de presentación.- Se consideró que la enfermedad principia aproximadamente a las 4 semanas de edad.

Mortalidad.- El dato de mortalidad también es de lo más variado, seguramente influyendo en éste factores agravantes, sin embargo, en promedio debe ser del 4%.

Epoca del año en que se presenta.- La enfermedad se presenta durante todo el año, si bien es importante señalar que se agrava considerablemente en la época fría de otoño e invierno.

Etiología.- En lo que a etiología se refiere, nos dieron una lista casi interminable, que puede resumirse en causas de origen tóxico, infeccioso, nutricional y climatológico.

Qué se ha hecho para contrarrestarla.- Paralelo al origen de la enfermedad, ha sido el número inacabable de remedios que se han usado para disminuirla.

Resultados.- Negativos en la enorme mayoría de los casos.

Factores agravantes.- Entre los factores agravantes de la enfermedad los más comúnmente mencionados fueron los siguientes: enfermedades infecciosas, temperaturas bajas de invierno y altitud.

Nuestra inquietud para tratar de hacer luz sobre el complejo problema de la ascitis aviar, comenzó en los grandes centros de producción de pollo de engorda, notándose que en todos ellos había ascitis (cuadro No.1). Para complementar nuestra información, recurrimos a otros centros de producción, observándose con sorpresa la ausencia de ascitis en esos lugares (cuadro 1).

En nuestro país, desde el nivel del mar, hasta los 1140 mts. de altura, la enfermedad no tiene importancia económica. En tanto que a partir de los 1300 mts. sobre el nivel del mar, hasta los 2680, la enfermedad ocasiona pérdidas considerables en las parvadas.

Nuestra observación de que la altura influye en la presentación de la ascitis, coincide con los trabajos de Hall y Machicao, en Bolivia (8); Cueva y colaboradores (9); Pizarro y colaboradores(10) en el Perú y Hernández Vázquez en Colombia(11,12),quienes describen una ascitis, debida a hipoxia ambiental, caracterizada por insuficiencia cardiaca derecha a partir de una hipertensión pulmonar y recomiendan que para arribar a un diagnóstico de ascitis, por hipoxia, se observen las lesiones macro y microscópicas en el ventrículo y válvulas derechas del corazón, así como también lesiones hepáticas y renales; complementándose el diagnóstico con estudios sobre el número de eritrocitos, concentración de hemoglobina y hematocrito.

CONCLUSIONES

1.- La altura sobre el nivel del mar agrava el problema de ascitis.

2.- Cuando la temperatura disminuye por el invierno, el problema de ascitis aumenta.

C U A D R O 1

ALGUNAS LOCALIDADES DE MEXICO EN QUE SE INVESTIGO LA PRESENCIA DE ASCITIS

POSITIVO	NEGATIVO
CUAUTLA, MOR.	ACAPULCO, GRO.
JILOTEPEC, EDO. MEX.	CIUDAD OBREGON, SON.
QUERETARO, QRO.	CORDOBA, VER.
SAN LUIS POTOSI, S.L.P.	CULIACAN, SIN.
TECAMACHALCO, PUE.	HERMOSILLO, SON.
TEHUACAN, PUE.	MERIDA, YUC.
TEPATITLAN, JAL.	MONTERREY, N. L.
TOLUCA, EDO. MEX.	TIJUANA, B. C.
VILLA DEL CARBON, EDO. MEX.	TORREON, COAH.

Confrontando estas dos informaciones (cuadro 2) constatamos la influencia que ejerce la altura sobre el nivel del mar, en la presentación de la enfermedad.

C U A D R O 2

ALTITUD DE LAS LOCALIDADES ESTUDIADAS EN DONDE SE PRESENTA O NO LA ASCITIS

NEGATIVO		POSITIVO	
LOCALIDAD	ALTURA SOBRE EL NIVEL DEL MAR	LOCALIDAD	ALTURA SOBRE EL NIVEL DEL MAR
TORREON, COAH.	1140 Mts.	TOLUCA, EDO. MEX.	2680 Mts.
CORDOBA, VER.	600 Mts.	JILOTEPEC, EDO.MEX.	2525 Mts.
MONTERREY, N. L.	538 Mts.	VILLA DEL CARBON, EDO. MEX.	2200 Mts.
HERMOSILLO, SON.	237 Mts.		
CIUDAD OBREGON, SON.	100 Mts.	TECAMACHALCO, PUE.	2055 Mts.
CULIACAN, SIN.	34 Mts.	SAN LUIS POTOSI, S.L.P.	1877 Mts.
TIJUANA, B. C.	29 Mts.		
MERIDA, YUC.	9 Mts.	QUERETARO, QRO.	1853 Mts.
ACAPULCO, GRO.	2 Mts.	TEHUACAN, PUE.	1676 Mts.
		TEPATITLAN, JAL.	1559 Mts.
		CUAUTLA, MOR.	1291 Mts.

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INCUBATOR DEHYDRATION AS A CAUSE OF HIGH MORTALITY IN CHICKS AND POULTS

R. C. Fanguy, Ph.D., R. J. Terry, D.V.M. and W. F. Krueger, Ph.D.
Department of Poultry Science, Texas A&M University
College Station, TX 77840

One factor affecting the success of any poultry operation is the quality of the chicks or poults at the time of their placement in brooding pens. Since only a limited reserve of water and nutrients remain after hatching, it is of vital importance that feed and water be made available before any great degree of physiological stress manifests itself, particularly dehydration.

The time required for completion of embryonic development shows considerable variation, much of which is under genetic control. Selection studies have shown that genetic factors can shift hatching time as much as 42 hours, almost two days. Experimental evidence suggests that poultry breeders, in an effort to increase total percent hatch, have been selecting breeders whose eggs hatch early. Though not harmful in itself, this practice can cause considerable problems for the commercial hatcheryman if not compensated for as indicated by this study.

Field studies of commercial turkey operations show first week mortality from starvation and dehydration can reach 10-12%. Post-mortem examination of these starve-outs often shows a preponderance of males.

Controlled laboratory studies of these same commercial stocks reveal hatching of poults begins on the 25th day and is 50% complete by the end of the 26th day. Comparable studies involving commercial broiler sotcks show hatching to begin on the 18th day and to be 50% complete by the end of the 19th day. Using the hatch-placement schedule practiced by industry, this would mean that approximately 30% of the chicks and poults are in excess of 72 hours of age when placed in brooder pens. Our follow-up studies show that this 30%, which hatch early, experience a mortality in the order of 25-30%. This would account for the 10-12% total mortality observed in the field. Post-mortem examination of all poults shows males to predominate during the first half of the hatch which could explain the preponderance of males dying, in our field studies, during the first week of brooding.

Dehydration studies indicate chicks and poults begin to lose moisture immediately following hatch even though they remain in the incubator cabinet at 70% relative humidity. By the time the birds are 72 hours old, this water loss is in the order of 15% of their initial hatch weight. Poults delayed in placement longer than this often loose 30% of their hatch weight through dehydration. When placed in brooder pens these birds often fail to ingest feed or water.

To prevent excess dehydration it is necessary for commercial hatcherymen to monitor the progress of each hatch rather than rely on a rigid calendar schedule. It may also be necessary to pull some hatches twice if the total hatch time is prolonged. This often happens with hatching eggs stored for extended periods of time. In our study this phenomenon was also observed occasionally with fresh hatching eggs.

BAJOS NIVELES DE HUMEDAD EN LA INCUBADORA COMO CAUSA DE MORTANDAD ELEVADA EN POLLOS Y PAVIPOLLOS

R.C. Fanguy, R.J.Terry, y W.F. Krueger

RESUMEN:

La buena calidad de los pollitos es uno de los factores principales que intervienen en el éxito de una empresa avícola. Debido a la limitada cantidad de agua y nutrientes que posee el pollito al nacer, es muy importante que tenga fácil acceso al agua y alimento en las criadoras, de lo contrario se presentará deshidratación.

Existe variación en cuanto al tiempo requerido para un completo desarrollo embrionario del pollo. Por medio de pruebas se selección se ha podido variar el tiempo de incubación hasta 42 horas, es decir, casi dos días. Las empresas avícolas de reproductoras han venido experimentando con estirpes de gallinas cuyos huevos son de corta incubación; estas medidas se han hecho con el fin de aumentar el porcentaje total de incubación. Esta práctica, aunque no es negativa, sí ha causado problemas a las empresas que se dedican a la incubación.

Estudios de campo realizados en granjas de pavos, han señalado porcentajes de mortandad e inanición hasta de un 1-12%. El examen postmorten indica que los machos son más afectados.

Se han hecho estudios controlados de estas casas incubadoras, encontrándose que la eclosión en pavipollos comienza al vigésimo quinto día, y al final del vigésimo sexto alcanza un 50%. Al hacer estudios comparativos en pollos, los resultados muestran que la eclosión comienza al décimo octavo vía y alcanza un 50% al final del décimo noveno. De acuerdo a la calendarización llevada a cabo por la industria, ésto significaria que un 30% de pollos y pavipollos tienen más de 72 horas de haber nacido, en el momento de ser alojados en las criadoras. Nuestros estudios indican que éste 30% de aves que eclosionan prematuramente, sufren mortandades hasta de un 25 a 30 por ciento. Estos datos explicarían el 1-12% del total de mortandad observado en el campo.

El examen postmortem de todos los pavipollos señala que los machos predominan durante el primer 50% de los nacimientos, lo cual explica su elevada -- mortandad durante la primera semana de crianza.

Los estudios realizados sobre el proceso de deshidratación demuestran que - los pollitos comienzan a perder líquidos en el momento de nacer, aunque estén alojados en la nacedora con un 70% de humedad. Al final de las 72 horas de edad, el ave ha perdido un 15% de su peso. Si los pavipollos son alojados más de 72 horas en la nacedora, llegan a perder hasta un 30% de su peso corporal, debido a la deshidratación; cuando son alojadas en las criadoras, es común que rehusen comer y beber.

Para prevenir la excesiva deshidratación, es necesario que el encargado de la incubadora verifique constantemente el avance de la eclosión, en lugar de apegarse estrictamente a una calendarización. También,se podrían revisar las charolas dos veces al día, en caso de prolongarse la duración de los -- nacimientos. Esto sucede frecuentemente con huevos que se almacenan durante períodos prolongados. En nuestro estudio, el mismo fenómeno fue observado - con huevos recientemente puestos.

Traducción: Cortesía del Dr. Armando Antillón Rionda.

INCUBATOR DEHYDRATION AS A CAUSE OF EARLY POULT MORTALITY

Introduction

The degree of physiological stress evident in poults at the time of their placement in brooder pens is related to the level of poult mortality during the first two weeks of brooding. Dehydration resulting from depletion of their limited water reserves present at hatching is a principal stressor (1).

The rate of embryonic development is influenced by both genetic and environmental factors. Studies have shown that hatching time, within a population, can be shifted as much as 42 hrs through genetic selection (2). Field evidence suggests that commercial poultry breeders, in an effort to increase total poult yield, have given a selection advantage to breeder hens whose eggs hatch early. Though not harmful in itself, this trend can cause considerable problems for the commercial hatcheryman who is unaware of the consequences.

Experimental

Controlled laboratory studies using commercial turkey hatching eggs show hatching to begin during the later half of the 25th day and to be 50% complete by the end of the 26th day of incubation, with a total hatching time of 56 hrs (Table 1).

Table 1

Hatch Distribution of Commercial Turkey Poults

Days of incubation	Cumulative % Hatch Trial 1	Trial 2
25.9	3	3
26.3	6	18
26.7	42	33
26.9	68	50
27.3	91	71
27.7	98	92
27.9	99	96
28.3	100	100

Using commercially accepted hatch-placement schedules, one finds from extrapolations of data in Table 1 that approximately 30% of the poults would be in excess of 72 hrs of age when placed in brooder pens. Brooder house mortality for poults between 72 hrs and 120 hrs of age at placement in this study was 35% compared with 6% for poults under 72 hrs of age. Post mortem examinations showed the majority of this early mortality to be males. A possible explanation could be that the males were slightly older at placement since they were found to predominate during the early part of the hatch (Table 2).

Table 2

Distribution of Sexes Within the Hatch

Days of	% of poults		Cumulative %	
incubation	male	female	male	female
26.3	55	45	55	45
26.7	59	41	57	43
26.9	60	40	58	42
27.3	40	60	53	47
27.7	36	64	49	51

Weight data shows measurable poult dehydration begins within 4 hrs following emergence of the poult from the egg even though birds remain within the hatcher. The rate of weight loss is essentially linear and amounts to approximately .2g/hr or about 7% body wt/24 hrs. Actual weight losses experienced are given in Table 3.

Table 3

Body weight Loss from Dehydration

Age(hrs) at placement	Placement wt. (g)	Cumulative % body wt. loss
0	67.0	0.0
24	62.5	6.7
48	57.1	14.8
72	53.6	20.0
96	49.1	26.7
120	45.2	32.5

To prevent excess dehydration and the resulting early brooder house mortality it is necessary for the hatcheryman to monitor the progress of each hatch rather than rely on a rigid calendar schedule. It may also be necessary to pull some hatches twice if the total hatch time is prolonged due to setting of old hatching eggs or to suboptimal incubation temperatures.

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MUESTREO MICROBIOLOGICO DE INCUBADORAS Y HUEVO INCUBABLE

RESUMEN:

R.A. Ernst, A.A.Bickford y J.Glick-Smith.

El presente estudio se hizo para evaluar la utilidad práctica de técnicas microbiológicas en el muestreo de niveles microbianos a nivel de incubadoras y huevo incubable. El muestreo se hizo en 13 incubadoras comerciales durante - 22 visitas separadas.
Para obtener un cálculo aproximado del número de hongos y bacterias contaminantes, se procedió a colocar durante 10 minutos en diferentes sitios de la incubadora, un pequeño número de cajas de Petri expuestas, las cuales contenían Agar Tripticasa Soya.
Para muestrear la cantidad de Escherichia coli por contaminación fecal del cascarón, se procedió a utilizar cinta estéril para enmascarar, se colocó -- sobre el polo más ancho del huevo, se desprendió junto con el material contaminante y finalmente se procedió a colocar suavemente por contacto la cinta sobre cajas de Petri conteniendo azul de metileno eosina.
Los resultados indicaron una variación muy amplia en el conteo bacteriano, lo cual estuvo aparentemente relacionado con las presencia de pollitos en el area, la hora de muestreo y el programa sanitario utilizado en la casa - incubadora. El conteo bacteriano se hizo más fácil después de 24 horas de - incubación; el conteo de hongos, después de 48 horas. El sistema de caja de Petri expuesta parece ser el más indicado para muestrear incubadoras. El método de muestreo con cinta para enmascarar es bastante efectivo, aunque muy tardío.

Traducción: Cortesía del Dr. Armando Antillón R.

MICROBIOLOGICAL MONITORING OF HATCHERIES AND HATCHING EGGS

SUMMARY:

This study was undertaken to evaluate the practicability of techniques for monitoring microbial levels in hatcheries and on hatching eggs. Measurements were made in 13 commercial hatcheries on 22 separate visits. Tryptic soy agar plates were exposed for 10 minutes in various hatchery areas to obtain an estimate of total bacterial and mold contamination. Egg surfaces were sampled with sterile sampling tape by first pressing the tape on the large end of the egg and then on eosin methylene blue plates for E. coli to estimate fecal contamination.

The results showed wide variation in microbial counts which appeared to be related to the presence of chicks in the area, the time of sampling, and the sanitation program used in the hatchery. Total bacterial counts were most easily made after 24 hours of incubation and molds were most effectively counted after 48 hours. The plate exposure technique appeared to be a simple and effective method of monitoring hatchery sanitation. The sterile sampling tape was an effective but time-consuming method of sampling egg surfaces.

<u>Hatchery Monitoring: Microbiological</u>, by R.A. Ernst, J. Glick-Smith, A.A. Bickford,
University of California, Cooperative Extension, Davis, California 95616.

The hatchery plays a central role in the production of poultry. In the past, sanitation of the hatchery was often given little attention. The development of larger integrated companies for production of commercial poultry has resulted in many larger hatcheries. These are usually designed for effective sanitation but the large and frequent hatches also result in the release of large numbers of microorganisms into the hatchery environment. Therefore, in large hatcheries there is a need for a good sanitation program and an effective method to monitor levels of contamination in the hatchery and on hatching eggs. The latter will assure management that the sanitation program is working effectively.

Despite the central role of the hatchery and its importance in disease control, few reports could be found evaluating methods of monitoring hatchery contamination.

Suggested Areas to Monitor

1. egg surfaces
2. egg holding room(s)
3. setters
4. setter room(s)
5. hatchers
6. hatcher room(s)
7. chick processing room(s)
8. chick holding room(s)
9. vaccine preparation area
10. various surfaces (e.g., clean flats, hatching trays, machine surfaces, etc.) as deemed necessary

Equipment Needed

1. Media plates
   a. Tryptic Soy Agar (TSA) or Nutrient Agar (NA) for bacteria and molds
   b. Sabouraud's Agar (SA) or Corn Meal Agar (CMA) for molds - optional
   c. Levine's Eosin Methylene Blue Agar (EMB) for *E. coli* on egg surfaces
2. Marking pencil
3. scotch tape
4. table top incubator (optional)
5. Sampling tape - "Con-tact-it," Birko Chemical Corp., PO Box 1315, Denver, CO 80201, (303) 623-4161 (optional)
6. Mechanical counter (optional)
7. Record forms (see Appendix 1)

Procedure: Air Sampling

1. Obtain plates and incubate upside down 24 hours to assure sterility. TSA or NA plates are recommended.
2. Expose plates in various areas of hatchery. Don't touch inside of lid or plate.
   a. Hatcher and rooms with chicks present - expose 5 min.
   b. Other areas - expose 10 min.
3. Label and tape plate after exposure; note location exposure time and media used on your record sheet.
4. Incubate plates 24 hours at 37°C (99°F) in small incubator or a setter. Plates should be positioned with agar on top. If a setter is used, place them in a plastic bag to keep out contamination.
5. Make plate counts at 24 and 48 hours. Bacterial colonies are generally glistening and smooth; mold colonies are generally hairy, powder-like and dull. If colonies are too numerous to count resample the area using a shorter plate exposure time.

6. Adjust counts using a proportion as follows:

Count = 14 bacterial colonies; 3 mold colonies
Time = 7 minutes

$\frac{7}{14} = \frac{10}{x}$; $7x = 140$; $x = \frac{140}{7} = 20$

$\frac{7}{3} = \frac{10}{x}$; $7x = 30$; $x = \frac{30}{7} = 4.3$

7. Record adjusted counts on record form (attached Appendix 1)

Procedure: Surface Sampling Without Tape

Press the large end of each egg carefully on the EMB plate. Be careful not to touch that part of the egg shell to be pressed against the plate. It is recommended that the plate be divided into six equal pie-shaped sections with a marking pencil before you start. One section should be left blank to assure that the plate was not contaminated by other means.

Procedure: Surface Sampling With Tape

1. Pull tape over sample area on tape container using care not to touch surface.
2. Press tape firmly on area to be sampled. If on egg surface, roll tape for complete contact.
3. Press tape on plate (5/plate). Use EMB plates for eggs.
4. Incubate and count as before at 24 hours; adjustment is necessary.

Standards

	Counts		
Score	Setters	Rooms	Mold Contaminants
Excellent	0-10	0-15	0
Good	11-25	16-36	1-3
Average	26-46	37-57	4-6
Poor	47-66	58-76	7-10
Worse than poor	67-86	77-96	10-12
Miserable	87 & over	97 & over	13 & over

from Sadler (1975)

Procedure: Reading Plates

We have found that TSA plates can be most easily read at 24 hours for total bacterial counts and at 48 hours for mold counts. After 24 hours mold colonies may overgrow the plate and make it hard to count the bacterial colonies. The EMB plates used for *E. coli* identification can best be read after 48 hours of incubation. The colonies of *E. coli* have a metallic sheen on the EMB media.

Field Testing

During the spring and summer of 1979 this procedure was used 23 times in 14 California hatcheries. The complete results are shown in Table 1. The results are most meaningful when they are compared with the standards provided by Sadler (1975). High counts are expected when chicks are present during sampling, however, the same areas should show low counts after clean up. In hatchery F this occurred. Sample F-1 was made at 9:30 a.m. after a hatch had just been pulled; a hatch was coming out in the hatchers sampled. The high counts in the hatchers and chick processing areas are expected during this time. Sample F-2 was taken later when no hatch was in progress, notice the low counts in all areas. This hatchery had obviously been effectively cleaned.

The results of field testing the air sampling procedure showed it to be a fast and effective method for estimating contamination in a hatchery. This method does not provide information about pathogenic organisms which might be present. Occasional culturing of pips or culls in a veterinary laboratory is suggested as a means of monitoring for pathogenic organisms.

The tape sampling procedure was useful for estimating fecal contamination of hatching eggs (see Table 1) and assessing the success of egg disinfection. This method has also been shown to be an accurate estimation of E. coli on egg surfaces (Arhienbuwa, et al., 1980). It was slow requiring about 10 minutes to sample 25 eggs. Pressing egg surfaces directly on the plate was tried as an alternative. This method was very fast and under these conditions appeared to give a reasonable index of contamination. However, it has not been carefully evaluated under controlled conditions and may be less accurate than the tape method. Shell sampling before and after egg sanitation is suggested as a useful method of monitoring flock management and egg sanitation under practical conditions.

Summary

1. The air sampling procedure was found to be simple and could be easily done by a hatchery manager or other employee. About one hour is required for exposing and counting the plates.

2. The cost of plates is small (30 to 35¢ each) although availability may be a problem. Media plates can be purchased or prepared in a clean room in the hatchery or other location.

3. Air sampling appears to provide a reasonable method for management to keep abreast of microbiological conditions in a hatchery.

4. The surface sampling of eggs proved to be an effective method to determine relative egg cleanliness. Pressing the egg directly on EMB plates seemed to be much easier than sampling with tape, however, it may have been less consistent.

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Hatchery____________________ Manager____________________

Date Sampled____________________ Sampler____________________

Egg treatments____________________ Conditions at sampling____________________

Approximate time of sampling________ Type of Incu.________ Birds________

Plate #	Location	Time (min.)	Notes	Count at (hours) 24	48	72	Media	Sample Air	Surface	Colony Type

Table 1 - HATCHERY SANITATION SURVEY RESULTS

Hatchery & (type)	Time of Count (hrs)	Location in Hatchery: Incubator Room	Incubator	Hatcher Room	Hatcher #1	Hatcher #2	Cold Egg Storage	Egg Tray Room	Chick Pull-Processing	Chick Vaccinating	Chick Holding	Washing Area	General Area	Egg Contamination #Eggs/25 sampled
*A (L,B,T)[a]	24	NA[b]	14	1	13	NA	1	NA	3	NA	NA	NA	NA	NA
	48	NA	23	4	20	NA	1	NA	7	NA	NA	NA	NA	NA
B-1 (B)	24	16-1m	29	NA	TNTC[c]	1	1	2	NA	NA	NA	3	NA	7/25, Ø/25
	48	26-1m	32	NA	TNTC	7	4-1m	5	NA	NA	NA	7	NA	14/25, 5/25
*B-2 (B)	24	16-32m	7m	2m	19	NA	2-31m	16-12m	31-6m	46-2m	TNTC	55-19m	NA	2/25, Ø/25
	48	28-49m	13m	2-6m	27	NA	6-52m	28-19m	43-11m	68-6m	TNTC	63-27m	NA	5/25, Ø/25
*C-1 (B)	24	2	NA	35	TNTC	TNTC	8	139-1m	53	NA	NA	NA	NA	4/25
	48	2-1m	NA	35+-6m	TNTC	TNTC	20	TNTC-1m	53+-1m	NA	NA	NA	NA	7/25
*C-2 (B)	24	3	Ø	24	128-3m	NA	Ø	1	TNTC-4m	48-3m	53-3m	NA	NA	1/25, Ø/25
	48	7	Ø	33	TNTC-8m	NA	1	4	TNTC-8m	63-9m	71-7m	NA	NA	1/25, Ø/25
D-1 (B)	24	84	NA	102	33	NA	38	85	153	83	118	NA	NA	9/25, 20/25
	48	84+	NA	102+6m	33+-5m	NA	53-3m	85+-3m	153+	83+	118+-1m	NA	NA	11/25, 20/25
*D-2 (B)	24	10	NA	16-27m	84	NA	5	78	TNTC	98-1m	93	NA	64-20m	7/25, 7/25
	48	14	NA	26-32m	98	NA	8	86	TNTC	TNTC-4m	TNTC	NA	78-31m	7/25, 9/25
E-1 (T)	24	10	2	31	TNTC	TNTC	Ø	1	3	NA	NA	NA	NA	Ø/25
	48	17	26	31	TNTC	TNTC	Ø	3	9	NA	NA	NA	NA	Ø/25
*E-2 (T)	24	2	Ø	TNTC	TNTC	NA	2	10	TNTC	TNTC	TNTC	NA	NA	NA
	48	3	Ø	TNTC	TNTC	NA	3	14	TNTC	TNTC	TNTC	NA	NA	NA
F-1 (L)	24	4	3	21-1m	95*	138*	5-2m	10-1m	81	144	NA	NA	NA	1/25
	48	8	3	31-1m	95+*	138+*	9-2m	10-1m	81+	144+	NA	NA	NA	1/25
F-2 (L)	24	2	1	3	Ø	NA	Ø	3	1	Ø	NA	NA	NA	Ø/25
	48	7	1	3	Ø	NA	Ø	4	1	Ø	NA	NA	NA	Ø/25
*G-1 (B)	24	15	4-1m	85	1	NA	3-1m	2	TNTC-1m	NA	NA	17-26m	NA	3/25, 5/25
	48	24	7-2m	85+	1-1m	NA	8-4m	2	TNTC-1m	NA	NA	17-42m	NA	5/25, 5/25
*G-2 (B)	24	10	1-1m	80	6-1m	NA	Ø	12	TNTC	NA	NA	53	NA	3/25
	48	21	1-1m	80+-5m	6-5m	NA	3	24-1m	TNTC	NA	NA	90-8m	NA	8/25
*H-1 (L)	24	8	Ø	12	46	26	78	NA	73	71	TNTC	NA	NA	2/25, Ø/25
	48	TNTC	Ø	12	49	36	TNTC	NA	73	71	TNTC	NA	NA	3/25, 1/25
H-2 (L)	24	4	1	TNTC	TNTC	7	9	NA	NA	NA	31	NA	NA	NA
	48	10-1m	3	TNTC	TNTC	11	18	NA	NA	NA	51	NA	NA	NA
I (L)	24	17	Ø	1	Ø	NA	Ø	6	3	NA	NA	9	NA	3/25
	48	17	Ø	2	Ø	NA	Ø	10	11	NA	NA	14	NA	3/25
J (L)	24	3-1m	Ø	6	1	NA	1	5	TNTC	NA	NA	22	NA	Ø/25
	48	6-1m	Ø	8	4	NA	3	7	TNTC	NA	NA	30	NA	Ø/25
*K (B)	24	3	40	7	5	61-1m	NA	25	81	11	48	40	NA	16/25, 17/25
	48	3	40+	10	8	61-1m	NA	30	100+	16	70	46	NA	22/25, 21/25
*L-1 (L)	24	5	4	3	NA	NA	39	NA	TNTC	TNTC	NA	19-2m	6-1m	Ø/25
	48	5	4	4	NA	NA	39	NA	TNTC	TNTC	NA	19-2m	6-1m	Ø/25
L-2 (L)	24	6	Ø	2	Ø	NA	9	Ø	3	22	1	5	1	Ø/25
	48	7	2	3	Ø	NA	11	1	7	28-1m	5-1m	12	1-1m	Ø/25
M-1 (B)	24	4	3	11	2	NA	Ø	4	137	NA	NA	6-1m	12	NA
	48	5-9m	5-34m	18-1m	3	NA	Ø	10	TNTC-2m	NA	NA	8-3m	21	NA
*M-2 (B)	24	29-1m	3-2m	127-1m	10-1m	NA	Ø	Ø	NA	NA	TNTC	42-60+m	120	6/25, 3/25
	48	29-53m	3-9m	TNTC-11m	10-12m	NA	Ø-1m	1	NA	NA	354-4m	TNTC	159	6/25, 3/25
N (D)	24	123	Ø	123	Ø	NA	15	28	NA	NA	NA	NA	12	22/25, 7/25
	48	TNTC	5	TNTC	Ø	NA	20	52	NA	NA	NA	NA	22	22/25, 8/25
AVERAGE														
Bacterial Count	24	17.1	5.2	45.0	54.2	76.1	9.8	22.5	88.0	74.8	94.4	24.6	35.8	5.0/25
	48	28.3	7.8	49.5	57.0	79.0	16.2	27.7	94.1	83.9	126.9	41.4	53.0	6.4/25
Mold Count	24	(5)[d]11.0	(4)2.8	(2)2	(3)1.7	(1)1.0	(3)11.3	(3)4.7	(3)3.7	(3)2.0	(1)3.0	(5)21.6	(2)10.5	
	48	(8) 18.6	(5)11.8	(8)8.5	(5)6.2	(1)1.0	(6)10.5	(5)5.0	(5)4.6	(4)5.0	(4)3.3	(6)23.7	(3)11.0	

* - Birds present in hatchery during sample.

[a](L) - layer, (B) - broiler, (T) - turkey, (D) - duck.

[b]NA - Not Available or not sampled.

[c]TNTC - Too numerous to count

[d]Number of samples with a mold count of one or more

INHIBITING DISEASE TRANSMISSION IN THE HATCHER BY AIR FILTRATION

Bernard L. Rosenberg
President, Robbins Incubator Co.
Adjunct Professor, Department of Animal Sciences,
Colorado State University

Abstract
Previous research and experience have long since demostrated the air-trans-miss-ability of disease organisms, but evidence clearly indicates that such organisms are transported as "passengers" on and in particles and do not appear to survive in truly clean air, Filtration has been shown to be quite effective in reducing specific organism counts in experimental hatchers of a particular desing. Further research indicates concomitant reduction in flock mortality and morbidity and improvements in bird weight when chicks have been hatched in the same experimental filteres hatchers. Commercial hatchers, from the prototype of which the above-mentioned experimental machines were adapted, have been marketed and successfully been shown to remove virtually all down from the hatcher air, and, in consequence, to - redice prganism counts to acceptable levels of lo% or less of those in non-filtered machines of standard design. These results are achieved with only a medium-efficiency and, therefore, economic filter rated at 1 full micron in particle size. Hatching results were superior in the experimental filte-red hatcher to those in a non-filtered control of identical design. At the commercial level, both filtered and non-filteres hatchers from the same manufacturer appear to produce equal hatchability when each is operated correctly, and the filtered hatcher appears to produce significantly better-quality flocks.

INHIBICION EN LA TRANSMISION DE ENFERMEDADES POR FILTRACION DE AIRE EN LAS NACEDORAS.

Las investigaciones y las experiencias previas han demostrado la transmisión aerea de organismos, pero la evidencia claramente indica que dichos organis-mos son transportados como "pasajeros" sobre y dentro de particulas y aparen temente no sobreviven por si solos en aire puro, la filtración ha demostrado ser totalmente efectiva en la reducción de la cantidad de organismos especí-ficos en nacedoras experimentales. Investigaciones posteriores indican una reducción concomitante en la mortalidad y morbilidad así como incremento en el peso de los pollitos cuando los huevos han sido incubados en esas nacedo-ras experimentales con filtros.Las nacedoras comerciales adaptadas al siste-ma del prototipo de las máquinas experimentales han tenido éxito al remover practicamente todo el plumón de la nacedora y como consecuencia la reducción en la cuenta de microorganismos a niveles del 10% o menos de los existentes en nacedoras carentes de sistemas de filtración.
Estos resultados son alcanzados solamente con filtros económicos de eficien-cia media, para particulas de 1 micron de tamaño. Se obtuvieron mejores re-sultados en los nacimientos en nacedoras experimentales con filtración en -- comparación con nacedoras del mismo diseño sin filtración. A nivel comercial las nacedoras con filtros y sin filtros con la misma fabricación, parecen - producir igual incubabilidad cuando cada una es operada correctamente y la nacedora con filtros aparentemente produce pollitos de mejor calidad.

Traducción: Cortesía del Dr. Carlos López Coello

RISK ANALYSIS IN AVIAN MEDICINE

R.K. Edson, D.V.M., M.P.V.M., and R. Yamamoto, Ph.D.
Department of Epidemiology and Preventive Medicine
University of California at Davis
Davis, California 95616

Abstract

Risk analysis is a technique for comparing the risks of a condition of interest (e.g. death, airsacculitis) between groups having different levels of exposure to some (possibly) causal factors. Groups are compared by calculating relative risk (= RR = incidence in exposed group ÷ incidence in unexposed group) and attributable risk (= AR = incidence in exposed group - incidence in unexposed group). If a larger proportion of the exposed group shows the condition of concern, that group is said to be at higher risk of the condition. This will be reflected in larger RR and AR values. RR is said to be significant if it is found to be significantly larger than 1.00 at some specified probability. AR is larger than expected if it is greater than 0.00 and RR is significant. If biological, spacial and temporal considerations warrant, it may be inferred that the agent of exposure plays a causal role in the condition. By reversing the logic used in the foregoing explanation, a factor may be found to be protective if it places individuals at significantly lower risk of the condition. This is the desired situation when animals are given vaccine prior to exposure. In this case RR will be significantly less than 1.00, and AR will have a negative value.

Analysis of the potential role of _Mycoplasma_ _meleagridis_ (Mm) in late incubation turkey embryo mortality is presented as an example of risk analysis. Risk of death was 25.6% for Mm+ embryos, but it was only 7.1% for Mm- embryos. This is reflected in the RR and AR values of 3.6 ($P < .001$) and .18, respectively. The inference is that Mm+ embryos are 3.6 times more likely to die during the 25-28 day period of incubation than are Mm- embryos, and that Mm may kill 18% of the embryos it infects. Extension of the analysis to the total population of embryos shows them to have RR = 1.6 ($P < .001$), and AR = .04. This suggests that the 25% prevalence of Mm in the population causes a 4% death loss. Sampling techniques and interpretation of findings are discussed.

ANALISIS DE RIESGOS EN MEDICINA AVIARIA.
Resumen.

El análisis de riesgos es una técnica por medio de la cual se comparan los
riesgos de una condición de interés (ej: muerte, aerosaculitis)entre grupos
que cuentan con diferentes niveles de exposición a posibles factores causa-
les. La comparación de los grupos se hace calculando el riesgo relativo ---
(=RR= incidencia en el grupo expuesto ÷ incidencia entre el grupo no expues-
to) y riesgo atribuido (=AR= incidencia en el grupo expuesto - incidencia -
en el grupo no expuesto). Si un mayor número del grupo expuesto muestra el
problema de referencia, entonces se dice que ese grupo tiene un riesgo ma--
yor a esa disposición. Esto se reflejará en valores RR y AR más altos. Se -
dice que el valor de RR tiene significado cuando es estadísticamente mayor
de 1.00 a una probabilidad previamente especificada. El valor AR es más ele
vado del esperado cuando es mayor de 0.00 y RR es significativo. Si justi-
fican las consideraciones biológicas, espaciales y temporales, entonces pue
de inferirse que el agente de exposición juega el papel de causante de la -
condición. Invirtiendo la lógica empleada en la anterior explicación,un fac
tor puede resultar protector, si se sitúan individuos en un nivel significa
tivo más bajo del riesgo de sufrir el problema. Esta es la situación desea-
ble, cuando se aplica una vacuna antes de exponerse la parvada. En este ca-
so,RR será significativamente menor que 1.00 y AR tendrá un valor negativo.

Como un ejemplo de este análisis de riesgos,tomamos la mortandad embriona--
ria en edad avanzada en pavos,causada por _Mycoplasma meleagridis_ (Mm). El -
riesgo de mortandad por Mm+ fue de un 25.6%,y solamente de un 7.1% para em-
briones Mm^-.Esto se refleja en los valores RR y AR de 3.6($P < .001$) y 0.18,
respectivamente. De lo anterior se infiere que los embriones Mm^+ tienen 3.6
veces mayores posibilidades de morir durante los 25-28 días del periodo de
incubación,que los embriones Mm^-;además de que Mm puede matar el 18%de los
embriones que infecta.Cuando se hace intensivo el análisis a la población -

total de embriones, éstos muestran una RR = 1.6 ($P< .001$) y AR = .04. Esto sugiere que el 25 % de prevalencia de Mm en la población, causa un 4 % de mortandad. Se discuten técnicas de nuestreo e interpretación de ha-- llazgos.

Traducción: Cortesía del Dr. Armando Antillón Riodna

RISK ANALYSIS IN AVIAN MEDICINE

Introduction

A requirement of all scientific study is data analysis, often called statistical analysis. For many people statistical testing procedures are difficult to understand and to perform. Outcomes of statistical tests such as χ^2 (Chi-square) and F tests may be difficult for the non-statistician to interpret. The purpose of this paper is to describe Risk Analysis, a mathematically simple and intuitively pleasing statistical method. The method is well suited to avian medicine where populations and factors influencing those populations are usually well-defined. Our discussion of risk analysis will involve only the most straightforward cases: those in which all populations and factors of interest are clearly defined, and where prevalence (or incidence) of the condition is not less than 1%.

Definitions

1. Condition: a disease, lesion, or production characteristic which is the result of factors whose identity may or may not be known.

2. Factor: a characteristic which can be measured in animals with and without the condition of concern, e.g., vaccination: yes or no; infection: yes or no. Refer to groups as factor-positive (F+) and factor-negative (F-).

3. Risk: the proportion of a factor-defined group which is affected by the condition of interest.

4. Population at risk: all individuals at risk of being affected by the condition of concern. In general there will be two sub-populations at risk as determined by factor distribution. These two added together comprise the total population at risk.

5. Relative Risk (RR): Relative risk is the measure of association between the factor and the condition. It is determined by dividing risk for the F+ group by the risk for the F- group.

$$RR = \frac{Risk_{F+}}{Risk_{F-}}$$

Values of RR close to 1.0 indicate that risks are similar for the two groups, and that the factor has little or no association with the condition. Values significantly different from 1.0 imply association between factor and condition.

(a) Values of RR significantly less than 1.0 imply that the factor decreases, or has a positive association with another factor which decreases the risk of the condition, e.g., fowl cholera vaccination might lead to a decreased risk of disease upon challenge.

(b) Values of RR significantly greater than 1.0 imply that the factor increases, or has a positive association with some other factor which increases risk of the condition, e.g., *Mycoplasma gallisepticum* infection of broiler breeding hens could result in an increased risk of chronic respiratory disease in their progeny.

6. Attributable Risk (AR): Attributable risk is a measure of how much the risk of the condition is increased (possibly caused by) or decreased (possibly prevented by) the factor. AR may be thought of as a measure of the importance of the factor. It is simply the difference between the risk of the two groups:

$$AR = Risk_{F+} - Risk_{F-}$$

Analytical Method

Data are first put into a 2 X 2 table (also called a 4-fold table) so that the various populations and subpopulations are clearly defined (Table 1). All calculations are made by adding, subtracting or dividing the numbers in this table. Methods for testing significant departure of RR from 1.0 are described by Schwabe et al. (5). None of the mathematical procedures requires more than a hand calculator.

Table 1. The 2 X 2 table as used in risk analysis[6].

		CONDITION OF CONCERN		
		AFFECTED	NOT AFFECTED	TOTALS AT RISK
FACTOR OF INTEREST	F+	A	B	[A + B][1]
	F-	C	D	[C + D][2]
	Totals	[A + C][3]	[B + D][4]	[A+B+C+D][5]

1 and 2 The F+ and F- subpopulation at risk totals, respectively.

3 and 4 The affected and unaffected subpopulation totals, respectively.

5 The complete population at risk.

6 Risks, Relative risk (RR), and Attributable risk (AR) are calculated as

(a) Risk for F+ group = A/[A+B]

(b) Risk for F- group = C/[C+D]

(c) Relative Risk = RR = $\frac{A/[A+B]}{C/[C+D]}$

(d) Attributable Risk - AR = A/[A+B] - C/[C+D]

Example of Risk Analysis

Carpenter et al. (1) investigated the possibility that *Mycoplasma meleagridis* (Mm) causes late incubation mortality of turkey embryos. They inoculated Mm into turkey embryos at the 9th day of incubation; controls were either placebo-inoculated (with PPLO broth) or uninoculated. All embryos still alive at the 25th day of incubation were defined as the population at risk; embryo death during the last 3 days of incubation was the condition of concern. Table 2 shows a summary of their data as we might normally see them presented (2-factor analysis of variance was used to analyze a more detailed data set which included information specifying location of the eggs within the incubator). Table 3 shows risk analysis of the same data with the uninoculated and placebo-inoculated embryos combined as one factor-negative group.

Table 2. Percent hatch of 25-day live turkey embryos for eggs inoculated at 9 days of inclubation with either 0.1 ml of *Mycoplasma meleagridis* (Mm) in PPLO broth (7.8×10^5 CFU per embryo) or with PPLO broth alone, and for uninoculated eggs.

Inoculation	No. Alive at 25 days	Number Hatched	Percent Hatched
M. meleagridis in PPLO broth	277	181	65.3
PPLO broth only	220	188	85.5
None	239	200	83.7

Table 3. Analysis of risk of late-incubation (25-28 days) embryo death as a function of *Mycoplasma meleagridis* (Mm) infection status of the 25-day live embryo.

Data Presentation in 2 X 2 Table Format	Die	Hatch	Total	Risk of Death	[% Mm+]	Relative Risk (RR)	[P]	Attributable Risk (AR)
Mm+	96	181	277	.347	100.0	2.24	[<.001]	.192
Mm-	71	388	459	.155	0.0	1.00	-------	-----
Totals	167	569	736					

A The probability that RR differs from 1.0 by chance alone.

Results and Discussion

The risk analysis reveals that infected embryos have a 2.24 time greater chance of dying (P<.001) than do uninfected embryos in the experimental situation described. This result agrees with earlier observations of Yamamoto and Ortmayer (7) and with a field study by Edson, et al. (2). The risk of embryo death attributable to Mm infection (AR) is .192, which means that nearly 20% of the deaths in the infected group are due to (attributable to) Mm infection.

The embryo mortality example is nice because the condition is of high prevalence and because the infected and uninfected subpopulations are clearly defined. However, this is not the case with all diseases, especially when the study is not of the investigator-controlled experimental type shown here. It is possible to apply risk analysis to diseases of low prevalence, and to diseases wherein factor distribution is not obvious but must be estimated. These situations require slightly different approaches, most of which are described in some of the references cited (3,4,5,6). Risk analysis is widely used in human epidemiology, and it seems reasonable to expect it to become a useful tool for avian medicine.

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IMMUNODEPRESSION TO POX VACCINES ADMINISTERED SUBCUTANEOUSLY

W. T. Springer, D.V.M., and R.W. Truman, M.S.
Department of Veterinary Science
Louisiana State University
Baton Rouge, Louisiana 70803

The immunodepressive effect of avian pox vaccines on the hemagglutination-inhibition antibody (HI) response to Newcastle disease virus (NDV) and numbers of immunoglobulin-M (IgM) secreting spleen cells of chicks was evaluated. Mild fowl pox and pigeon pox vaccines were given subcutaneously in the dorsum of the neck of Specific-Pathogen-Free White Leghorn chicks at day-1. Treatment levels for each vaccine studied included non-vaccinated controls, and 1:2, 1:10, and 1:50 dilutions of the recommended wing-web vaccination dose. A modified hemolytic plaque assay with sheep red blood cells was used to enumerate IgM cells.

Immunodepression of NDV-HI geometric titers resulted from all vaccines given at every dilution, with titers of groups given pigeon pox vaccines at a 1:2 dilution significantly lower than of both the controls and the group given a 1:50 dilution. An inverse vaccine dose-response was apparent for HI titers at 4 weeks (3 weeks post-NDV administration in water at day 7).

The number of IgM secreting spleen cells was significantly less ($P < 0.01$) at day 14 in groups receiving either fowl pox or pigeon pox vaccines at the 1:2 and 1:10 dilutions and less in those given the 1:50 dilution than in controls. Cell numbers remained depressed at day 28 in all groups receiving pox vaccines but the groups given pigeon pox vaccines at the 1:50 dilution.

INMUNODEPRESION DEBIDA A VACUNAS DE VIRUELA ADMINSITRADAS POR VIA SUBCUTANEA
El efecto inmunodepresión de las vacunas de viruela aviar es la respuesta -- producida hacia el virus de la Enfermedad de Newcastle (NDV) en la prueba de inhibición de la hemoaglutinación (HI), y el número de inmunoglobulinas IgM secretadas por las células del bazo, fueron evaluados.
Vacunas suaves de viruela fueron administradas subcutáneamente en el dorso del cuello del pollitos White Leghorn libres de patógenes especificos al --- primer día. Junto con los diferentes niveles de vacuna administrados en cada tratamiento, fueron incluidos aves control no-vacunadas, así como diluciones 1:2, 1:10, 1:50 de la dosis recomendada para la punción en el ala.
Una modificación a la pruena hemolítica en placa con glóbulos rojos de ovino fue utilizada para enumerar las células IgM.
En todas las diluciones se observó una inmunodepresión geométrica de ENC-HI en los títulos que resultaron tras la aplicación de las vacunas. Los títulos obtenidos de la dilución 1:2 de vacuna de la viruela de pichón resultaron -- significativamente más bajos que los controles y que el grupo al que se le aplicó la dilución 1:50. Una respuesta inversa a la dosis vacunal fue aparente en los títulos de HI a las 4 semanas (3 semanas después de la administración de ENC en el agua en el séptimo día).
El número de células secretoras IgH del bazo fue significativamente menor - (P 0.01) en el día 14 en los grupos que recibieron tanto las vacunas de viruela aviar como las vacunas de viruela de pichón con las diluciones 1:2 y 1:10, y menor en aquellos que recibieron la dilución 1:50, en comparación - con los controles.
El número de células permaneció disminuído durante el día 28 en todos los - grupos que recibieron vacunas de viruela aviar, excepto los grupos que recibieron la vacuna de viruela tipo pichón con dilución 1:50.

Traducción: Cortesía del Dr. Carlos López Coello

IMMUNODEPRESSION TO POX VACCINES ADMINISTERED SUBCUTANEOUSLY

W. T. Springer, D.V.M., Ph.D., and R.W. Truman, M.S.
Department of Veterinary Science
Louisiana State University
Baton Rouge, Louisiana 70803

Introduction

Fowl pox, one of the first disease syndromes recognized in poultry, remains a problem for the poultry industry. Prophylactic measures are used eight months or more each year in the southern United States. Recently subcutaneous administration of fowl pox vaccines in combination with Marek's disease vaccine at day-1 has been employed to reduce labor and production costs.

In studies evaluating the subcutaneous route of pox vaccine administration, lower Newcastle disease hemagglutination-inhibition antibody titers were noted in chickens vaccinated for fowl pox than in non-pox vaccinated chickens. Subsequently studies were initiated to evaluate the effect of fowl pox vaccines given subcutaneously at different levels on day-1 on the immune response of SPAFAS White Leghorn chicks. The immune responses were measured by determining the hemagglutination-inhibition (HI) antibody titers to Newcastle disease virus three weeks post-vaccination for Newcastle disease, and the numbers of immunoglobulin-M (Ig-M) secreting spleen cells in chicks two weeks post-vaccination and four weeks post-vaccination for fowl pox.

The four pox vaccines evaluated in separate studies were: vaccine A, a fowl pox vaccine propagated in tissue culture; vaccine B, a fowl pox vaccine propagated in chick embryos; vaccine C, a pigeon pox vaccine propagated in chick embryos; and vaccine D, a pigeon pox vaccine propagated also in chick embryos. The four treatment levels used to evaluate each vaccine consisted of: 1.) controls receiving no vaccine, 2.) 1/2 the recommended wing-web dose, 3.) 1/10 the recommended wing-web dose, and 4.) 1/50 the recommended wing-web dose.

Determination of Newcastle disease HI titers

Five chicks per treatment level were housed together in each of eight replicates, giving a total of 40 chicks per treatment level. Chicks in all four treatment levels were vaccinated with a commercial B_1-type Newcastle disease vaccine in the drinking water seven days after pox vaccination. One chick from each of the eight replicates was bled at day-28 (21 days post-vaccination for Newcastle disease). The microtiter HI test was used to determine titers. Data were evaluated for statistical significance using the least squares means and Duncan's multiple range test.

The results shown in table 1 reveal that all pox vaccines given subcutaneously at the three treatment levels depressed HI antibody response to Newcastle disease virus.

Table 1

Newcastle Disease GM HI-titers (log_2) of Chicks given Pox Vaccines Subcutaneously

Pox vaccine	Pox vaccine treatment levels* 0	1/2	1/10	1/50
A	6.5	4.5	4.8	6.2
B	5.0	3.8	4.5	4.0**
C	5.5	2.8***	4.6	5.2
D	3.5	1.8	2.2	3.3

*Fraction of recommended wing-web dose.
**1/25 of the recommended dose used.
***Significantly different from controls (P 0.01)

Enumeration of Ig-M secreting spleen cells

Three chicks from each pox treatment level were housed together in each of four Horsfall-Bauer isolation units. Four chicks per treatment level were injected intra-abdominally with 1 ml of 20% washed sheep red blood cells. The chicks were sacrificed 6 days post-injection and spleens were removed and triturated to release the cells.

The spleen cells were placed in modified Cunningham chambers with sheep red blood cells and fresh chick serum containing 10% guinea pig serum to serve as the complement source. Ig-M secreting spleen cells were counted at both day-14 and day-28 post-vaccination for pox.

The results shown in table 2 indicate that all pox vaccines given subcutaneously at the three treatment levels reduced the number of Ig-M secreting spleen cells at day-14, and at day-28 the number of Ig-M secreting spleen cells remained significantly reduced in the spleens of chicks receiving the 1/2 recommended wing-web dose of any of the four pox vaccines.

Table 2

Number of Ig-M Secreting Cells per 10^6 Spleen Cells from Chicks given Pox Vaccines Subsutaneously

Vaccine	Day post-pox vac.	Pox vaccine treatment levels*			
		0	1/2	1/10	1/50
A	14	18.8 a**	4.3 b	10.0 c	9.4 c
	28	15.5 a	2.2 b	3.2 b	13.9 a
B	14	16.5 a	1.9 b	3.6 b	4.7 b
	28	16.9 a	4.5 b	7.4 c	10.4 c
C	14	29.9 a	5.7 b	9.8 b	20.8 c
	28	28.4 a	17.3 b	21.9 b	31.4 a
D	14	34.0 a	2.6 b	10.8 b	29.0 c
	28	25.4 a	10.1 b	17.3 a	26.4 a

*Fraction of recommended wing-web dose.
**Means on each horizontal line with different letters are significantly different at the P 0.05 level.

RATIONALE FOR RATIO SELECTION OF LINCOMYCIN AND SPECTINOMYCIN COMBINATION FOR POULTRY

A. H. Hamdy, D.V.M., Ph.D.
Agricultural Division, The Upjohn Company
Kalamazoo, Michigan 49001

ABSTRACT

This paper reviews *in vitro* and *in vivo* data describing the rationale used for ratio selection of lincomycin (L) and spectinomycin (S) combination for use in poultry. *In vitro*, the block test design was utilized against *Mycoplasma gallisepticum* (MG), *Staphylococcus aureus* (SA) and *Escherichia coli* (EC). *In vivo*, the protection test was conducted in laboratory animals and in young chickens to determine the optimal ratio of LS against lethal doses of SA (in lab animals) and MG, SA and EC (in young chickens).

The *in vitro* and *in vivo* data demonstrated that LS in a 1 to 2 ratio was the ratio of choice to make a positive contribution to the total efficacy of the antibiotic combination. L contributed significantly to LS against SA, while S contributed significantly to LS activity against EC, and LS was more effective than either L or S against SA and EC.

ESPOSICION RAZONADA PARA LA SELECCION DE LA RELACION DE ASOCIACION LINCOMICINA-ESPECTINOMICINA PARA AVES DE CORRAL

Extracto

En este trabajo se revisan los datos *in* vitro e in vivo que describen la exposición razonada aplicable para seleccionar la relación de asociación lincomicina (L) y espectinomicina (E) para su aplicación en aves de corral. *In vitro*, se utilizó el diseño del ensayo en bloque contra *Mycoplasma gallisepticum* (MG), *Staphylococcus aureus* (SA) y *Escherichia coli* (EC). *In vivo*, se realizó el ensayo de protección en animales de laboratorio y en polluelos a fin de determinar la relación óptima de LE contra dosis letales de SA (en animales de laboratorio) y MG, SA y EC (en polluelos).

Los datos in vitro e in vivo demostraron que LE, en una proporción de 1 a 2, fue la relación de elección para hacer una contribución positiva a la eficacia total de la asociación antibiótica. L contribuyó en forma significativa a LE contra SA, mientras que E contribuyó también en forma significativa a la actividad de LE contra EC. LE fue más eficaz que L o E contra SA y EC.

RATIONALE FOR RATIO SELECTION OF LINCOMYCIN AND SPECTINOMYCIN COMBINATION FOR POULTRY

This report reviews the rationale used for ratio selection of lincomycin (L) and spectinomycin (S) combination (LS) in 1 part L and 2 parts S for use in poultry.

The procedures used for the studies reported herein were described previously (1-4).

Results and Discussion

In vitro - The results of antimicrobial activities of the drugs tested are listed in Tables 1 and 2. For *mycoplasma gallisepticum* (MG), LS in 1 to 1, 1 to 2, 1 to 3, and 4 to 1 were the most active ratios tested. Greater anti-*Staph. aureus* (SA) activity was achieved with L or LS than with S, and greater anti-*E. coli* (EC) activity was recorded with S and LS than with L. A wider spectrum activity was demonstrated with LS than with either L or S. Such activity varied from mycoplasmastatic to cidal activity according to the antibiotic concentration and the organism tested (1,2).

In vivo - Table 3 shows the results of the protection test against lethal doses of SA in laboratory animals (4). Mice infected with SA that received no drug had 100% mortality. Mice treated with either 5 mg L/kg or 10 mg S/kg had 80% and 100% mortality respectively. However, infected mice treated with LS at 5 mg L and 10 mg S/kg had 40% mortality.

MG Infection - The results of the efficacy of the injectable antibiotics against MG infection in chickens are listed in Table 4. Combinations containing 5 mg L and 5 mg S; 10 mg L and 20 mg S; and 20 mg L and 20 mg S/kg (1 to 1 and 1 to 2 ratio) gave 100% protection against MG infection and the lowest index of airsacculitis (1).

Early Chick Mortality - Four ratios of LS were further evaluated against early chick mortality caused by single and dual SA and EC infections (Table 5). For SA infection, all the antibiotics tested reduced mortality, however, L or LS was more effective in reducing mortality than S ($P < 0.05$). In addition, L and LS were equally effective in reducing mortality caused by SA. For EC infection, chicks treated with S or any dose of LS except 1.25 mg L and 5.0 mg S/bird had a lower mortality than chicks treated with L. Furthermore, L and S alone contributed in part to the total LS activity against the dual infection (3). Chicks treated with LS at 2.5 mg L and 5.0 mg S per bird had a lower mortality rate than chicks treated with either L or S ($P < 0.05$). LS in a 1 to 2 ratio was consistently more efficacious against the dual infection than the other ratios and was more effective in reducing mortality rate than either L or S alone ($P < 0.05$). The in vitro and in vivo data demonstrated that L contributed more to LS against SA than S did, whereas, S contributed more to LS activity against EC than L did. LS in a 1 to 2 ratio was the ratio of choice to make a positive contribution to the total efficacy of the antibiotic combination.

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Table 1

Minimal Inhibitory Concentration (MIC) and Minimal Lethal (Mycoplasmacidal) Concentration (MLC) of Lincomycin and Spectinomycin on Mycoplasma gallisepticum after Two and Seven Days Incubation (1)

Lincomycin mcg/ml	Spectinomycin mcg/ml											
	0		1		2		3		4		5	
	Days Following Incubation											
	2	7	2	7	2	7	2	7	2	7	2	7
0	+	+	+	+	(-)	+	-	(-)	-	-	-	(-)*
1	+	+	(-)	+	-	(-)	-	(-)*	-	-	-	-
2	(-)	+	-	+	-	-	-	-	-	-	-	-
3	-	+	-	+	-	-	-	-	-	-	-	-
4	-	+	-	(-)*	-	-	-	-	-	-	-	-
5	-	+	-	-	-	-	-	-	-	-	-	-
7	-	(-)	-	-	-	-	-	-	-	-	-	-
10	-	(-)*	-	-	-	-	-	-	-	-	-	-

+ = visible growth; - = no visible growth; (-) = MIC; (-)* = MIC

Table 2

Minimum Inhibitory Concentration (MIC) and Minimum Bactericidal Concentration (MBC) of Lincomycin and Spectinomycin, Alone and in Various Combinations, on *Staph. aureus* and *E. coli.*[A] (3)

Spectinomycin (µg/ml)	Lincomycin (µg/ml)																							
	0.0		0.09		0.19		0.39		0.78		1.56		3.12		6.25		12.50		25.00		50.00		100.00	
	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC	MIC	MBC
Staph. aureus																								
0.00	+	+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.09	+	+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.19	+	+	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.39	+	+	+	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
0.78	+	+	+	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
1.56	+	+	+	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3.12	+	+	+	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
6.25	+	+	+	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
12.50	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
25.00	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
50.00	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
100.00	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
E. coli																								
0.00	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
0.09	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
0.19	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
0.39	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
0.78	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
1.56	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
3.12	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
6.25	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	-	+	-	+
12.50	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+
25.00	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+
50.00	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+
100.00	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+

[A] +, bacterial growth; -, bacterial inhibition

Table 3

Effect of Lincomycin and Spectinomycin on Mortality Rate in Mice Infected with Staphyloccus aureus (4).

Lincomycin mg/kg	Spectinomycin mg/kg					
	0	10	20	40	80	160
0	100.0*	100.0	90.0	90.0	70.0	10.0
2.5	80.0	70.0	70.0	70.0	40.0	0.0
5.0	80.0	40.0	40.0	60.0	30.0	10.0
10.0	50.0	20.0	40.0	50.0	20.0	0.0
20.0	30.0	10.0	0.0	30.0	10.0	0.0
40.0	10.0	0.0	10.0	0.0	0.0	0.0

Table 4

Efficacy of Injectable Lincomycin (L) and Spectinomycin Alone and in Combination (L/S) on Mycoplasma gallisepticum in Ten-Day Old Chickens (1).

Dose mg/kg	% Mortality	AS Index*
L 5	20.0	3.6
L 20	60.0	4.0
S 5	30.0	3.4
S 20	10.0	2.6
L/S 5/5	0.0	0.0
L/S 5/10	0.0	5.3
L/S 10/5	50.0	5.6
L/S 10/10	20.0	3.5
L/S 10/20	0.0	0.1
L/S 20/10	20.0	0.4
L/S 20/20	0.0	0.0
Infected Control	70.0	7.3
Noninfected Control	0.0	0.0

* Score index of airsacculitis.

Table 5

Efficacy of a Single Injection of Lincomycin (L), Spectinomycin (S), and Linco-Spectin (L/S) on Chick Mortality by Experimental *Staph. aureus* (SA) and *E. coli* (EC) Infections.[A] (3)

Drug dose (mg/chick)	SA infection			EC infection			Dual infection			
	% mortality	% SA recov	AWG (g)	% mortality	% EC recov.	AWG (g)	% mortality	% recovery SA	% recovery EC	AWG (g)
0.0[B]	77.0a	76.0a	18.8c	56.0ab	58.0ab	42.2ab	98.0a	55.0a	98.0a	3.5d
L 1.25	10.0c	10.0c	32.3abc	84.0a	84.0a	28.9ab	91.7ab	5.0b	90.0a	12.1cd
L 2.5	11.0c	10.0c	38.3a	48.0ab	58.0ab	45.3a	85.0ab	8.0b	92.0a	17.2bcd
L 5.0	10.0c	11.0c	31.8abc	50.0ab	50.0ab	31.9ab	68.3b	20.0ab	77.0a	22.8abc
L 10.0	7.0c	8.0c	35.1ab	36.0bc	42.0bc	24.0b	71.7ab	8.0b	82.0a	27.2abc
S 5.0	29.0b	32.0b	21.0bc	12.0d	14.0d	32.6ab	20.0cd	13.0b	22.0cb	12.0cd
L/S 1.25/5.0	10.0c	16.0c	29.4abc	26.0cd	24.0cd	26.3ab	21.7c	10.0b	28.0b	29.5abc
L/S 2.5/5.0	4.0c	5.0c	38.9a	14.0d	12.0d	42.8ab	1.7e	3.0b	2.0d	36.3a
L/S 5.0/5.0	3.0c	9.0c	37.2a	14.0d	14.0d	36.6ab	5.0de	8.0b	5.0d	32.7ab
L/S 10.0/5.0	4.0c	8.0c	34.6ab	4.0d	4.0d	37.3ab	10.0cde	3.0b	12.0cd	33.6ab
C.V.%[C]	40.0	37.0	44.0	33.0	33.0	35.0	22.0	58.0	19.0	61.0
Uninoculated[D]	2.0	0.0	38.9	0.0	22.0	32.7	0.0	0.0	3.0	29.7
Vehicle[D]	0.0	0.0	30.4	0.0	20.0	32.7	0.0	0.0	27.0	22.0
Sterile broth[D]	2.5	8.0	41.0	NT[E]	...	...	6.7	10.0	7.0	38.0

[A]Means, within a column, followed by different letters are significantly different at $P < 0.05$.

[B]Infected unmedicated controls.

[C]Coefficient variance %.

[D]Uninfected unmedicated controls; not analyzed statistically (1 to 5 replicates of 5 to 10 birds each)

[E]Not tested.

Each treatment group had 5 to 10 replicates of 10 birds each (total number of chicks used was 2,365).

HOW TO USE ANTI-BACTERIALS MORE EFFECTIVELY

by

Dr. Rocky J. Terry
Technical Veterinarian
Sterwin Laboratories, Inc.
PO Box 1235
Gainesville, Georgia 30503
(404) 534-7356

Anti-bacterial drugs are very important tools for modern poultry operations. The use of certain antibiotics in food animals, however, is being questioned by some groups of scientists and the Food and Drug Administration, because of human health implications. There is concern that the selective pressure of low level exposure to antibiotics would increase the number of drug resistant bacteria in animals which could transfer the drug resistance to bacteria in humans.

Anti-bacterial treatment for E. coli, fowl cholera, and the turkey coryza syndrome in large multiage farms in several different areas of the country have not been as effective as the treatments should be. A literature review was undertaken to find out just how the anti-bacterials could be expected to perform.

A most important discovery was the amount of antibiotic resistance that had developed in bacteria infecting poultry. Because of a constant exposure to antibiotics, there has been an increase in antibiotic resistance in E. coli staphylococcus, salmonella and fowl cholera. A bacterial isolation and sensitivity test is a very inexpensive test when compared to the cost of a thousand dollars worth of antibiotics which did not work.

The anti-bacterial resistance pattern will vary from area to area depending upon anti-bacterial use and the methods of treatment. The following resistance patterns are given as examples of the types and amount of resistance that can occur in one poultry producing area.

The following information obtained from scientific publications or company provided literature is presented so that these anti-bacterials can be used in a more effective and profitable manner.

Understand these terms

Words such as minimum inhibitory concentration (M.I.C.), bacteriocidal and bacteriostatic are important in understanding how to use antibacterial agents.

The M.I.C. is the minimum concentration of drug which will prevent the growth and multiplication of the bacteria. Blood levels should exceed the M.I.C. at least three times. This causes another problem because the blood levels of antibiotics and other antimicrobial agents which can be achieved in poultry are not easily obtained from the literature. It may be that blood levels for some drugs in poultry have never been tested.

A bacteriocidal drug kills the bacteria while a bacteriostatic drug only prevents the growth and multiplication of the bacteria.

The degree of absorption into the blood is another critical factor. Bacteria could be sensitive to a drug, but treatment would be ineffective if proper blood levels could not be reached. The antibiotic could not pass the intestinal barrier into the blood.

Problems in water administration

Water administration of drugs to poultry at this time has several serious problems which need to be re-evaluated. Recommendations for most products specify a set dose, regardless of weight of birds or amount of water consumption.

If a tetracycline is given at 200 milligrams per gallon in the drinking water, a 6- week-old tom turkey weighing 4 pounds would consume 3.6 milligrams per pound of body weight of the drug.

A 20-week-old tom, weighing 25.50 pounds, given the same 200 milligrams per gallon of water, would have an intake of 1.7 milligrams per pound of body weight.

Poultry increase water consumption about 4 to 5% for each 1 degree F rise in air temperature above 70 degrees F. At 90 degrees F, medication intake and cost will be double that obtained at 70 degrees F.

The following information on antibacterials is intended to provide the reader a realistic idea of how antibiotics work and what can be expected of them under field use. This information is not expected of them under field use. This information is not designed to replace company information provided on package labeling.

Some product information needs to be updated. However, the costs incurred by a company to add new poultry uses on a label for a product is becoming prohibitive from the standpoint of cost-benefit basis.

Sulfonamides

The sulfonamides were among the first antibacterial agents introduced into medicine. Sulfonamides, such as sulfaquinoxaline and sulfadimethoxine are readily absorbed and are bacteriostatic. The newer sulfonamides have been improved by increasing palatability and by changing the chemical characteristics to increase length of time treatment levels are maintained.

Successful treatment with sulfonamides is dependent upon the blood and tissue levels of the drug, presence of sensitive bacteria, and an intact defense system of the bird. Active destruction by the white blood cells is required for the ultimate destruction of the infective organism.

The sulfonamides are bacteriostatic, so they only prevent the rapid growth and multiplication of the bacteria. Therefore, they are most effective when used during the early stages of infection. During this period, the cellular (white blood cells) and humoral (antibody) defense mechanisms of the bird are mobilized and act together with the bacteriostatic action of the drug. Sulfonamides are much less effective after the disease has become chronic.

Bacterial resistance can occur after prolonged exposure to a sulfonamide, and this resistance will usually extend to the other sulfonamide drugs. If this happens, an antibiotic sensitivity test should indicate which antibiotic would have the best chance of being effective.

Recommended counter measures to minimize the occurrence of acquired bacterial resistance to sulfonamides include:

1. Avoiding promiscuous use of sulfonamides.
2. Initiating therapy as early in the course of the disease as possible.
3. Using correct levels to establish and maintain bacteriostatic concentrations in birds.

Avoid overdosing with sulfonamides because sulfa toxicity can cause problems in blood formation and with excretion of the drug by the kidneys. New products, such as sulfadimethoxine, have a much wider range between treatment levels and toxicity levels. Because of palatability problems, poultry will drink less water before toxicity problems develop.

Sulfadimethoxine (Agribon®) is available as a water treatment or as a feed additive preventative (Rofenaid®), Rofenaid® is unique in that a potentiator is combined with sulfadimethoxine. Sulfadimethoxine inhibits formation of bacterial folic acid, while ormetoprim (potentiator in Rofenaid) inhibits the reduction of bacterial folic acid. The combined action of the drug combination produces an antibacterial action that is greater than the total activity of the two individual drugs.

Rofenaid® is approved for use only as a preventative against fowl cholera, because preventative level is only one-fourth of the level needed for treatment. If the fowl cholera breaks through the preventative level, then the antibiotic sensitivity of the bacteria should be re-evaluated. Should the bacteria be sensitive to sulfadimethoxine,

Agribon® can be used at a treatment level. If the bacteria is resistant to sulfonamides, chlortetracycline is usually effective at a high level.

Sulfaquinoxaline is still an excellent drug for acute infections susceptible to this sulfa. The dosage is critical because the treatment level is close to toxic levels. Reported toxicities include a complete disappearance of white blood cell precursors in bone marrow, severe anemia and fluid around the heart. When sulfaquinoxaline is used, use extra caution with dosage and length of time the drug is used.

Tetracyclines

The group of antibiotics called tetracyclines are broad spectrum antibiotics which exhibit a wide range of antibacterial effects against both gram-positive bacteria, such as staphylococcus and gram-negative bacteria such as *E. coli* or *Pasteurella multocida*. The tetracyclines are the drug of choice against chlamydiae (bacteria in ornithosis) and have some effects against mycoplasmas. Anti-protozoal activity has also been observed when used at high levels.

The tetracyclines are only partially absorbed in poultry, primarily because calcium binds with the antibiotic, preventing complete absorption. The benefits of chlortetracycline or oxytetracycline can be increased through the use of a potentiator such as sodium sulfate or calcium sulfate, or by decreasing the calcium level of the feed. The potentiator binds with the calcium in the upper intestinal tract, allowing the antibiotic to be absorbed. The use of either a potentiator or low calcium feed will double serum levels.

Serum levels of chlortetracycline also increase in proportion to increase antibiotic level, but only up to a point. A potentiator is not needed if chlortetracycline is given to females in production, but would be needed for the males. To prevent excess chloride and resulting flushing of the intestinal tract levels, salt should not be added to the ration when sodium sulfate is used.

Achieving the needed blood levels of antibacterial drugs can be a problem in trying to correlate expected results with the antibiotic sensitivity tests. The Kirby-Bauer sensitivity test indicates that the M.I.C. would be equal to or less than 4 micrograms per milliliter. Using chlortetracycline at 200 grams per ton with 0.8% calcium, only 0.12 micrograms per milliliter blood level can be achieved. If sodium sulfate is used, approximately 0.24 micrograms per milliliter blood level is reached.

In another similar test using 0.8% calcium in the feed, blood levels of 0.35 micrograms per milliliter at 200 grams chlortetracycline and 0.67 micrograms per milliliter at 500 grams per ton were obtained.

One source listed *Pasteurella multocida* as having an M.I.C. of 0.5 micrograms per milliliter. In order to reach treatment levels, approximately 500 grams of chlortetracycline per ton of feed or 200 grams of chlortetracycline with a low calcium level or a potentiator would have to be used.

If water medication had to be used, 1,000 milligrams per gallon would have to be given to equal 500 grams per ton in the feed.

If an *E. coli* with an M.I.C. of 1 microgram per milliliter was to be treated, there is no way a tetracycline antibiotic could be used. The *E. coli* could be sensitive to furazolidone or a sulfa drug.

Erythromycin

Erythromycin, marketed as Gallamycin®, is bacteriostatic and has a narrower spectrum than the tetracycline group. The spectrum of activity is primarily against *Staphylococcus aureus* and erysipelas on the gram-positive side, and it can also be effective for the gram-negative *Pasteurella multocida* (fowl cholera). This antibiotic appears to concentrate in the liver and lungs and maintains a very low blood level.

Novobiocin

Novobiocin is a narrow spectrum antibiotic which is used in poultry for prevention or treatment of staph and fowl cholera. Cost is a factor in its use in poultry.

Neomycin

Because neomycin is poorly absorbed into the blood, it is used primarily to treat E. coli and salmonella infections in the intestinal tract. In poultry, E. coli infections are usually secondary infections of the respiratory system by aerosol route, and neomycin given orally would not be very effective. Neomycin persists in the tissues for a prolonged period if injected.

Spectinomycin

Spectinomycin has been used to control respiratory diseases such as E. coli, Salmonella typhimurium and fowl cholera in poultry, and it is used to prevent Arizona infections in day-old turkey poults and Mycoplasma synoviae, S. typhimurium and E. coli in day-old chicks. The combination of lincomycin and spectinomycin is reported to be more effective for Mg. and E. coli infected chickens than either lincomycin, spectinomycin or tylosin used alone.

Furazolidone

The nitrofurans are synthetic compounds which possess antimicrobial activity through interference with the bacterial enzyme systems. Furazolidone is used primarily for treatment of salmonella and E. coli infections. This drug does not eliminate all salmonella infections, and intestinal shedding can still occur. The levels of furazolidone in blood and tissues are never very high because of rapid metabolism and the fact that the drug does not persist in the body more than 24 hours.

Furazolidone at 400 parts per million (ppm) produced neurological symptoms when fed in combination of either zoalene (1 to 5 ppm) or amprolium (125 ppm).

Summary

The antibacterial drugs have a special place in food animal production. These drugs will be much more cost effective if the producer knows what disease is being treated and how to use them. The antibiotic sensitivity test is a very important tool in closing treatment. Management methods such as ventilation, ammonia levels, bird density and temperature should be constantly monitored.

ANTIBIOTIC RESISTANCE PATTERN OF STAPH IN TURKEYS

Antibiotics	Number Cultures	% Resistant
Penicillin	13	69
Streptomycin	13	77
Tetracycline Group	13	85
Erythromycin	12	33
Novobiocin	13	46
Sulfadimethoxine	13	100

PASTEURELLA ANTIBIOTIC SUSCEPTIBILITY

Antibiotic	Type 3 #1	Type 3 #2
Penicillin	Resistant	Resistant
Streptomycin	Resistant	Resistant
Sulfa Group	Resistant	Resistant
Erythromycin	Sensitive	Resistant
Tetracycline	Sensitive	Resistant

ANTIBIOTIC RESISTANCE PATTERN OF E. Coli IN CHICKENS

Antibiotic	Number Cultures	% Resistant
Penicillin	28	100
Chlortetracycline	28	96
Furoxone	28	14
Neomycin	28	11
Erythromycin	28	82
Sulfa Dimethoxine	26	100

VIRGINIAMYCIN PROPHYLAXIS IN NECROTIC ENTERITIS

*Davis, R; **Oakley, R.G; **Free, M; **Miller, C; and **Rivera, R.

*Department of Avian Medicine
College of Veterinary Medicine
The University of Georgia
Athens, GA 30605

**SmithKline Animal Health Products
P. O. Box 7929
Philadelphia, PA 19101

ABSTRACT

Introduction: In 1977 Harris found that *in vitro C. perfringens* type C required a minimal inhibitory concentration (MIC) of 0.2 mg/ml of Virginiamycin. It was then of interest to see the effect of Virginiamycin (VAM) in farms experiencing necrotic enteritis.

Materials and Methods: Twenty-four pens with six replications of each programme in a randomized block design were used to compare four feeding programmes: 1) control feed was regular non-supplemented, 2) 7.5 gm/ton Virginiamycin, 3) 12.5 gm/ton Virginiamycin, and 4) 20.0 gm/ton Virginiamycin. Each pen, started with 70 chicks, reduced to 58 chickens after two weeks.

Chicken counts, total weight and feed consumption data were recorded for each pen at two weeks, four weeks and seven weeks; at seven weeks counts and weights were recorded by sex. After the first two weeks deaths were recorded by pen and date. Necrotic enteritis deaths were specifically noted. At the end of the study seven birds of each sex were assigned a "shank color" using the Roche Fan.

Conclusions: VAM supplements from 7.5 - 20.0 ppm has a measurable prophylactic effect against necrotic enteritis in broiler chickens with higher death rates in the control pens. The higher survival rates in the VAM pens show the benefits reported as greater pen weight, lower feed conversion, and higher shank color scores. Data suggest a dose-response relationship for VAM over the range of doses required.

PROFILAXIS DE LA ENTERITIS NECROTICA CON LA VIRGINIAMICINA.

Resúmen.

Introducción.- En 1977 Harris descubrió que el C. perfringens in vitro típo C, requería de una concentración mínima inhibitoria (C M I) de 0.2 mg/ml. de Virginiamicina, por lo tanto se presentó la necesidad de observar el efecto de la Virginiamicina (VAM) en granjas cuyos animales presentaban problemas de Enteritis Necrótica.
Material y Métodos.- Veinticuatro secciones con seis réplicas de cada programa diseñados al azar fuéron empleadas para comparar cuatro programas de alimentación: 1.- El alimento - control fué regular y no suplementado. 2.- 7.5 gm/ton. de Virginiamicina. 3.- 12.5 gm/ton. de Virginiamicina y 4.- 20.0 gm/ton. de Virginiamicina. Cada sección se inició con 70 pollitos y se redujo a 50 pollos 2 semanas más tarde.
Conteo de pollos, peso total y datos de consumo de alimento fuéron registrados en cada sección a las 2, 4 y 7 semanas. Se anotaron los conteos y pesos por sexo a las 7 semanas.
Se registró la mortalidad después de las primeras 2 semanas por sección y fecha. Las bajas por Enteritis Necrótica fuéron especificamente anotadas al final del estudio 7 aves de cada sexo fuéron calificadas por su "pigmentación en el tarso", empleando el abanico colorimétrico de Roche.
Conclusiones.- Los suplementos con VAM variando de 7.5 a 20.0 p.p.m. tuvieron efectos profilácticos medibles contra la Enteritis Necrótica en pollos de engorda con altas tasas de mortalidad en las secciones control. Las tasas de mayor sobrevivencia en las secciones tratadas con VAM, demuestran los beneficios que se obtienen tales como mayor peso, conversión alimenticia más baja y calificaciones más altas en la pigmentación del tarso. Estos resultados sugiéren que existe una relación entre la dosis y la respuesta cuando se administra la VAM arriba del nivel de las dosis requeridas.

Traducció:cortesía del Dr. Miguel A. Márquez R.

Introducción

La enteritis necrótica en pollos fue primeramente descrita por Parish 1961 (1).

La reproducción de la enfermedad fue hecha por Long y Truscott 1976, (2) y Truscott et al 1977 (3). La prevención de la enfermedad fue investigada por Trammell et al 1975 (4).

En 1977 Harris (5) encontró que in vitro C. perfringens tipo C requiere de una concentración mínima inhibitoria (MIC) de 0.2 mg/ml de Virginiamicina. Es por lo tanto de interés el investigar si la Virginiamicina tiene un efecto profiláctico en granjas que tienen la enfermedad.

Material y Métodos

24 jaulas fueron diseñadas para comparar 4 dietas:

1. Dieta control no suplementada.
2. 7.5 gm/ton de Virginiamicina.
3. 12.5 gm/ton de Virginiamicina.
4. 20.0 gm/ton de Virginiamicina.

Seis replicas al azar de cada dieta fueron utilizadas, todas las jaulas contenían 70 pollos y después de dos semanas fueron reducidas a 68 animales.

Conteo de animales, peso total y consumo de alimento fueron practicados para cada jaula a las 2, 4 y 7 semanas; a las 7 semanas el conteo de animales y el peso fueron hechos para cada sexo.

Los animales muertos fueron contados después de las primeras dos semanas por jaula y por fecha. Muertes por enteritis necrótica fueron anotadas en especial.

Al termino del estudio 7 animales de cada sexo fueron evaluados para pigmentación, utilizando la técnica de Roche.

Resultados

A) Muertes: Existe la sugerencia de una dosis-respuesta en las dosis estudiadas y de éstas contra el control de ($p < .05$).

B) Peso Total: La única comparación que es estadisticamente significativa es cuando Virginiamicina a 20 ppm se comparó con el control. Sin embargo, los datos soportan una respuesta en todos los niveles.

C) Peso Por Ave: El peso por sexo es estadisticamente significativo ($p < .05$) entre el promedio de peso del control (3.40) y el promedio de peso de la Virginiamicina 20 ppm (3.50).

D) Conversión Alimenticia: A la cuarta semana se notó una diferencia entre los tratamientos, los datos fueron ajustados por la proporción de machos en las jaulas, tanto el valor ajustado y el no ajustado fueron iguales.

Cada dieta de Virginiamicina comparada con la dieta control es estadisticamente significativa ($p < .05$).

Cuando Virginiamicina 20 ppm (2.16) y Virginiamicina 12.5 ppm (2. 21) fueron comparados, existió una diferencia estadisticamente significativa ($T = 1.32$) ($p = .10$).

E) Pigmentación: Siete animales de cada sexo fueron escogidos de cada jaula, todas las dietas de Virginiamicina contra el control fueron estadisticamente significativas ($p < .05$). También es estadisticamente significativo entre las distintas dietas de Virginiamicina.

Conclusiones

Virginiamicina 7.5 a 20.0 ppm tiene un efecto profiláctico medible en contra de la enteritis necrótica en el pollo de engorda.

Esto fue demostrado por mayor número de muertes en las dietas control.

En las dietas con Virginiamicina hubo una sobrevivencia mayor, mejor peso por juala, baja conversión alimenticia y mejor pigmentación. Por lo tanto, todos los datos estadísticos demuestran una dosis respuesta de la Virginiamicina.

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TIAMULIN IN THE CHICKEN: A SUMMARY REVIEW OF COMPATIBILITY STUDIES

S. Goff, T. M. James, and M. Merino
(E. R. Squibb and Sons, Inc., Princeton, N.J.
Squibb de Mexico, S. A., Mexico, D. F.)

ABSTRACTO

El fumarato de tiamulina, un antibiótico diterpeno semisintético derivado de la pleuromulina ha evidenciado excelente actividad en el laboratorio y en el campo, contra infecciones por mycoplasma en aves y cerdos.

Se le administra a las aves en solución en el agua de bebida a dosificaciones cuya eficacia ha sido demostrada en el tratamiento y/o prevención de infecciones causadas por *Mycoplasma gallisepticum* y *Mycoplasma synoviae* en pollos parrilleros y pollas de reemplazo.

En los programas de uso práctico, se administra tiamulina en el agua de bebida a concentración de 0.0125% ó 0.025% durante 3 ó 5 días. La administración durante la primera semana de vida del ave sirve para prevenir la enfermedad respiratoria crónica debida a gérmenes de Mycoplasma. Régimenes similares de tratamiento (3 ó 5 días) en pollos en crecimiento son terapéuticamente efectivos contra Mycoplasmas.

En el curso de un estudio de compatibilidad o tolerancia cruzada, se observó que las aves que recibían monensina simultáneamente con fumarato de tiamulina presentaban ataxia y paresis. Los grupos que ingerían ambos compuestos también mostraron incremento de la mortalidad y disminución en el consumo de alimento y tasas de crecimiento. El grado de intolerancia presentado está relacionado con la dosificación.

A través de un programa intensivo de pruebas se ha demostrado que monensina y salinomicina, ambos antibióticos polietéricos anticoccidianos, son los únicos aditivos para el alimento comercialmente disponibles probados hasta la fecha que han evidenciado incompatibilidad cuando se administran a aves que están bajo tratamiento con fumarato de tiamulina. Los signos de incompatibilidad observados con salinomicina son similares a los que ocurren con monensina, solo que de menor severidad.

Con lasalocid (también anticoccidiano polietérico), arprinocid, amprolium, halofuginona, clopidol, decoquinato, robenidina, zoalene y furazolidona, no se presentaron signos de incompatibilidad durante la administración concomitante de fumarato de tiamulina.

La información obtenida en este programa de pruebas de compatibilidad será presentada. Los estudios indican que el fumarato de tiamulina, tal vez potencializa la actividad anticoccidiana de algunos polietéricos y se discutirá el posible mecanismo bioquímico de las reacciones observadas en las aves.

En más de 8 años de trabajo experimental y en más de 2 años de uso práctico en muchos países del mundo, el fumarato de tiamulina ha sido administrado a muchos millones de aves que consumían raciones que contenían el conjunto completo de aditivos estándares para el alimento. Estos incluyen promotores de crecimiento, arsenicales, nitrofuranos, antibióticos, antimicóticos, vitaminas, minerales, etc. Nunca se observaron o reportaron problemas de compatibilidad más que con los dos compuestos polietéricos previamente mencionados.

Esta breve revisión está basada en el trabajo hecho por los doctores L. Cruthers y J. J. O'Connor, del Instituto de Investigación Médica de Squibb y por los doctores G. Laber, H. Walzl, J. G. Meingassner, F. P. Schmook, R. Czok y H. Mieth del Forschungsinstitut Sandoz.

Tiamulin hydrogen fumarate a semi-synthetic diterpene antibiotic derived from pleuromutilin, has exhibited excellent activity in the laboratory and the field against Mycoplasma infections in poultry and swine.

Among the pathogenic Mycoplasmas sensitive to tiamulin hydrogen fumarate are M. gallisepticum and M. synoviae, responsible for Chronic Respiratory Disease (CRD) in chickens, and M. hyopneumoniae, the causative agent in Swine Enzootic Pneumonia. The antibiotic has demonstrated efficacy against these and other pathogens when administered in the drinking water, and also when blended with the feed.

In poultry, drinking water supplemented with 0.0125% or 0.025% tiamulin hydrogen fumarate is administered continuously for 3 to 5 days during the first week of life in a program designed and timed to prevent the development of CRD. Similar periods of administration later in the life cycle of the birds have been shown to treat Mycoplasma infections successfully.

In the course of early development studies, after establishing the effectiveness of tiamulin in the treatment of well controlled laboratory and field infections of M. gallisepticum and M. synoviae work was undertaken to determine the performance of tiamulin hydrogen fumarate administered in the drinking water with the birds consuming commercial feeds containing additives routinely used in poultry production.

The first test was done with healthy birds and feeds containing standard levels of furazolidone and monensin. Tiamulin was given in the drinking water at 0.025% for the first three days of the birds' lives. Details of the experiment are shown in Table 1. There were 80, one-day-old birds in each treatment. Starting on the 2nd day of the test, birds in the two groups receiving feeds containing monensin (125 ppm) and tiamulin in the drinking water showed signs of paresis, ataxia and reduced feed consumption. The mortality figures in these two groups were notably high.

Reviewing the data in Table 1, it can be seen that in this test which was terminated at 10 days, normal weight gains and mortalities were recorded for all treatment groups except those in which there was simultaneous administration of monensin and tiamulin. The toxic signs, high mortalities and reduced weight gains of the birds receiving this particular combination of active agents were then studied further.

The effects of administering the two compounds sequentially and simultaneously were then compared in two groups of 3 week old broilers. Neither group had received tiamulin nor monensin prior to the trial. Tiamulin was given to both groups in the drinking water at 0.025%, for 3 consecutive days (21st-24th). One group received monensin (125 ppm) in the feed continuously from the 21st through the 31st day. The other received a control diet without monensin during tiamulin administration. Monensin supplemented feed was introduced to these birds 24 hours after tiamulin dosage was discontinued. Clinical signs of ataxia and paralysis were seen only in the birds that received the two treatments simultaneously. One death occurred in this group. As shown in Table 2 weight gain was depressed in the birds treated with both compounds concurrently. Sequential administration permitted normal performance.

After establishing that an untoward effect resulted when monensin was present in the feed of birds being treated with tiamulin, other possible combinations were studied. Table 3 presents the data summary when the anticocidials monensin, amprolium, clopidol, robenidine, buquinolate, and decoquinate were present in the feed during tiamulin administration.

The birds (80 per treatment) were started on test at one day of age. Tiamulin was administered to the appropriate groups for 3 consecutive days. Data were recorded for ten days. When amprolium, clopidol, robenidine, buquinolate or decoquinate containg feeds were used during tiamulin treatment, weight gains and feed conversions were normal, and no signs of toxicity occurred.

With monensin at the level of 125 ppm, some of the birds showed typical paralysis and depressed body weight gains. It should be noted that in this set of tests, tiamulin was used at 0.025% and 0.0125% along with monensin. In both instances mortality was within the control or normal range. Weight gains, however, were definitely below controls. In the group receiving tiamulin at 0.0125% plus monensin, while the weight gains recorded were below the controls and other combinations, they were better than those registered by the birds given monensin and the higher concentration of tiamulin in the water. This dose-response relationship will be discussed in some detail later.

The results obtained in the studies carried out to this point indicated that the incompatibility difficulty might be specific for monensin. Since monensin is a polyether, lasalocid, another member of this family of compounds, was also tested. Several experiments were run, and happily, no problems were encountered.

In Table 4 are the data obtained in a test using birds infected with coccidiosis and treated simultaneously with tiamulin and laslocid. Tiamulin was given in the drinking water at both 0.0125% and 0.025% for 5 consecutive days (days 1-5). Lasalocid supplemented feed (125 ppm) was given continuously starting on the first day. Results to 28 days of age were recorded. The data show that no negative interactions resulted from the administration of lasalocid during tiamulin dosage.

A series of trials undertaken by Meingassner and associates investigated the apparent dose-response relationships between the polyethers (monensin, lasalocid) and tiamulin hydrogen fumarate. In this work, the effects of varying dosages of tiamulin and monensin, and tiamulin and lasalocid were studied. The results obtained are presented in detail in "Poultry Science" (1979, see references).

When monensin and tiamulin were administered at their recommended levels (monensin 125 ppm in the feed, tiamulin at 0.0125% in the water) in coccidiosis infected birds, the efficacy of monensin was not affected, but weight gains were lower than normal. With lasalocid, the results were equally striking. With tiamulin in the water at 0.0125%, anticoccidial effectiveness and normal weight gains were achieved with as little as 25 ppm of lasalocid. Wen monensin was held at a constant 125 ppm, normal anticocidial action and weight gains were seen when tiamulin was reduced to 0.00125% in the water. With the level of tiamulin kept constant, but reduced concentrations of monensin in the feed, an unexpected result was found. Both anticoccidial efficacy and normal weight gains were seen when monensin was at the reduced levels of 25-50 ppm.

A liver perfusion study conducted by Meingassner *et al.* indicated that when tiamulin and monensin were present together, the concentration of monensin in the liver tends to increase. Theoretically, this might account for the observed toxicity and the enhancement of activity when the two compounds are administered simultaneously.

Time does not permit detailing all the studies that have been carried out to determine the compounds that may be used safely during tiamulin administration. An overall summary of the findings at this time are shown in Table 5. Aside from monensin, only alinomycin, also a polyether anticoccidial was found to be involved in the development of an incompatibility picture when ingested during tiamulin administration. Some of the birds given salinomycin supplemented feed while being treated with tiamulin showed only mild paresis and moderately decreased weight gains. Increased mortality was not characteristic and the birds recovered rapidly after the tiamulin treatment was completed. In all the other combinations listed, no signs of incompatibility were seen, and weight gains, and feed efficiencies were equal to or better than controls.

In more than 8 years of developmental work and over the two years of practical use in many countries around the world, tiamulin hydrogen fumarate has been administered to many millions of birds that consumed rations containing the full array of standardly used additives. These include growth promoters, arsenicals, nitrofurans, antibiotics, antifungals, vitamins, minerals, etc. No compatibility problems were ever observed or reported other than with the two ionophores compounds discussed.

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TABLE 1

SIMULTANEOUS ADMINISTRATION
TIAMULIN, FURAZOLIDONE, MONENSIN
ONE-DAY-OLD BIRDS - 80 BIRDS PER TREATMENT

Tiamulin % in Water	Furazolidone Feed ppm	Monensin Feed ppm	Mortality at 10 days %	Av. Wt. Gain Gm/bird at 10 days	Wt. Gain Index %
0	0	0	0	122.0	100
0.025	0	0	2.5	118.2	97
0	150	0	0	125.7	103
0	0	125	1.2	124.0	102
0	150	125	0	119.7	98
0.025	150	0	1.2	115.3	95
0.025	0	125	31.2	68.9	57
0.025	150	125	26.2	75.6	62

TABLE 2

SIMULTANEOUS ADMINISTRATION OF TIAMULIN, MONENSIN

Tiamulin % in Water	Administered Days	Monensin Feed ppm	Administered Days	Av. Wt. Gain Days 21 - 31
0.025	21 - 24	125	25 - 31	140.7
0.025	21 - 24	125	21 - 31	49.1*

*1 died in group receiving monensin plus tiamulin.

TABLE 3

SIMULTANEOUS ADMINISTRATION
TIAMULIN AND VARIOUS ANTICOCCIDIAL AGENTS
ONE-DAY-OLD BIRDS 80 BIRDS/TREATMENT

	Tiamulin H_2O, %	Anticoccidial	PPM	Mortality 5 Day %	Av. Wt. Gain Gm/bird 10 Days	Feed Conv. 10 Days
I	0.025	monensin	125	1.2	66.6	1.664
II	0.0125	monensin	125	3.7	78.9	1.555
III	0.025	amprolium	125	1.2	99.0	1.477
IV	--	amprolium	125	2.5	97.1	1.443
V	0.025	clopidol	125	3.7	93.7	1.372
VI	--	clopidol	125	2.5	93.2	1.503
VII	0.025	robenidine	33	1.2	99.2	1.437
VIII	--	robenidine	33	5.0	96.7	1.513
IX	0.025	buquinolate	82.5	2.5	93.0	1.527
X	--	buquinolate	82.5	5.0	90.0	1.541
XI	0.025	decoquinate	30	2.5	97.6	1.362
XII	--	decoquinate	30	2.5	94.5	1.466
XIII	--	--	--	0	95.4	1.548
XIV	--	monensin	125	5.0	95.9	1.451
XV	0.025	--	--	2.5	98.6	1.439
XVI	0.025	--	--	3.7	101.2	1.370

TABLE 4

SIMULTANEOUS ADMINISTRATION OF TIAMULIN, LASALOCID

Birds infected with coccidiosis
Treatments initiated 1st day of age

	Av. Weight Gains		Mortality
TREATMENTS	0-7 Days Gm	0-28 Days Gm	0-28 Days %
Controls, infected, unmed.	34.6	617.5	73
Controls, uninfected, unmed.	89.1	708.1	0
Lasalocid 125 ppm, continuous	85.3	736.8	< 1
Lasalocid 125 ppm cont., plus			
Tiamulin 0.025% x 5 days	89.4	810.1	0
Tiamulin 0.0125% x 5 days	88.4	787.7	< 1
Tiamulin 0.025% x 5 days	37.5	627.3	52.5
Tiamulin 0.0125% x 5 days	29.1	611.3	70

TABLE 5

TIAMULIN: COMPATIBILITY SUMMARY

Feed additives which may be used (compatible) during tiamulin administration:

Lasalocid	Zoalene
Arprinocid	Decoquinate
Amprolium	Nicarbazin
Halofuginone	Furazolidone
Robenidine	Virginiamycin
Clopidol	Gentian Violet
Buquinolate	

Feed additives which should not be used (incompatibile) during tiamulin administation:

Monensin
Salinomycin

ANTICOCCIDIAL EFFICACY OF SALINOMYCIN IN BATTERY AND FLOOR PEN TRIALS

Thomas T. Migaki, D.V.M.
Animal Health Department
Pfizer Inc.
Terre Haute, IN 47808

D. P. Conway, Ph.D., J. P. Raynaud, Dr. Med. Vet and J. Phillippe, Dr. Med. Vet.
Pfizer International Inc.
New York, NY 10017

The anticoccidial efficacy of salinomycin (Coxistac®)* was compared with eight other commercially available drugs in 76 battery trials and with two drugs in 3 floor pen trials. In battery trials, chickens were fed rations containing salinomycin or approved levels of other anticoccidial drugs for one day before and seven days following oral inoculation with sporulated coccidia oocysts. Birds were medicated continuously for eight weeks in floor pens containing litter naturally contaminated by infected seeder birds. Various combinations of the major species of avian coccidia obtained from recent field outbreaks or as pure culture isolates were used in both types of trials. Evaluation of drugs was based upon mortality, lesion score and weight gain for the battery trials and identical parameters plus feed efficiency in the floor pen trials.

Salinomycin at 60 ppm was highly efficacious against coccidiosis induced by a wide diversity of coccidia isolates. In battery study comparisons, salinomycin was superior or equal to other anticoccidial drugs tested (clopidol, halofuginone, lasalocid, monensin, robenidine, amprolium combinations and zoalene). Salinomycin exhibited excellent lesion control and this was consistent against all species. In floor pens, the performance of birds given salinomycin was superior to monensin and halofuginone.

*Trademark of Pfizer Inc.

EFICIENCIA ANTICOCCIDIANA DE LA SALINOMICINA EN SISTEMAS DE BATERIA Y PISO

La eficiencia anticoccidiana de Salinomicina (Coxistac*) fue comparada contra ocho drogas comerciales en 76 pruebas en bateria, y contra dos drogas - en tres pruebas en piso. En los trabajos realizados en batería. Las aves recibieron raciones conteniendo Salinomicina o niveles recomendados de otras anticcodianas desde un día anterior hasta siete días después de la inoculación oral con oocistos esporulados.

Las aves recibieron medicamentos continuamente durante ocho semanas en sistema de piso conteniendo cama contaminada naturalmente por pollos. Tanto en los estudios en baterías como en jaulas fueron utilizadas varias combinaciones de las principales especies de coccidias aviaries obtenidas de brotes de campo o con aislamientos en cultivos puros. La evaluación de la droga se efectuó en base a la mortalidad, grado de lesiones y ganancia de peso en los trabajos en batería; los mismos parámetros, además de la conversión alimenticia, se llevaron a cabo en las pruebas de piso.
La Salinomicina en concentraciones de 60 p.p.m. resultó altamente eficaz contra diversas variedades de coccidias. Comprando los resultados obtenidos en las pruebas de batería, la Salinomicina fue superior o igual a otras drogas experimentadas: Clopidol, Halofuginona, Lasalocid, Monensina, Robenidina, y combinaciones de Amprolium y Zoalene. La Salinomicina demostró excelentes resultados en relación al control de lesiones y fue más consistente contra todas las especies. En los trabajos realizados en piso, el desarrollo de las aves que recibieron Salinomicina fue superior a Monensia y Halofuginona.

* Marca Registrada por Pfizer Inc.

Traducción: Cortesía del Dr. Carlos López Coello.

Introduction

The anticoccidial, salinomycin (Coxistac®)* has been reported to have a broad spectrum of activity in battery and floor pen studies (1,2,3,5,6,8,9). This report summarizes the efficacy of salinomycin when compared directly with other anticoccidial drugs in 76 battery and 3 floor pen trials.

Experimental Methods

Battery trials. Day-old Hubbard broiler cockerels purchased from a commercial hatchery were reared in wire-floored starting batteries until trial initiation. The birds ranged from 9 to 24 days old when weighed and randomly allotted into treatment groups of 3 pens of 10 chicks each. Salinomycin was compared with monensin in 63 trials, halofuginone in 49 trials, amprolium + ethopabate (A+E) in 25 trials, amprolium + ethopabate + sulfaquinoxaline (A+E+S) in 15 trials, lasalocid in 21 trials, and clopidol, robenidine and zoalene in 11 trials each. The drugs were added at the manufacturer's recommended use level to a basal ration containing approximately 22% protein and 2820 kcal of ME/kg. The rations were fed *ad libitum* beginning 1 day before the oral coccidia exposure and continued to trial termination. The coccidia cultures were either recent mixed population field isolates (44 trials) or single species cultures (32 trials). Birds were observed daily and necropsy was conducted on all dead and dying birds to record the cause of death. Seven days after inoculation the surviving birds were weighed, sacrificed and the intestinal tract lesions were scored by the method of Johnson and Reid (4,7).

Floor pen trials. Day-old Hubbard chicks were randomly allotted to 4 pens (2 male and 2 female) of 55 chicks each. The treatments in each trial were: nonmedicated and medicated with either salinomycin, monensin or halofuginone. The medications were given continouosly in the feed *ad libitum* for 8 weeks at the recommended use level. The basal rations contained 21% protein and 3200 kcal ME/kg in the starter and 21% protein and 3295 Kcal ME/kg in the finisher. The chicks were kept in the back of the pens for the first 15 days of test. During this time, 8 seeder birds inoculated with either Coccivac D (*Eimeria acervulina*, *E. brunetti*, *E. hagani*, *E. maxima*, *E. mivati*, *E. necatrix*, *E. praecox*, and *E. tenella*) at 5 X the recommended vaccination dose (1 trial) or with a mixture of *E. acervulina*, *E. necatrix* and *E. tenella*) at levels of $1x10^5$, $1.5x10^4$ and $1x10^4$ oocysts/bird, respectively (2 trials) were placed in a cage on a wood stand in the front of each pen. The seeder birds were removed after 10 days. The litter under the seeder cage in each pen was kept moist and aerated by daily spraying of water and stirring of the litter with a rake. On day 15 of test, the contaminated litter was spread evenly in the pen and the test chicks were given access to the entire pen. The number of birds in each pen was adjusted to 50 at this time. In trial 3, the litter in each pen was further contaminated by addition of watery feces containing sporulated oocysts of *E. necatrix*.

Criteria for evaluation of drug performance were similar to those used in battery trials with the addition of feed efficiency. Lesions were scored on either day 23 or day 28.

Results and Discussion

Battery trials. The results of all trial comparisons are summarized in Table 1. The high lesion score (2.91 to 4.44) and mortality (4.5 to 16.5%) in nonmedicated birds demonstrate the pathogenicity of the various combinations of coccidia used. Salinomycin, monensin, halofuginone, lasalocid, clopidol and robenidine effectively controlled lesions of coccidiosis, while amprolium combinations and zoalene were less effective. Salinomycin was significantly ($P<.05$) or numerically superior in comparison with other anticoccidials in reducing lesion scores. The weight gains for birds in the medicated treatments were significantly ($P<.05$) greater than the gains for the nonmedicated treatment. Weight gains by salinomycin treated birds were greater than or equal to the other medicated treatments except for lasalocid.

TABLE 1 - EFFICACY OF SALINOMYCIN COMPARED TO EIGHT ANTICOCCIDIAL DRUGS IN EIGHT-DAY BATTERY TRIALS.

Treatments*	No. of Trials	Lesion Score		Percent Coccidiosis Mortality**	Average Weight Gain	
		Av.**	% Red.		Grams**	Index
Nonmedicated	63	4.18^{c}	--	14.8^{b}	152^{c}	100
Salinomycin, 60 ppm	63	$.30^{a}$	92.8	$.1^{a}$	270^{a}	178
Monensin, 100 ppm	63	$.58^{b}$	86.1	$.3^{a}$	254^{b}	167
Nonmedicated	49	4.12^{c}	--	10.6^{b}	172^{b}	100
Salinomycin, 60 ppm	49	$.22^{a}$	94.7	$.0^{a}$	273^{a}	159
Halofuginone, 3 ppm	49	$.66^{b}$	84.0	$.0^{a}$	287^{a}	167
Nonmedicated	25	4.44^{c}	--	14.4^{b}	167^{c}	100
Salinomycin, 60 ppm	25	$.33^{a}$	92.6	$.1^{a}$	272^{a}	163
A+E, 125 ppm	25	3.38^{b}	23.9	$.9^{a}$	225^{b}	135
Nonmedicated	15	4.14^{c}	--	7.8^{b}	182^{b}	100
Salinomycin, 60 ppm	15	$.14^{a}$	96.6	$.0^{a}$	276^{a}	152
A+E+S, 165 ppm	15	1.75^{b}	57.7	$.4^{a}$	280^{a}	154
Nonmedicated	21	4.39^{c}	--	16.5^{b}	171^{c}	100
Salinomycin, 60 ppm	21	$.38^{a}$	91.3	$.1^{a}$	275^{b}	161
Lasalocid, 100 ppm	21	1.28^{b}	70.8	$.5^{a}$	306^{a}	179
Nonmedicated	11	2.91^{b}	--	4.5^{a}	224^{b}	100
Salinomycin, 60 ppm	11	$.02^{a}$	99.3	$.0^{a}$	305^{a}	136
Clopidol, 125 ppm	11	$.65^{a}$	77.7	$.0^{a}$	313^{a}	140
Nonmedicated	11	2.91^{b}	--	4.5^{a}	224^{b}	100
Salinomycin, 60 ppm	11	$.02^{a}$	99.3	$.0^{a}$	305^{a}	136
Robenidine, 33 ppm	11	$.56^{a}$	80.8	$.0^{a}$	312^{a}	139
Nonmedicated	11	2.91^{c}	--	4.5^{a}	224^{b}	100
Salinomycin, 60 ppm	11	$.02^{a}$	99.3	$.0^{a}$	305^{a}	136
Zoalene, 125 ppm	11	2.15^{b}	26.1	$.3^{a}$	285^{a}	127

*All birds inoculated with coccidia seven days before trial termintion.

**Values with the same superscript are not different at the 5% level of significance.

Floor pen trials. Coccidia lesions were observed in the birds removed in each test, but the severity overall was moderate (Table 2). All medications were efficacious in reducing coccidia lesions as compared with the nonmedicated birds. Salinomycin and halofuginone were more efficacious than monensin in preventing coccidia lesions and mortality. Birds given salinomycin were heavier than birds in all other treatments (Table 2), but the differences in total weight gain were not statistically significant ($P>.05$). The feed conversion for halofuginone was significantly improved ($P<.05$) compared to the nonmedicated treatment, but salinomycin and monensin were significantly ($P<.05$) more efficient than halofuginone.

TABLE 2 - SUMMARY OF LESION SCORE, MORTALITY, WEIGHT GAIN AND FEED EFFICIENCY FOR 3 FLOOR PEN TRIALS

Treatment	Lesion Score		Percent Coccidiosis	Average Weight Gain		Average Feed Conversion	
	Av.	% Red.	Mortality	Kg*	Index	F/G*	Index
Nonmedicated	3.48	--	10.0	2.18[a]	100.0	2.31[c]	100.0
Salinomycin 60 ppm	1.12	67.8	0.0	2.25[a]	103.5	2.20[a]	104.8
Monensin 100 ppm	1.68	51.7	0.5	2.19[a]	100.7	2.21[a]	104.4
Halofuginone 3 ppm	0.85	75.6	0.0	2.21[a]	101.7	2.25[b]	102.4

*Any two means with the same superscript are not significantly different at the 5% level of probability.

Conclusion

Salinomycin at 60 ppm was highly efficacious against coccidiosis induced by a variety of *Eimeria* species isolates. In battery comparisons, salinomycin was superior or equal to the other anticoccidial drugs tested (clopidol, halofuginone, lasalocid, monensin, robenidine, amprolium combinations and zoalene), and its excellent lesion control was exhibited and found consistent against all species. In floor pen trials, the performance of salinomycin was superior to monensin and halofuginone.

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EFFICACY OF SALINOMYCIN IN CONTROLLING COCCIDIOSIS IN BROILERS RAISED UNDER COMMERCIAL CONDITIONS IN LATIN AMERICA.

Robert C. Wornick, Pfizer Latin America, Coral Gables, Fla. 33134
Willer P. Coelho, Pfizer S. A., Guarulhos, Brazil
Jorge Parada A., Pfizer S.A. de C.V., Mexico D.F.
Carlos R. Ramis, Pfizer S.A.C.I., Buenos Aires, Argentina
Jorge M. Castro, Pfizer S. A., San José/Heredia, Costa Rica
Hugo Perez G., Pfizer S. A., Valencia, Venezuela

Summary

Salinomycin (COXISTAC*) is a new member of the ionophore family. It has been shown to exert a coccidiocidal effect on all major species of poultry *Eimeria*. Preliminary trials in the United States and Latin America, in batteries and in floor pens, have established its efficacy for preventing coccidiosis in broilers.

This report summarizes the results of 102 field trials, involving 1,956,057 birds, which were conducted throughout Latin America during 1978-79. These trials involved different geographical areas, times of year, feed formulations, breeds of chicks and management conditions. The data confirm the high anticoccidial efficacy of 60 ppm salinomycin in the feed, based on weight gain, feed conversion, reduced mortality and clinical observations.

Furthermore, there were no problems during these trials with growth depression, poor feathering, reduced feed palatability, difficult feed mixing, or wet droppings as has been associated with other anticoccidials. The data also show a small, but consistent, superiority of salinomycin over monensin and other anticoccidial programs, in almost every comparative trial conducted during the past two years. This superiority was statistically significant ($P<0.001$).

*COXISTAC is the trademark of Pfizer Inc., New York, N. Y. for salinomycin

EFICACIA DE SALINOMICINA EN EL CONTROL DE COCCIDIOSIS EN POLLOS PARRILLEROS BAJO CONDICIONES COMERCIALES IN AMERICA LATINA.

Resumen

Salinomicina (Coxistac*) es un nuevo miembro de la familia de los ionoforos. Ha demostrado ejercer un efecto coccidiocida sobre todas las especies principales de *Eimeria* que afectan la avicultura. Pruebas preliminares en los Estados Unidos y en América Latina, en baterías y en corral, han establecido la eficacia de salinomicina en la prevención de coccidiosis en pollos parrilleros.

Este reporte resume los resultados de 102 pruebas en el campo, que incluyeron 1,956,057 pollos, conducidas en toda la América Latina durante 1978-1979. Estas pruebas incluyeron diferentes áereas geograficas, épocas del año, formulaciones de los alimentos, razas de pollos y condiciones de manejo. Los datos obtenidos confirman la alta eficacia coccidiocida de 60 ppm de salinomicina en el alimento, basada en la ganacia de peso, conversión alimenticia, reducción de la mortalidad, y observaciones clínicas.

Además, durante estas pruebas, no se presentó ningún problema del depresión en el crecimiento, pobre emplume, reducción en el consume del alimento, mezcla del mismo o heces húmedas, casos que se ven asociados con otros agentes anticoccidiales. Los datos mostraron también una pequeña pero consistente superioridad de salinomicina sobre monensina y otros programs anticoccidiales en casi todas las pruebas comparativas llevadas a cabo durante los dos años pasados. Esta superioridad fue significativa estadísticamente ($P<0.001$).

*COXISTAC es la marca registrada de Pfizer Inc., Nueva York, para salinomicina.

Introduction

The ionophores are a relatively large chemical family. Only a few members of this family have been found to exhibit potent anticoccidial activity (14, 20). The newest anticoccidial ionophore to be discovered and developed is salinomycin (COXISTAC*)(8,15).

Various chemical studies have shown the structure of salinomycin to be a carboxylic polyether ionophore (12, 16). The ionophores have been found to possess the ability to transport alkali cations across cell membranes (13, 19). This ion transfer disrupts the biological system within the cell. It is believed to be the mechanism of action of the anticoccidial ionophores and the reason why resistance in the Eimeria species to these unique compounds has not been encountered (9).

The anticoccidial efficacy of salinomycin was initially established in battery trials (5, 10, 17, 18). This activity was also confirmed in floor pen trials (1, 2, 6). Safety studies have been reported (11) and also the results of preliminary field trials in the United States (4).

The site of action of salinomycin has also been established (3). A coccidiocidal effect was demonstrated with action, early in the life cycle of the parasite, against sporozoites and early schizonts.

Definitive evaluation of any anticoccidial requires testing in large numbers of birds under a wide variety of commercial conditions. Many of the problems associated with products now in use were only discovered and reported after extensive field trials. These problems include varying efficacy against field strains of Eimeria, development of resistance, growth depression, poor feathering, decreased feed palatability, feed mixing problems and wet droppings.

A three year trial program with salinomycin was begun throughout Latin America in 1977. Comparisons were made with other anticoccidials used alone and also combined in various "shuttle programs". This paper summarizes the results from 102 field trials, with 1,956,057 birds (Table 1). Many additional trials are still in progress and others will be initiated soon.

Table 1. Coxistac Field Trial Summary++

Country	No. Trials	No. Birds
ARGENTINA	4	46,380
BRAZIL	80	1,411,454
CENTRAL AMERICA	6	98,897
MEXICO	10	339,326
VENEZUELA	2	60,000
TOTAL:	102	1,956,057

*COXISTAC is the trademark of Pfizer Inc., New York, N. Y. for salinomycin.
++Does not include battery or floor pen trials.

Materials and Methods

Confirmatory Trials: Regular broiler chicks received commercial feed containing 60 ppm salinomycin continuously during each trial. Commercial broiler houses were used and all birds in each individual trial received the same feed. From 10 to 25 birds from each house were sacrificed at 4-5 weeks, depending on flock size, for lesion scoring (7). Body weights, feed consumption and mortality were determined for each trial. In several cases, successive flocks of birds were tested consecutively in the same houses. The purpose of these trials was to detect any possible adverse reactions when Coxistac was fed to large numbers of birds under widely varying commercial conditions.

Commercial Trials: Same procedure as for "Confirmatory Trials" above. However, similar birds in an adjacent house received the same commercial feed containing another anticoccidial product at the manufacturer's recommended level for comparative purposes.

Products: Salinomycin was provided as Coxistac Premix containing 6% sodium salinomycin activity. Fresh medicated feed was prepared in commercial feed mills for all trials. Competitive anticoccidial products were obtained from regular commercial sources by the feed mills that produced the final feeds.

Results

Confirmatory Trials: The results from 48 trials in Argentina and Brazil, where 872,975 birds received only salinomycin, are summarized in Table 2. Prevention of coccidiosis was highly successful in all 48 trials, based upon clinical observations. Local management conditions varied widely as indicated by feed conversions, ranging from 2.262 and 2.714,

and mortality from 1.72% to 15.28%. Mortality due to coccidiosis was not encountered in any of these trials. There was no evidence of any problems with these 872,975 birds receiving salinomycin, such as depressed growth, poor feed palatability, poor feathering, wet droppings, or feed mixing problems as has been reported with other anticoccidial products.

Table 2. 48 Confirmatory Trials - Performance Data

No. Birds	Days on Feed*	Final Weight (grams)	Feed Conversion	Mortality** (%)
872,975	60	1901	2.494	5.59
Ranges:	(56-67)	(1589-2423)	(2.262-2.714)	(1.72-15.28)

*Commercial feeds contained 60 ppm salinomycin.
**Mortality from all causes other than coccidiosis.

Commercial Trials: Average results of 54 field trials summarizing the performance of salinomycin vs other anticoccidial programs in 1,083,082 birds are presented (Tables 3-6). The performance of 545,955 birds on 11 different anticoccidial programs now used throughout Latin America was compared to 537,127 birds receiving only salinomycin at 60 ppm continuously. The competitive programs included continuous medication with monensin, nicarbazin, halofuginone, lasalocid or amprolium combinations. The "shuttle programs" included monensin/zoalene, monensin/amprolium, monensin/halofuginone, nicarbazin/zoalene or amprolium combinations.

Salinomycin gave performance superior to monensin in 23 trials with 393,922 birds in Brazil, Central America and Venezuela (Table 3). Management conditions varied widely as indicated by feed conversions ranging from 2.115 to 2.958 and mortalities from 1.33 to 10.14%. The superiority was statistically significant ($P<0.05-0.001$).

Table 3. 23 Commercial Trials - Performance Data

	No.	No.	Final Weight		Feed Conversion		Mortality*
Treatment	Birds	Days	Grams	Index	F/G	Index	%
Monensin (90-100 ppm)	199,838	56	1714	100.0	2.419	100.0	3.93
Ranges:		(44-69)	(1376-2066)		(2.115-2.958)		(1.84-10.14)
Salinomycin (60 ppm)	194,084	56	1764	102.9[a]	2.388	101.3[b]	3.72
Ranges:		(44-69)	(1395-2001)		(2.150-2.859)		(1.33-7.64)

*From all other causes other than coccidiosis.
Statistical significance: a = $P<0.001$, b = $P<0.05$.

The results of 10 commercial trials involving 190,577 birds in Brazil and Mexico are presented (Table 4). In these trials, salinomycin fed continuously gave performance superior to "shuttle programs" employing monensin in the starter and either amprolium, zoalene or halofuginone in the finisher ration. Salinomycin was consistently superior even with feed conversions varying from 2.080 to 2.841 and mortality from 1.37-5.9% between trials. The superiority was statistically significant ($P<0.001$).

Table 4. 10 Commercial Trials - Performance Data

	No.	No.	Final Weight		Feed Conversion		Mortality*
Treatment	Birds	Days	Grams	Index	F/G	Index	%
Monensin Shuttles	95,439	56	1731	100.0	2.419	100.0	3.51
Ranges:		(53-65)	(1595-1988)		(2.114-2.841)		(1.37-5.97)
Salinomycin (60 ppm)	95,138	56	1792	103.5[a]	2.356	102.7[b]	3.27
Ranges:		(53-65)	(1636-2080)		(2.080-2.773)		(1.40-5.44)

*From all causes other than coccidiosis.
Statistical significance: a = $P<0.01$, b = $P<0.001$.

The data from 21 commercial trials in Mexico, Argentina, Brazil and Central America with 498,583 birds are presented (Table 5). Again, salinomycin gave performance superior to all other anticoccidial programs which employed halofuginone, nicarbazin, zoalene and sulfaguanidine, or amprolium combinations. Between trial variation was typically high for feed conversions (2.062-2.616) and mortality (1.29-14.12). The superiority was statistically significant ($P<0.10-0.001$).

Table 5.	21 Commercial Trials - Performance Data						
	No.	No.	Final Weight		Feed Conversion		Mortality*
Treatment	Birds	Days	Grams	Index	F/G	Index	%
Non-monensin Programs	250,678	60	1855	100.0	2.436	100.0	6.81
Ranges:		(50-66)	(1276-2240)		(2.165-2.616)		(2.09-14.12)
Salinomycin (60 ppm)	247,905	60	1908	102.9[a]	2.333	104.4[b]	5.93[c]
Ranges:		(50-66)	(1350-2173)		(2.062-2.565)		(1.29-13.97)

*From all causes other than coccidiosis.
Statistical significance: a = $P<0.005$; b = $P<0.001$; c = $P<0.10$.

Table 6 summarizes the results of all 54 commercial trials in Latin America, involving 1,083,082 birds. The 537,127 birds receiving salinomycin showed superior weight gains and feed conversion over all other anticoccidial programs. Feed intake was also equal or better which rules out any feed palatability problems. As was the case in the 48 confirmatory trials (Table 2), there were no instances of growth depression, poor feathering, wet droppings or feed mixing problems in birds receiving salinomycin. This superior performance was consistently obtained under diverse management conditions as exemplified by feed conversions of 2.062 to 2.958 and mortality from causes other than coccidiosis of 1.29-14.12%. This superiority was statistically significant ($P<0.001$). Mortality was also reduced significantly ($P<0.025$).

Lesion Scores: These values were determined in most of the trials and showed Coxistac to be equal or superior to all other anticoccidials in reducing lesions in the intestines and ceca. The scores could not be conveniently presented here because of the voluminous data involved from these many trials.

Table 6.	54 Commercial Trial Summary - Performance Data						
	No.	No.	Final Weight		Feed Conversion		Mortality*
Treatment	Birds	Days	Grams	Index	F/G	Index	%
All Other Programs	545,955	58	1772	100.0	2.426	100.0	4.97
Ranges:		(44-69)	(1276-2240)		(2.114-2.958)		(1.37-14.12)
Salinomycin (60 ppm)	537,127	58	1825	103.0[a]	2.361	102.8[a]	4.50[b]
Ranges:		(44-69)	(1350-2173)		(2.062-2.859)		(1.29-13.97)

*From all causes other than coccidiosis.
Statistical significance: a = $P<0.001$; b = $P<0.025$.

Discussion

The results from the 102 trials presented in this paper, which involved 1,956,057 birds, clearly show that salinomycin is highly effective in preventing coccidiosis in broilers. This efficacy is the same against all of the major Eimeria species in natural infections occurring under widely varied commercial conditions throughout Latin America (Table 1), based on each of the criteria studied.

Furthermore, the data show a small but definite superiority in performance for birds receiving 60 ppm salinomycin over monensin and other anticoccidial programs now used in Latin America. This superiority was determined to be statistically significant ($P<0.001$). It is also important that this superiority has appeared continuously for over two years in almost every battery, floor pen and commercial trial conducted regardless of geographic location, time of year, feed formulation, breed of chick and variation in management conditions.

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HALLAZGO DEL ACARO *ORNITHONYSSUS BURSA* EN GALLINAS PONEDORAS DE MEXICO

Ma. Teresa C. Quintero M. M.V.Z.* y Antonio Acevedo H. M.V.Z. **

*DEPTO. de Parasitología, F.M.V.Z. U.N.A.M. Y Lab. de Acarología E.N.C.B I.P.N.

** Lab. de Parasitología E.N.E.P. Cuautitlán, U.N.A.M.

O. bursa es un ácaro que se localiza en el plumaje de aves domésticas y que también ha sido encontrado en ciartas aves como; canarios, palomas patos y otras aves silvestres, se ha encontrado ocasionalmente en el hombre, ha sido citado de varios paises del continente americano, tales como- E.U., Brasil, Panamá, Costa Rica y Colombia.

El material utilizado en la presente investigación consistió en ácaros colectados sobre aves ponedoras de dos localidades de México: Cuernavaca, Morelos y Tezoatlán, Oaxaca: con este material se realizaron preparaciones empleando líquido de Hoyer, al estudiar estas preparaciones se concluyó que se trataba de *O. bursa*.

Dado que en México no existe a la fecha ninguna publicación sobre este ácaro y ya que las pérdidas económicas causadas por ectoparásitos en la Avicultura son del 4-10%, consideramos de gran interés comunicar que: 1o.- *O. bursa* se encuentra en aves de postura de dos localidades de México.,2o. Insistir en la recomendación de determinar el género de los ácaros parásitos para establecer la forma más adecuada de combate.

A REPORT OF ORNITHONYSSUS BURSA MITE, ON LAYER HENS FROM MEXICO

Ma. Teresa C. Quintero M. M.V.Z.* and Antonio Acevedo H. M.V.Z. **
*Depto. de Parasitología, F.M.V.Z. UN.A.M., y Lab. de Acarología E.N.C.B, I.P.N.
** Depto. de Parasitología, E.N.E.P. Cuautitlán, U.N.A.M.

O. bursa is a mite found on the feathers of domestic fowl also on canaries, pigeons, ducks and other wild birds and domestic birds, occasionally are find it on man. It is found in some countries of America being, -- U.S.A., Brasil, Panamá, Costa Rica and Colombia the main countries.

The material employed in this work consisted of mites collected from layer hens in two localities in México: Cuernavaca, Morelos and Tezoatlán-Oaxaca. whit this material were prepared some microslides with Hoyer fluid - all mites were O. bursa.

There is no existing publications about this specific mite, and due - to the losses which it causes in Aviculture which range from 4-10% (including other ectoparasites) we consider of interest the comunication of the-folling conclutions: 1o. O. bursa exists in laying hens in two localities - of México: Cuernavaca, Morelos and Tezoatlán, Oaxaca 2o. To make emphasis-- on the recomendations as to determine the parasitic genus in order to --- stablish a more adequate control.

Ornithonyssus bursa es un ácaro que parasita en regiones tropicales y subtropicales de los continentes incluyendo diferentes paises, ha sido citado también con los nombres siguientes:

Ornithonyssus bursa Berlese, 1888
Leiognathus bursa Berlese 1888
"Starling mite" (Australia)
"Tropical fowl mite" (E.U.).

HUESPEDES

Este ácaro ha sido encontrado en aves tales como Passer domesticus ó gorrión común (2), canarios, palomas, patos, pavos, gallinas domésticas y también en el hombre (3), (5,6,16).

DISTRIBUCION GEOGRAFICA

Se considera, como ya se mencionó anteriormente, que se localiza en regiones tropicales y subtropicales de los continentes; al revisar la-literatura existente acerca de este ácaro, se encontraron los siguientes datos publicados: en el continente americano, en regiones calurosas de E.U. (3), en Hawaii, (1), Costa Rica, Panamá (17), Puerto Rico-(5), Colombia (17), Venezuela (14), Brasil (4), Argentina (10); En Europa: Gran Bretaña (8), Italia ( ); En Africa en la Región Etiope (11) - en Australia: en Nueva Guinea, Papua, Sidney, Nueva Zelanda (15) y - en Asia: India y Filipinas (7).

MORFOLOGIA

Este ácaro es semejante a O. sylviarum y a O. bacoti, pero se puede diferenciar del primero en la presencia de tres pares de sedas en la - placa esternal y de O. bacoti, en que las sedas de la placa dorsal son más cortas (3).

CICLO BIOLOGICO

Consta de las fases de huevo, larva, ninfa y adulto. O. bursa ponelos huevos fuera del huésped, éstos incuban aproximadamente en 3 dias, de ellos emerge la larva hexápoda que no se alimenta, ésta muda en 8 a 17 horas y pasa a la fase de ninfa octápoda, y de ésta a la fase adulta (9).

EFECTOS PATOLOGICOS

Este ácaro ha sido señalado como la causa de irritación, caida de - las plumas, anemia y especialmente en pollitos, la muerte (3). Según Southcott, 1978 es un ácaro que provoca erupciones comunmente en Australia, este autor considera que esto es debido a que algunos pájaros construyen sus nidos en las ventanas de las habitaciones, sobre techos etc, cuando dichos pájaros abandonan sus nidos, los ácaros que ahí viven se diseminan por las habitaciones atacando al hombre. Otro caso interesante sobre infestaciones en el hombre, fué citado por Fox, 1957, a donde en un Aeropuerto de Puerto Rico se encontró invadido por O. -- bursa; Los ácaros se encontraron en lockers, paredes, en las alas de - los aviones y se menciona que alrededor de 10 personas se vieron afectadas con dermatitis, se citó a los pájaros conocidos como "changos" - como los transmisores de los ácaros.

A este ácaro también se le ha mencionado como transmisor de virus como el de la Encefalomielitis del Oeste Silkin,1945 (12), según Herms's 1972, se han aislado Sindbis virus de O. bursa en la India (15).

ANTECEDENTES

En México se han desarrollado algunos trabajos tendientes a conocer los ácaros ectoparasitos, que afectan a las aves domesticas, en especial en 1977, se llevó a cabo un trabajo sobre la presencia de O. sylviarum - en diversa granjas de la Rep. Mexicana (13), se encontró en todos los casos la presencia de O. sylviarum y fueron en las localidades de: Gómez - Palacio, Durango, Aguascalientes, Leon Guanajuato, Querétaro, Tehuacán, Puebla, Tecamachalco, Puebla; recientemente hemos agregado datos de otras localidades del Edo. de Puebla y del Edo. de México; al realizar la búsqueda de O. sylviarum en aves ponedoras de Tezoatlán, Oaxaca, se nos entregó material de acaros colectados en una explotación a nivel familiar en esa localidad por Mirna Luna casi al mismo tiempo al realizar una visita a una granja de Cuernavaca, Morelos, Jesus Avilés nos entregó otro material colectado de una granja de aves ponedoras de Cuernavaca, que -- sufria problemas de infestación por ácaros.

En ambos casos los ácaros colectados fueron tratados con Na.OH y posteriormente montados en liquido de Hoyer, se realizaron más de 100 preparaciones.

RESULTADOS

Al realizar la observacion de las caracteristicas morfológicas de los ácaros, pudimos determinar que se trataba en ambas colectas de O. Bursa.

Nos encontramos con que aparentemente no existia ningún trabajo publicado sobre la presencia de O. bursa en Mexico por lo que consideramos de interes comunicarlo.

DISCUSION

En México se tienen referencias sobre la presencia de O. sylviarum en diferentes regiones del país, pero no se tenian referencias de O. bursa , por lo que podemos decir que este ácaro está presente presente en aves de Tezoatlán, Oaxaca y en aves de Cuernavaca, Morelos, suponemos que exista en otras regiones de este propio pais; las dos regiones a donde fué - encontrado el ácaro, ambas son consideradas como de clima templado húmedo con lluvias en verano, algunos autores consideran este tipo de clima como semicálido.

El hecho de haber detectado la presencia de este ácaro plantea la posibilidad de que quizá esté pasando desapercibido, ya que si no se realiza el estudio morfológico de los ácaros colectados, puede confundirse -- con O. sylviarum en cuyo caso el ciclo de vida se realiza totalmente sobre el huésped, a diferencia de O. bursa en donde los huevos son depositados sobre el piso.

Otro hecho interesante de señalar es el concerniente a que este ácaro se ha mencionado como habitante de nidos de aves como el gorrión común - ó doméstico; por lo que debe ponerse especial atención a la presencia de estos nidos en las explotaciones, a fin de eliminar la posibilidad de -

infestación a las aves de importancia económica, así como también posi - bles ataques al hombre como los mencionados por Southcott y Fox en Australia y en Puerto Rico.

CONCLUSIONES

1.-Se comunica la presencia de O. bursa en dos regiones de México: Tezoatlán, Oaxaca y Cuernavaca, Morelos, dicho ácaro encontrado en ambos - casos en gallinas ponedoras, en el 1er caso explotación a nivel familiar y en el segundo granja tecnificada.

2.-Se hace la recomendación de determinar el género y especie de los ácaros que se encuentren afectando a aves domésticas, con el fin de realizar en forma más adecuada el combate, ya que en el caso de O. bursa

clo biológico no se realiza totalmente sobre el huésped y deberá por lo tanto ponerse mayor cuidado en las instalaciones de las granjas.

3.-Por último debe ponerse especial atención en la eliminación de nidos hallados en las granjas, ya que en ellos pueden encontrarse los ácaros y en un momento dado cuando los pájaros abandonan los nidos, los ácaros buscarán alimento en las aves domésticas y en el hombre.

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CONTROL OF EXTERNAL POULTRY PESTS -- AN UPDATE WITH SPECIAL EMPHASIS ON NORTHERN FOWL MITE

Edmond C. Loomis, Ph.D., Parasitologist,
Veterinary Medicine Unit, Cooperative Extension
Surge IV Complex, University of California
Davis, California, 95616

Although external poultry pests include a wide range of parasites such as the fowl tick, numerous mites (depluming, red, scaly-leg), turkey chiggers, numerous species of biting lice as well as black flies and mosquitoes, the primary focus of this report will be on Northern Fowl Mite (NFM), Ornithonyssus sylviarum. Because of its general widespread occurrence and economic importance to the poultry layer industry, this mite has become the most important blood-sucking parasite in modern commercial caged layer houses in the temperate zone of many countries. Changes in management and housing of caged layers have made it difficult to achieve as high a level of mite control compared to that obtained during earlier years when laying hens were kept in fewer number, on litter, and on smaller-sized ranches.

NFM control resolved itself to acaricide applications and thoroughness of application has always been of prime importance. Current registered acaricides commonly used in the United States and elsewhere are carbaryl (Sevin®), coumaphos (Co-Ral®), dichlorvos (Vapona®), malathion, nicotene sulfate, and stirofos (Rabon®). Resistance by NFM to malathion, carbaryl, and lately, stirofos, has occurred in some poultry-raising areas of the United States. New classes of compounds such as insect growth regulators and synthetic pyrethroids are currently being investigated. The efficacy and status of these latter compounds will be discussed as will management practices which can aid in NFM control.

CONTROL DE ECTOPARASITOS CONSIDERACIONES ACTUALES, ESPECIALMENTE SOBRE EL ACARO DEL NORTE.
Los ectoparasitos comprenden una gran variedad de parásitos como las garrapatas, numerosos ácaros (ácaros desplumador, ácaro rojo y el de la sarna escamosa de las patas), larvas, piojos, asi como moscas y mosquitos. Sin embargo, el punto de atención en este estudio es el ácaro del norte Ornithonyssus sylviarum.
Debido a su gran distribución geográfica, presentación e importancia económica para la industria avícola, este ácaro se ha convertido en el parásito hematófago más importante en las gallinas de postura en muchos países con zonas templadas. Cambios en los sistemas de explotación y el confinamiento en jaulas de las gallinas han dificultado el control de los ácaros comparando con las primeras granjas avícolas, en las que los animales eran confinados en grupos pequeños bajo sistemas de explotación en piso.
El control y la aplicación de acaricidas ha sido siempre un tema de importancia. Actualmente los acaricidas registrados y comúnmente utilizados en los Estados Unidos de Norteamerica y en otros países son Carbaryl (Sevin), Coumaphos (Co-Ral), dichlorvos (Vapona), malatión, Sulfato de nicotina y Stirofos (Rabon).
Se ha producido resistencia el Acaro del Norte al Malation, Carbaryl y últimamente al Stiropos en algunas zonas avícolas de los Estados Unidos. Nuevas clases de compuestos como reguladores del crecimiento de los insectos y piretroides sintéticos actualemente son investigados. La eficacia de estos últimos compuestos será discutidos así como su administración, lo cual puede auxiliar en el control del Acaro del Norte.

Traducción: Cortesía del Dr. Carlos López Coello.

Ectoparasite control on poultry has been and still is, an important part in bird management and health. In Mexico and the United States, sporadic outbreaks of fowl ticks, lice and red mites (Dermanyssus), are seen on various types of poultry ranches in certain areas. Together, these poultry parasites have declined in importance on a national scale but may remain as common problems usually associated with small ranch operations. Older poultry housing and particularly those with all-wood construction, poor bird management, and a low level of sanitation, have always provided hiding places for, and the transmission of, such parasites as ticks, lice, and mites.

The placing of hens off the ground and away from litter and nest boxes was the first step in helping increase the importance of the northern fowl mite, Ornithonyssus sylviarum. Secondly the rapid trend towards multi-thousand, raised wire-cage layer operations has magnified the northern fowl mite problem on birds. With the present system of multiple cage-row houses, the increased density of birds per cage, and the practice of debeaking hens, management has provided excellent conditions for mite populations and, at the same time has deprived birds from using their natural defenses against this parasite. Another favorable factor for mite survival is the construction of total environmental control housing. Building units designed for greater bird health and maximum egg production have, inadvertently, created year round optimum conditions for mite populations. These changes in management and housing of caged layers have made it more difficult to achieve as high a level of mite control compared to that obtained during the past when layers were kept in fewer numbers, on litter, and on small-sized ranches. It is not surprising, therefore, that DeVaney (1978), in a national survey concerning external parasites of poultry, reported O. sylviarum as the most important parasite of the layer industry in the United States.

In contrast to changes in poultry management, the life cycle of the northern fowl mite has remained the same. This permanent blood-sucking parasite still involves five stages--egg, larva, protonymph, deutonymph, and adult. The period of time for complete development from egg to adult takes about 8 to 11 days. The principal method of transmission has always been from bird to bird and access to poultry housing by wild birds contributes to the transfer of mites. Other methods have resulted from changes in bird management and housing. For example, the suspension system used for raised wire cages inside large layer houses provides mites with greater access to in-row and between-row transfer to more hens. Equipment such as egg crates, carts, and manure removal machinery serve as mechanical carriers of mites within and between houses as well as from ranch to ranch. In a few instances O. sylviarum has been found on rodents and these also may serve as transport hosts for this mite.

The method for detecting permanent parasites on poultry, such as lice or the northern fowl mite, has not changed through the years. Hand inspection of suspect birds quickly shows if these parasites are present. With large commercial layer operations, the presence of mites on eggs is one of the first signs of a mite infestation. The second sign is complaints by egg gathers or workers in egg processing rooms who complain of mites on their bodies. By this time, however, mite populations have reached a dense level of infestation and traditional chemical control methods must be used. Rarely will a layer house manager institute mite control based on a gradual or sudden drop in egg production or feed intake; the occurrence of these latter circumstances are most often associated with the probability of disease, changes in milled-feed rations, and/or associated with changes in lighting, temperature, or water control.

A list of registered acaricides for use against the northern fowl mite is shown in Table 1. Of the nine compounds listed, five are organophosphates--coumaphos, dichlorvos, Malathion, naled and stirofos; one is the carbamate, carbaryl. Nicotene sulfate is a particularly hazardous material and its use is greatly restricted and not economically feasible for mite control on large, commercial layer operations. Sulfur, although not highly efficacious, is a safe material from the standpoint of no residue problems in meat, fat, or eggs and in consideration of bird and human toxicity at the concentration and dosage recommended. The mixture of pyrethrins and piperonyl butoxide can be used for a high level of initial mite mortality but has no residual killing power and is expensive to use.

In some areas of the United States, mite-resistance to malathion has been documented. More recently, reports of carbaryl resistance are known and, as of 1980, reports on the failure of stirofos are not uncommon. One should not, however, blame all acaricide failures as due to mite resistance. Few poultrymen bother to check proper operation of their spray equipment including correct pressure and spray-nozzle delivery. Numerous cases of so-called mite-resistance have proven, upon proper investigation, to actually result from insufficient acaricide coverage of mite-infested birds. Until poultrymen realize this, any new compounds may incur criticism of poor or no mite control.

Table 1   Acaracides Under Federal Register For Northern Fowl Mite Control, USDA, 1979

Chemical	ppm Tolerance Meat	Fat	Eggs	No. Days to Slaughter	No. Days In Treatment Frequency	Application[1]
Calcium Polysulfide	---	Safe	---	0	--	2% D
carbaryl (Sevin)	5	5	Ex.[2]	7	28	1-0.5% Sp, D
coumaphos (Co-Ral)	1	1	0.1	0	--	0.25% Sp, 0.5% D
dichlorvos (Vapona)	0.05	0.05	0.05	0	--	0.12% Sp
Malathion	4	4	0.1	0	7	0.7% Sp, 5% D
naled (Dibrom)	Ex.	Ex.	Ex.	0	--	0.3% Mist Sp
Nicotine Sulphate	Ex.	Ex.	1	0	--	0.5% Sp, 2% D, 40% A.I. Paint, 2 Drops/Bird
Pyrethrins & Piperonyl Butoxide	3	3	1	14	7	0.1% Sp, 2% D
stirofos (Rabon)	0.1	0.75	0.1	0	14	0.5% Sp

1. Sp = Spray, D = Dust
2. Extended

The more recent development of a new class of compounds called "pyrethroids" (permethrin, resmethrin, etc.) has resulted in successful field trials to control O. sylviarum. Reports by Hall et al (1978), Loomis et al (1979), Loomis and Dunning (1980) and Williams and Berry (1980) have shown from 2 to 3 months mite control using as little as 0.05% a.i. sprays of Atroban and Ectiban (permethrin), and Ectrin (fenvalerate). These compounds are not currently registered for use on poultry in the United States. Registered animal systemic insecticides commonly used in the United States for control of cattle grubs (Hypoderma spp.) in cattle, were tested by DeVaney and Ivie (1980) against the northern fowl mite. Single and multiple doses given orally in gelatin capsules or mixed in the feed rations to hens did not control O. sylviarum. Compounds tested were coumaphos, famphur, crufomate, ronnel and phosmet; tri-chlorfon and fenthion were not tested because of their instability in water and feed formulations.

A nonchemical method of controlling O. sylviarum was reported by DeVaney and Beerwinkle (1980). Vent feathers were clipped by the use of hand scissors to within 2 to 3 mm above the body surface of mite-infested hens and roosters. Mite populations on hens but not on roosters were reduced but those on hens were not completely eliminated. The authors suggested either the use of a mechanical device that could be rapidly operated to cut feathers of starter pullets when they are transfered to lay cages or, retention of starter pullets at 24 to 26 weeks of age before transfer to lay houses. Either suggestion involves considerable economic expense in labor or in loss of egg production which most commercial layer operations try to avoid. Unpublished data from tests conducted by several investigators including the present author have shown no northern fowl mite control using extended feeding of registered drugs such as Amprolium, Clopidol (Coyden), and Menensin (Coban). These results should not discourage the search for an acaricidal drug which can be used as a safe but yet effective feed additive for poultry. The short-term feeding of medicated rations during the winter months when O. sylviarum is most prevalent would be the most economic solution affordable to the layer industry.

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DISEASE CONTROL ON LARGE MULTIPLE AGE TURKEY FARMS

M. C. Kumar B.V.Sc. & A.H., Ph.D.
Mile High Turkey Hatchery, Inc.
711 S. Bowen St.
Longmont, CO 80501

Abstract

In recent years the turkey production in United States has been undergoing a change. It has become a year round production instead of producing for the Thanksgiving market. This has necessitated building of large farms with multiple ages. These farms are rarely depopulated. This has resulted in higher mortality due to colibacillosis and other diseases.

To reduce these losses in a company raising three million turkeys a year, the following program was instituted:

a. Isolation of flocks
b. Strict farm security
c. Sanitation and disinfection
d Rodent, predator and wild bird control
e. Depopulation in case of contagious disease

The above program caused a reduction of three percent in losses due to diseases in three million turkeys raised per year.

The program and its benefits will be discussed in detail.

CONTROL DE ENFERMEDADES EN GRANJAS DE PAVOS CON PARVADAS DE DIFERENTES EDADES.

Resúmen.

En los últimos años la producción de pavos en los Estados Unidos ha sufrido una serie de cambios. Se ha convertido en una producción a todo lo largo del año en lugar de una producción exclusivamente para el mercado del Día de Acción de Gracias. Esto ha requerido la construcción de grandes granjas conteniendo, parvadas de edades múltiples, las cuales raramente se encuentran totalmente sin animales, lo cuál ha desencadenado altas mortalidades a causa de la Colibacilosis y otras enfermedades.
Con el objeto de reducir éstas enfermedades, una compañía productora de 3 millones de pavos al año ha implantado el siguiente programa:

a).- Aislamiento de las parvadas.
b).- Control estricto de la granja.
c).- Higiene y desinfección.
d).- Control de roedores, predadores y aves silvestres.
e).- Despoblación en caso de enfermedades contagiosas.

El programa antes mencionado se ha reflejado en la reducción de un 3% de las pérdidas debidas a enfermedades en 3 millones de pavos criados al año.
El programa y sus beneficios se discuten con detalle.

Traducción: Cortesía del Dr. Miguel A. Márquez R.

In recent years the turkey production in United States has been undergoing a change. The production is becoming more and more geared towards year round marketing. This has necessitated building of large multiple age turkey farms. These farms are rarely depopulated. This has resulted in high mortality due to colibacillosis and other respiratory diseases.

Colibacillosis occurs as a respiratory disease. It usually is preceded by Newcastle disease (NCD) or avian influenza or environmental stresses like ammonia, overcrowding, excessive dust in the air, rapid changes in temperature, etc. The losses due to this disease may run very high. In majority of instances of the outbreaks of colibacillosis it was found to be preceded by NCD infection. These outbreaks usually occured during brooding period. Since there is no depopulation of the farms, a program was developed to control the losses due to colibacillosis and other diseases.

The program implemented was as follows:

a. Strict farm security: Traffic between farms was restricted. The farm supervisors were asked to visit only one farm a day and if they were to visit other farms then they were asked to stop at the gate and make inquiries from the farm manager. No equipment was to be moved from farm to farm without disinfection at the points of origin and destination. After moving the equipment it was to be used the following day. Feed truck driver was asked to stay in his cab while making feed delivery on a farm. The farm manager was made responsible for receiving feed.

The employees coming on to the farm were given coveralls and boots for working on the farm and they were discouraged from going home for lunch.

b. Isolation of flocks on the farm: Traffic between brooder houses and the growing houses was limited. The employees taking care of brooder houses were restricted to the brooder houses. The farm manager was asked to go from younger to older flocks and not vice-versa. This was done to avoid lateral spread of diseases from older to younger birds.

c. Sanitation and disinfection: After the houses were empty they were thoroughly cleaned and disinfected and fresh shavings spread in the buildings. Various disinfectants and methods used will be discussed.

d. An ongoing program of rodent, predator and wild bird control was implemented on each farm.

e. Depopulation in case of contagious disease: When a contagious disease like <u>Mycoplasma gallisepticum</u> (MG) infection or avian influenza occurs on a farm then the farm should be depopulated since it is almost impossible to eradicate these diseases by any other means.

In the three million turkeys raised per year there was a reduction in the overall mortality per year from 9.3 to 6.3%.

ESTUDIO SOBRE LA ORNITOFAUNA DE LAS ISLAS GALAPAGOS

MIGUEL CASTRO LAMBOGLIA
ISLA DE SANTA CRUZ
GALAPAGOS, ECUADOR

Se hace 1 descripción de la ornitofauna terrestre y acuática, incluyendo brevemente su habi tat:

1° Gran Pinzón de Tierra. (Geospiza magnirostris)
2° Mediano Pinzón de Tierra. (Geospiza fortis)
3° Gran Pinzón de las Opuntias. (Geospiza conirostris)
4° Pinzón de las Opuntias. (Geospiza scandens)
5° Pinzón de Tierra de Pico Agudo. (Geospiza difficiles)
6° Pinzón Artesano. (Camarhunchus pallidus)
7° Pequeño Pinzón de Tierra. (Geospiza fulinginosa)
8° Cucuve de las Galápagos. (Nesominus parvulus)
9° Gavilán de las Islas Galápagos. (Buteo galapagoensis)
10° Paloma tierrera. (Zenaida galapagoensis)
11° Aguatero. (Coccyzus melacoryphus)
12° Gallereta común. (Gallinula chloropus)
13° Gallereta púrpura. (Porphyrula martinica)
14° Golondrina. (Hirundo rustica)
15° Golondrina púrpura. (Progne subis)
16° Pinguino de las Galápagos. (Spheniscus mendiculus)
17° Pato cuervo de las Galápagos. (Nannopterum harrisi)
18° Albatros. (Diomedea irrorata)
19° Pata Pegada (pterodroma phaeopygia)
20° Piquero Enmascarado. (Sula dactylatra)
21° Piquero de Patas Rojas. (Sula sula)
22° Piquero de Patas Azules. (Sula nebouxii)
23° Rabijunco o Contramestre. (Phaethon aethereus)

STUDY ON THE ORNITHOFAUNA OF THE GALAPAGOS ISLAND

A description is made of the Galapagos Archipelago ornithofauna of land and aquatic birds including their habitat:

1° Large Ground Finch. (Geospiza magnirotris)
2° Medium Ground Finch. (Geospiza fortis)
3° Large Cactus Finch. (Geospiza Conirostris)
4° Cactus Finch. (Geospiza scanders)
5° Sharp-Beaked Ground Finch. (Geospiza difficilis)
6° Woodpecker Finch. (Geospiza pollidus)
7° Mangrove Finch. (Camarhynchus heliobates)
8° Small Ground Finch. (Geospiza fuliginosa)
9° Galapagos Mocking bird (Nesomimus parvulus)
10° Galapagos Hawk. (Buteo galapagoensis)
11° Galapagos Dove. (Zenaida galapagoensis)
12° Dark-Billed Cuckoo. (Coccyzus melaryphus)
13° Common Gallinule. (Gallinula chloropus)
14° Gallinule. (Porphyrula martinica)
15° Barn Swallow. (Hirundo rustica)
16° Purple Martin. (Progne subis)
17° Galapagos Penguin. (Sphenicus mendiculus)
18° Flightless Cormorant. (Nannopterum harrisi)
19° Albatrosses. (Diomedea irrorota)
20° Hawaian Petrel (Pterodroma phaeopygia
21° Masked Booby. (Sula dactylatra)
22° Red-Footed Booby. (Sula sula)
23° Blue-Footed Booby (Sula nebouxii)
24° Red-Billed Tropic Bird. (Phaethon aethereus)

ESTUDIO DE LA ORNITO-FAUNA DE GALAPAGOS

MIGUEL CASTRO L. Biólogo
Santa Cruz, Galápagos, Ecuador.

Se originó con la llegada del sabio inglés Charles Darwin a las Islas en el año de 1835. Con sus observaciones, particularmente a los pinzones en su ambiente, descubrió que ésta especie se había ramificado en varias subespecies debido al medio que los rodeaba, los mismos que son conocidos científicamente como Darwin Finches (Pinzones de Darwin).

GRAN PINZON DE TIERRA-Geospiza magnirostris.- Tiene el pico muy abultado semejando el de un loro, ayudado de sus potentes músculos rompe las semillas duras que las encuentra removiendo la tierra en áreas desérticas donde habita esta ave. El más grande de este género se encuentra en la isla Tower.

MEDIANO PINZON DE TIERRA.-Geospiza fortis.- Esta especie que vive en áreas semi-desérticas se alimenta de semillas blandas que las encuentra en el suelo, como lo hace el magnirostris. Es muy frecuente en todas las islas excepto unos islotes.

GRAN PINZON DE LAS OPUNTIAS-Geospiza conirostris.- Encuéntrase en todas las islas, como predilección en el área de la Opuntias, es muy fácil confundirlo con el fortis. Su alimentación puede ser tanto de semillas como insectos, con preferencia las semillas del fruto de las Opuntias, las mismas que abre con su tosco pico.

PINZON DE LAS OPUNTIAS-Geospiza scandes.- Esta avecilla es muy fácil de reconocer por su pico agudo que le sirve para introducirlo en las flores de las Opuntias, siendo esta su principal alimentación y sin lugar a dudas el polinizador de las Opuntias. Siempre lo podemos apreciar en la época en que florecen las Opuntias, introduciendo su agudo pico dentro de una flor para obtener el néctar; también se alimenta de pequeñas frutas en épocas que el cactus no florece.

PINZON DE TIERRA DE PICO AGUDO-Geospiza difficilis.- Esta pequeña avecilla en algunas islas se ha extinguido, posiblemente debido a los gatos domésticos introducidos en las islas. Merodea siempre en el suelo en busca de semillas.

En la isla Wenman, peñón desértico, con el afán de encontrar humedad destruye la base de las plumas que comienzan a nacer de los piqueros patas verdes, al sangrar eso beben.

PINZON ARTESANO-Camarhunchus pallidus.- Se encuentra en todas las islas mayores. Se alimenta de insectos que los encuentra regularmente en los troncos viejos para extraerlos, cuando no los alcanza con el pico, se ayuda con una espina de Opuntia o una ramita que la podemos llamar herramienta, así excita al insecto a salir del agujero y ponerlo al alcance de su pico.

Una especie similar a ésta y que vive estrictamente en los manglares, tiene el mismo hábito de alimentación, se llama PINZON DEL MANGLE-Camarhynchus heliobates.

PEQUEÑO PINZON DE TIERRA-Geospiza fuliginosa.- Lo vemos prácticamente en todas las islas e islotes, excepto los islotes con extremado suelo árido. Intentan cualquier cosa para sobrevivir; donde hay habitantes, merodean la zona en busca de migajas de pan, incluso entran a los almacenes obteniendo arroz y otros víveres de los sacos; en el suelo buscan semillas o plantas tiernas. En ciertos lugares como Fernandina los vemos alimentándose de los parásitos de las iguanas marinas, lo mismo lo hacen con las tortugas terrestres "Galápagos" en la Isla Pinzón.

Siempre anda en grupos, los he podido apreciar intentando cruzar de una isla a otra; los canales si los cruzan aunque a veces en varios intentos, según el viento.

CUCUVE DE LAS GALAPAGOS-Nesominus parvulus.- Es muy común en todas las islas. Se hace presente a los visitantes por su misma curiosidad y su alimentación depende de cualquier cosa; es muy llamativo verlos luchar con un ciempies hasta aniquilarlo. Cuando anidan las palomas tierreras andan atrás de ellas para robarles los huevos, igualmente cuando las iguanas marinas disputan por el nido, luego que han puesto, los cucuves en tanto destruyen los huevos, los cuales tienen cáscara muy suave, en general huevo que ven tratan de romperlo. Los lentes de las cámaras los atraen bastante. En la isla Floreana se han extinguido debido a los gatos salvajes y en la isla Pinzón han desaparecido desde el siglo pasado, ya que las ratas en busca de alimentos los detruían directamente en el nido. Anidan en los arbustos y en las Opun-

tias.
GAVILAN DE LAS ISLAS GALAPAGOS-Buteo galapagoensis.- Su alimentación depende básicamente de las ratas, pero también se alimenta de iguanas marinas, lagartijas y ciertas aves.
Su ciclo de anidación comienza precisamente con el de las iguanas marinas, siendo este el momento oportuno para atraparlas, de este modo el gavilán tiene la alimentación segura para su polluelos con el nacimiento de las iguanas marinas, las que tan pronto nacen se refugian en los manglares o en la lava para escapar de sus enemigos naturales.
El gavilán reconstruye su nido cada vez que empieza el período de anidación. Cuando el hombre altera el ambiente pertubando el lugar donde éstos habitan, destruyéndolos, los jóvenes son los que sufren, se retiran de la población, incluso emigra a otra isla.
PALOMA TIERRERA-Zenaida galapagoensis.- Siendo su alimentación estrictamente semillas que se encuentran en la tierra siempre anda escarbando el suelo donde en muchos casos es presa del gavilán y la lechuza. A falta de alguna grieta propicia para hacer el nido, lo hacen bajo un pequeño matorral. Es muy notable que el cucuve siempre las persigue con el fin de localizar sus nidos para arrebatarle los huevos, no importa si los pichones recién hayan nacido, igualmente los destruye con su agudo pico.
Casi en todas las islas e islotes se la encuentra pero el mayor número que he podido apreciar ha sido dentro del cráter de Fernandina.
AGUATERO-Coccyzus melacoryphus.- Ave resistente, considera al hombre como su enemigo, siempre huye; es muy difícil apreciarla sin la ayuda de los binoculares. Son frecuentes en las pampas de la isla Isabela, se alimenta de insectos, especialmente los saltamontes.
GALLARETA COMUN-Gallinula chloropus.- Esta es otra de las aves que recientemente llegaron a las islas, se encuentra en los charcos tanto de agua salada (cerca de la orilla)como en las pozas de agua dulce en el interior de las islas. Cuando notan la presencia del hombre huyen desaforadamente, por tanto, es muy difícil fotografiarlas.
Anida al borde de las pozas donde ellas encuentran su alimentación, aunque en el tiempo seco estas aves sufren disminuyéndose considerablemente, al extremo de ser escasas.
GALLARETA PURPURA-Porphyrula martinica.- En enero 1964 al norte de Santa Cruz vi algo flotando, al recoger ésto comprobé que era una gallareta púrpura adulta en estado de descomposición. Un año más tarde, viajando al continente observé que una ave daba vueltas al barco en que viajábamos, al atraparla estaba tan agitada que tres horas más tarde murió. Nuestra posición geográfica era de 300 millas desde la costa firme; era una gallareta púrpura joven, seguramente una vestisca la sacó del continente mar adentro y lo único que le quedaba era volar a favor del viento.
Sin lugar a dudas muchas aves que salen del continente caen al mar agitadas y quizás otras con la ayuda del viento llegan a las Galápagos.
GOLONDRINA-Hirundo rustica.- Estabamos 200 millas al Este de las Galápagos cuando una de estas aves revolotéo el buque posándose en la jarcia, al acercarme nuevamente salió en rápido vuela para regresar más tarde, la estuve observando por más de dos horas, parece que descansaba. Después pude verla alzar el vuelo, dirigiéndose al Oeste siguiendo la corriente del ligero viento hacia las Galápagos.
GOLONDRINA PURPURA-Progne subis.- Esta golondrina que eventualmente visita las Galápagos se la puede ver en la parte alta de las islas mayores, en cráteres y depresiones, desde Septiembre hasta Enero. Refiriéndome al año 1939 una bandada de estas golondrinas llegó a la parte alta de la isla Santa Cruz, fue esa la única ocasión que he podido apreciar un número tan grande, quizás un par de miles, tan sólo permanecieron una semana.
PINGUINO DE LAS GALAPAGOS-Spheniscus mendiculus.- El pinguino de las Galápagos con sus aletas de color azúl obscuro está comprendido en el grupo de las aves marinas siendo su origen la Antítida fueron arrastrados por la corriente de Humboldt llegando a Galápagos donde evolucionaron en una sub-especie. El área en que se encuentran es el Oeste de la isla Isabela y en Fernandina, muy pequeños grupos en otros sectores del Archipiélago. Siempre andan en parejas, posiblemente en las Islas Galápagos su número no alcanza los 600 pares.
PAT CUERVO DE LAS GALAPAGOS-Nannopterum harrisi.- Esta ave seguramente llegó a las islas ayudada por sus alas, las que se atrofiaron por falta de enemigo

natural y por la facilidad de alimentarse. Su alimentación básica son los pulpos que los encuentra en el fondo irregular de la lava.
ALBATROS-Diomedea irrorata.- Es el ave más grande, que aunque se le caracteriza como de las Galápagos sólo viene a reproducirse en la isla Española --- que es el único lugar donde anida. En el programa de conservación, en 1960, marcamos 1600 de estas aves. Considerando el número de éstas, durante el ciclo de anidación, de unos cinco mil pares.
La isla Española ha sido escogida para anidar porque su acantilado da directamente al sur-este y así el albatros aprovecha el viento que golpea el --- acantilado y forma una corriente de ascenso que evita que este corra para despegar como lo hace en las partes planas, por lo pesado de la ave; tiene una envergadura de dos metros.
PATA PEGADA-Pterodroma phaeopygia.- Esta ave que anida en dos puntos muy -- distantes que son las Galápagos y Hawai, se mantiene en alta mar durante -- los meses que no anida, para esto los pares se acercan a la costa próxima - donde fabrican su nido, aprovechándose de cuevas naturales o hacen hoyos en la tierra para depositar sus huevos.

Según las observaciones efectuadas por más de 10 años, esta ave regresa al lugar donde nació o ha anidado; comprobando por aves anilladas que han vuelto al mismo lugar y al mismo nido. Desde que pone el huevo hasta que el polluelo abandona el nido pasan 105 días, aproximadamente unos 15 días antes el adulto abandona el polluelo que comienza a perder peso y así abandona el nido.
AVES MARINAS RESIDENTES.- Tenemos algunas en las Islas Galápagos como son - tres variedades de piqueros.
PIQUERO ENMASCARADO-Sula dactylatra.- Su alimentación la encuentra, entre - las islas, en aguas profundas de preferencia el pez volador. Anidan en los peñones expuestos siempre al viento. En busca de alimentación puede abrirse unas 100 millas.
PIQUERO DE PATAS ROJAS-Sula sula.- Esta ave anida en pocos sitios, construye el nido en los árboles; su mayor número se encuentra en la isla Tower; - es de color obscuro alimentándose por la noche.
Los jóvenes siempre se posan en las embarcaciones que llegan a la isla --- Tower.
PIQUERO DE PATAS AZULES-Sula nebouxii.- Esta ave es la más frecuente y anida en la mayoría de las islas. Su hábito de alimentación es a lo largo de - la costa en áreas de poca profundidad; sin embargo, Galápagos no aporta con la suficiente alimentación y es así que el ciclo de anidación de esta ave - fracasa, manteniendo de este modo un equilibrio ecológico.
Si hablamos en términos de la Ornito-fauna Migratoria no podemos decir que Galápagos está dentro de este movimiento y las aves que podemos apreciar -- cada año llegan accidentalmente, quizás porque perdieron la ruta, otras --- arrastradas por la corriente como el Northen Phalarope-Lobipes lobatus o -- Red-necked, aunque no llegan grandes grupos, en ocasiones los he podido ver en bandas de un par de cientos, hay años también que son escasas.
RABIJUNCO o CONTRAMESTRE-Phaethon aethereus.- Esta ave marina que es residente anida en las grietas que encuentra en los acantilados, sin embargo, - en la isla Tower anida en una área completamente plana en las grietas de la lava.
Por más de un año estuve haciendo observaciones y marcando esta ave; uno de los records más sobresalientes fue una ave que había sido atrapada y puesta en libertad a 500 millas al sur-este de su nido (el ave estaba anillada y - su anillo reportado). Una semana más tarde la misma ave fue rechequeada en el nido.

TREATMENT OF RESPIRATORY ACARIASIS IN CANARY BIRDS

M.V.Z.,M.C.V.,R. MORENO D. Departamento de Producción Animal:Aves
Facultad de Medicina Veterinaria y Zootecnia, U.N.A.M.

SUMMARY.

Sternostoma sp. was found to be the cause of respiratory distress affecting 90% of the canaries in an aviary in México City.
Several drugs were tested in an effort to find one that is effective and easy to administer in drinking water. Groups of 4 birds each were treated three times for 10 day with a 20 day interval period.
Levamisole,Thiabendazole and Neguvon were administered in the drinking water, a doses of 100 mg of Neguvon/lt was found to be the most effective, while 200 mg/lt were found to be lethal for 50% of the birds.
Levamisole and Thiabendazole were less effective, in addition there were problems in solubilizing Thiabendazole.
In a field trial 200 canaries were treated with Neguvon (100 mg /lt), and 200 birds were treated with Levamisole (200 mg/lt).
Ninety percent of those canaries treated with Neguvon recovered, while only 60 % recovered after treatment with Levamisole .

TRATAMIENTO DE LA ACAROSIS DE LAS VIAS RESPIRATORIAS EN CANARIOS

RESUMEN.

En un criadero de canarios de la Ciudad de México el 90% de los pájaros mostraban signos respiratorios en diferente grado, a la necropsia se encontró que estaban parasitados con Sternostoma sp.
Se ensayaron varios medicamentos que pudieran ser efectivos y fáciles de administrar a un gran número de pájaros con este problema.
Se llevaron a cabo dos pruebas en las que se efectuaron tres tratamientos de 10 dias cada uno con descansos de 20 dias.
En una primera prueba terapeútica se ensayo con Levamisol,Neguvon y el-Tiabendazol a diferentes concentraciones en grupos de 4 canarios cada uno.
El tratamiento más efectivo fué el del Neguvon a una dósis de 100 mg/lt , la dosis de 200 mg/lt del mismo medicamento produjo la muerte del 50% de los canarios. Tanto el Levamisol como el Tiabendazol dieron resultados menos efectivos, además el Tiabendazol resultó difícil de administrar en el agua por ser poco soluble.En un segundo experimento se formaron dos grupos de 200 canarios afectados, tratándose un grupo con Neguvon a la dósis de 100 mg/lt y otro con Levamisol a la dosis de 200 mg /lt.
El tratamiento con Neguvon a la dósis de 100 mg /lt dió una efectividad del 90% , mientras que el Levamisol a la dosis de 200 mg/lt fué efectivo en el 60% de los canarios.

TRATAMIENTO DE LA ACAROSIS RESPIRATORIA EN CANARIOS

MORENO, D.R. Departamento de Producción Animal: Aves
Facultad de Medicina Veterinaria y Zootecnia

INTRODUCCION:

La acarosis respiratoria producida por varias especies del genero Sternostoma ha sido reportada tanto en canarios como en otros pájaros de ornato en varios paises del mundo(4,6,7,10,11). Así mismo, se han ensayado varios medicamentos tratando de encontrar algunos con efecto terapeútico en este tipo de parasitosis.De los medicamentos probados, unos se han aplicado por aspersión o nebulización de polvos(3,12),otros se han administrado en el alimento(1,8) , por inyección(9) y otros en el agua de bebida (2,5) . Los resultados obtenidos con los medicamentos administrados en esta última forma han sido muy variables, por lo que se consideró interesante efectuar varios ensayos con otros medicamentos que pudieran ser efectivos en el tratamiento de la acarosis respiratoria y que además fueran de uso práctico para ser administrados a un número elevado de pájaros.

MATERIAL Y METODOS

El presente trabajo se llevó a cabo con canarios de un criadero de la Ciudad de México en donde aproximadamente el 90% de 600 pájaros estaban parasitados en diferentes grados con Sternostoma sp. . El trabajo se realizó en dos etapas; en la primera etapa se ensayó con varios medicamentos - administrados en el agua de bebida a varias concentraciones con grupos de 4 canarios c/u de la siguiente forma : grupo testigo, sin tratamiento.
Grupo utilizando Levamisol con subgrupos medicados con 100,200y 300 mg/lt.
Grupo utilizando Neguvon*con subgrupos medicados con 50,100 y 200 mg/lt.
Grupo utilizando Tiabendazol con subgrupos medicados con 100,200 y 400mg/lt
El consumo de agua medicada fué administrada at libitum y el periodo de tratamiento fué de 10 dias continuos seguidos de 20 dias de descanso,repitiéndose por tres veces. Durante el periodo de tratamiento se observó diariamente a los pájaros para ver cualquier efecto que pudiera producir cada uno de los medicamentos. Al final del periodo de tratamiento se procedió a sacrificar a los canarios para establecer una relación entre los signos clínicos y el grado de parasitosis existente, correlacionado con el grado de efectividad de cada uno de los medicamentos.
En la segunda etapa, se efectuaron ensayos solo con los medicamentos que en la primera etapa del trabajo demostraron tener los mejores efectos terapeúticos para el control del problema.Se formaron grupos con 200 canarios afectados en diferente grado, a los que se proporcionó agua de bebida at libitum, medicada con la dosis mas efectiva de los medicamentos probados inicialmente.Los grupos fueron los siguientes: 1) grupo medicado con Neguvon*a la dosis de 100 mg/lt . 2) grupo medicado con Levamisol a la dosis de 200 mg / lt. El periodo de tratamiento fué de 10 dias continuos, seguidos de 20 dias de descanso, repitiéndose por 3 veces.

RESULTADOS

De los medicamentos probados en la primera etapa de este trabajo se obtuvieron mejores resultados con el Neguvon* a la dosis de 100 mg/lt. y con el Levamisol a la dosis de 200mg/lt. (cuadros 2 y 3).
En la segunda etapa se observó que el Neguvón* tuvo un grado de efectividad del 90% en tanto que la del Levamisol fué del 60% tomando en cuenta la desaparición de los signos clínicos.(cuadro 4). Así mismo, el Neguvon* demostró tener un efecto mas rápido y duradero que el del Levamisol(cuadro 5) .De los medicamentos probados, el Tiabendazol resultó poco práctico en su uso, debido a su baja solubilidad con el agua (cuadro 6)

CUADRO 1. Relación entre signos clínicos y grado de parasitosis(&)

Signos clínicos	Escala de calificación	No.de ácaros a la necropsia
SEVEROS	3+	10 en adelante
LIGEROS	2+	de 5 a 9
AUSENTES	1+	de 1 a 4

*= Triclorfón
(&)= promedio obtenido de 40 canarios

PRUEBA A CUADRO 2

Grado de efectividad del Levamisol,Neguvon y Tiabendazol despues de tres tratamientos

Medicamento	Dosis(mg/lt)	% de efectividad
Levamisol*	100	25
	200	50
	300	50
Neguvon*	50	75
	100	100
	200	50% de mortalidad al 2°dia
Tiabendazol *	100	25
	200	50
	400	50

*= grupos con 4 canarios cada uno

PRUEBA A CUADRO 3

Grado de parasitosis en canarios después de tres tratamientos con Levamisol, Neguvon y Tiabendazol

Medicamento	Dosis (mg/lt)	Grado de parasitosis a la necropsia	
Ninguno	___	4/4	3+
Levamisol	100	3/4	3+
		1/4	2+
	200	3/4	2+
		1/4	1+
	300	3/4	1+
		1/4	0
Neguvon	50	2/4	1+
		2/4	0
	100	1/4	1+
		3/4	0
	200	LETAL	
Tiabendazol	100	3/4	3+
		1/4	2+
	200	3/4	2+
	400	2/4	1+
		2/4	0

PRUEBA B CUADRO 4

Porcentaje de curación en canarios después del tratamiento con Neguvon y Levamisol

Medicamento (grupos con 200 canarios c/u)	Dosis(mg/lt)	Desaparición de signos clínicos en el tratamiento			Total
		1	2	3	
Neguvon	100	60%	25%	5%	90%
Levamisol	200	30%	20%	10%	60%

PRUEBA B CUADRO 5

Observación de signos clínicos 2 meses después del último tratamiento

Medicamento	Signos clínicos
Neguvon	No volvieron a aparecer en el 90% de los canarios En los restantes se redujo la intensidad de los signos en un 50% después del último tratamiento.
Levamisol	Del 60% de los canarios recuperados, volvieron a aparecer un 3% con signos leves(1+) .En los restantes(40%) con signos se redujo la intensidad en un 25% después del último tratamiento.

CUADRO 6 . Inconvenientes de los medicamentos

Medicamento	Dosis(mg/lt)	signos de toxicidad	solubilidad
Levamisol	100	-	+
	200	-	+
	300	-	+
Neguvon	50	-	+
	100	+-	+
	200	LETAL	+
Tiabendazol	100	-	-
	200	-	-
	400	+-	-

DISCUSION.

Se ensayaron varios medicamentos que administrados en el agua de bebida,pudieran ser efectivos en el tratamiento de un problema respiratorio en canarios de un criadero de la Ciudad de México. En este criadero se encontró que el 90% de los pájaros estaban parasitados con Sternostoma sp,, la especie de Sternostoma presente en los canarios de este estudio por el momento no fué establecido, sin embargo se piensa que se trata de -- Sternostoma tracheacolum .
En la primera etapa de este trabajo se formaron varios grupos con 4 canarios cada uno(cuadro 2) para ver el efecto de diferentes dosis de Levamisol, Neguvon y Tiabendazol sobre los pájaros, de modo que si determinada dosis resultaba letal el número de bajas fuera reducido. En la relación entre signos clínicos y grado de parasitosis(cuadros 1 y 5) se observó que no siempre había una relación directa , ya que en algunos casos el problema respiratorio que presentaban los canarios se debía a lesiones crónicas en los sacos aéreos mas que a la presencia de los parásitos,los cuales en algunos casos ya no se encontraban;de donde se sugiere que además del tratamiento contra los ácaros, se administren otros medicamentos para el tratamiento simultaneo de este otro problema. De los medicamentos probados en la primera etapa se encontró que el Neguvon a la dosis de 100 mg/lt de agua dió los mejores resultados,sin embargo este mismo fármaco utilizado en la segunda etapa con un mayor número de pájaros, causó en un 2% de las aves, ligeros signos de toxicidad manifestándose como tristeza,pluma erizada y baja en el consumo de alimento,lo cual no fué observado en la primera etapa con grupos pequeños;por lo que se recomienda que que cuando tales signos aparezcan en algún pájaro mas sensible,se le separe del grupo medicado hasta que desaparezcan los signos y después sea tratado con la dosis de 50mg/lt de Neguvon,teniéndose como un inconveniente que el grado de curación será menor. Para la segunda etapa del trabajo se consideró que los mejores medicamentos fueron el Neguvon y el Levamisol tomando en cuenta la desaparición de signos clínicos y el grado de parasitosis encontrada en la primera etapa. El Tiabendazol fué descartado aún cuando demostró tener efectos terapeúticos, por ser poco soluble en el agua.
En cuanto al porcentaje de curación obtenido con el Neguvon y el Levamisol a la dosis de 100 y 200 mg/lt respectivamente, es posible que se logren mejores resultados si se da un mayor número de tratamientos, sobre todo con el Levamisol,cuyo efecto terapeútico fué mas lento y de menor grado que el Neguvon (cuadro 4 y 5). Así mismo, es importante recalcar que no todos los pájaros que reinciden en los signos clínicos después del tratamiento pueden estar todavía parasitados ,lo cual nos haría pensar en un momento determinado que el medicamento no sirvió. Debemos tener en cuenta las lesiones crónicas que pueden existir principamlente en los sacos aéreos y pulmones y que nos pueden estar dando el cuadro respiratorio.

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RELATIONSHIP BETWEEN POXVIRUS OF PARROTS AND OF OTHER BIRDS

S.B. HITCHNER* V.M.D. and SUSAN L. CLUBB** D.V.M.
* Department of Avian & Aquatic Animal Medicine
New York State College of Veterinary Medicine
Cornell University, Ithaca, N.Y. 14853
** Pet Farm Inc.
7000 N.W. 68 th Avenue
Miami, Florida 33166

Introduction

Although pox has been recognized for a long time as a disease in many avian species,only recently has it been reported in parrots and other psittacine birds. The recognized subgroups of Poxviridae are fowl, turkey, pigeon and canary. The subgroups are antigenically and inmunologically distiguishable, but varying cross relationships do exist which raised the question of whether the avian poxviruses have a common ancestry and the subgroups are a ' modification of the fowl virus (1). Mayr studied the host spectrum of four subgroups in chickens,turkeys,pigeons,ducks and canaries, demostrating the differences but also revealing certain cross relationships (4). Recent outbreaks of pox in parrots opens the question of whether this is another host adapted variant of fowl pox,requiring a distinct subgroup classification.

A review of collected references on avian pox revealed that pox in psittacine birds has been reported only recently. Sharma et al (5) reported on an isolate from parakeets that showed some relationship to canary pox virus. Kraft and Teufel (3) demonstrated pox virus from love birds, and Graham (2) more recently reported a poxvirus infection in a spectacled Amazon parrot (Amazona albifrons).

In this report pox virus was isolated from an outbreak in Blue-fronted Amazon parrots (Amazona aestiva) received from Bolivia. The first signs were an eye infection in a few birds which spread to penmates and eventually resulted in many birds with pasted eyelids and lesoons of wetpox in the pharnyx. The cutaneous eruptions frequently associated with pox in other " avian species were not prominent in this outbreak.

Virus was isolated from eye swabs from affected birds which produced plaques on the CAM of embryinating eggs. Histologic section of CAM lesions showed cytoplasmic inclusions typical for poxvirus. Attemps to infect chickens by wing puncture or brushing virus into festher follicles or on scarified comb were negative. This virus was not infectious by wing web puncutu-

re for two canaries unexposed to pox, or for two that were inmune to canary pox. One of three budgerigars (Melopsittacus undulatus) inoculated by wing web stab gave a suspicious reaction at the site of inoculation. Two budgeri -gars inoculated by brushing virus into the feather follicles showed a slight swelling of a few,but not all,follicles two weeks after application.

To determine what psittacine species are susceptible to the parrot pox, records have been kept on the genera and species in which a clinical diagno sis of diphtheritic pox was made Table 1). Those with the asterisk have al so been exposed by the feather follicle method to the pox virus isolated from the outbreak and were found to be susceptible.

The numerous recent observations of pox in psittacine birds raises the question of whether we are observing the emergence of a new subgroup of Poxviridae.

Table 1

ESPECIES PSITACIDAS SUSCEPTIBLES A LA VIRUELA EN BASE A DIAGNOSTICO CLINICO

Pox Susceptible Psittacine Species Based On Clinical Diagnosis

* Blue Fronted Amazon (Amazona aestiva)
* Red lored Amazon (Amazona autumnalis)
* Yellow naped Amazon (Amazona ochrocephala aurophalliata)
Blue crowned Amazon (Amazona farinosa guatemalae)
* Spectacled Amazon (Amazona albifrons)
Lilac crowned Amazon (Amazona finschi)
* Double yellow headed Amazon (Amazona ochrocephala tresmariae)
Bluehead Pionus (Pionus menstruus)
Dusky Pionus (Pionus fuscus)
Maxinillian (Pionus maximiliani)
White Crowned Pionus (Pionus seniloides)
Red fronted Macaw (Ara rubrogenys)
Hawkhead Parrot (Deroptyus accipitrinus)
* Slenderbill Conure (Enicognathus leptorhynchus)
* Mitred Conure (Aratinga mitrata)
Black Headed Caique (Pionitis melanocephala)
Grey cheeked parakeet (Brotogeris pyrrhopterus)
* Fishers lovebird (Agapornis fisheri)
* Black masked lovebird (Agapornis personta)
* Peachface lovebird (Agapornis roseicollis)
Golden Mantled Rosella (Platycercus eximius)
Red Rumped Parakeet (Psephotus haematonotus)

* Susceptibilidad al virus de loro también demostrado por inoculación del folículo de la pluma.
*Susceptibility to parrot pox also demonstrated by feather follicle inocula tion

RELACION ENTRE POXVIRUS DE LOROS Y DE OTRAS AVES

Introduccion

Aunque la viruela ha sido reconocida por largo tiempo como una enfermedad que afecta a muchas especies aviares recientemente ha sido reportada en lo- ros y otras aves psitacidas.

Los subgrupos reconocidos de Poxvirus son:gallina, pavo, paloma y canario. Estos subgrupos son antigénica e inmunológicamente distinguibles,pero exis- ten varias relaciones cruzadas, lo cual nos hace preguntarnos si los distin tos poxvirus aviares tienen un ancestro común y los subgrupos son una modi ficación del virus de las gallinas (1). Mayr estudió el espectro de huespe- des de cuatro subgrupos en pollos, pavos, palomas,patos y canarios, demos-- trando las diferencias pero también mostrando ciertas relaciones cruzadas (4). Brotes recientes de viruela en loros hacen surgir la pregunta de si és ta es otra variante del virus de gallina adaptada al huésped,requiriendo una distinta clasificación de subgrupo.

Una revisión de referencias sobre viruela aviar revela que esta enferme-- dad en aves psitacidas ha sido reportada sólo recientemente. Sharma et al (5) reportó un aislamiento en periquitos que mostró alguna relación con el virus de canario. Kraft y Teufel (3) obtuvieron virus de la viruela de peri quitos y Graham (2) recientemente informó de una infección de viruela en lo ros (Amazona albifrons).

En este reporte, un virus de viruela fue aislado de un brote el loros *Ama-zona aestiva* provenientes de Bolivia. Los primeros signos fueron una infección ocular en algunas aves, la que se propagó a aves vecinas produciendo eventualmente en muchas de ellas párpados pastosos y lesiones de viruela húmeda en la faringe. Las erupciones cutáneas, frecuentemente asociadas con viruela en otras especies aviares, no fueron prominentes en este brote.

El virus fue aislado a partir de los ojos de animales afectados, produciendo placas en la MCA de embriones. Cortes histológicos de lesiones en MCA mostraron inclusiones citoplasmáticas típicas de poxvirus. Los intentos de infectar aves por punción en el ala o por pincelada ("brochazo") en el folículo de la pluma o en crestas escarificadas fueron negativos. Este virus no causó infección por punción en el ala a los canarios que no habían sido expuestos a viruela ni en los que fueron inmunes a viruela de canario. Uno de tres pericos australianos (*Melopsittacus undulatus*) inoculado por punción en el ala produjo una reacción sospechosa en el sitio de inoculación. Dos de estas mismas aves inoculadas por el folículo de la pluma mostraron una ligera inflamación de algunos, pero no todos los folículos dos semanas después de la aplicación.

Para determinar que especies se aves psitacidas son susceptibles al virus de loro, se ha llevado control de géneros y especies donde se ha diagnosticado clínicamente la viruela diftérica (Tabla 1). Aquellos con un asterisco también han sido expuestos por el método del folículo de la pluma con virus aislado del brote, y fueron encontrados susceptibles.

Las numerosas y recientes observaciones de viruela en aves psitacidas plantean la duda de si estamos observando el surgimiento de un nuevo subgrupo de poxvirus.

Traducción: Cortesía del Dr. Heroldo Palomares Hilton.

EVALUATION OF PROPIONIC ACID AS MOLD GROWTH INHIBITOR IN POULTRY FEEDING.

P.M.V.Z. ROBERTO LUGO N. *
M.V.Z. RENE ROSILES M. **
M.V.Z. LUIS OCAMPO *

* Departamento de Farmacología y Fisiología, Fac.Med. Vet.y Zoot.
** Departamento de Patología, Fac. Med. Vet. y Zoot. Cd.Universitaria,UNAM.

Ninety broilers one-day old were divided in 2 groups and fed with comercially formulated feed which was previously analysed with thin layer chromatography and for mold growth to detect levels and quantity for mycotoxims. A suspension of *Aspergillus flavus* was added to the feed to achieve a final concentration of 360,000 spores/g. All the broilers were fed with this mixture for 63 days, but the feed from one group received supplementation of 1 pound/ton of propionic acid.

The feed consumption and the weight gain of the two groups were carefully monitored. Cuantity and types of molds in the feed as well as aflatoxin B, and ochratoxins A were determined throughout the experiment and histopathological studies were performed in randomly-selected broilers.

Analysis of Variance showed a statistical difference in weight gain between the two groups ($p \leq 0.05$) at the 2nd, 8th, and 9th week (10.83, 29.8 and 6.026 respectively). The weight gain efficiency analysis between the two groups showed no statistical difference throughout the experiment ($p \leq 0.05$).

The analysis of the feed for mycotoxin levels revealed that during the 7 week various bands appeared having a RF larger and o shorter than that corresponding to aflatoxin B_1. No ochratoxins were detected in either groups.

The histopathological study revealed lesions in both groups. However, the lesions were more pronounced in the group which received propionic acid-free feed.

EVALUACION DEL ACIDO PROPIONICO COMO INHIBIDOR DE HONGOS EN ALIMENTO PARA AVES.

Se utilizaron un total de 90 pollos de un día de edad, los cuales fueron divididos al azar en 2 lotes de 45 pollos cada uno. Las 90 aves recibieron alimento comercial previo muestreo del mismo para detectar niveles y clase de micotoxinas por cromatografía de capa fina y cultivo de hongos, el alimento fue contaminado con una suspensión de esporas (*Aspergillus flavus*) para dar un inóculo final de 360,000 esporas por gramo de alimento, el cual fue suministrado a las aves durante 9 semanas; durante la maniobra de inoculación sólo el alimento del lote 2 fue tratado con ácido propiónico a razón de 1 libra/tonelada de alimento permaneciendo el lote 1 sin dicho tratamiento.

Se determinó el consumo de alimento semanalmente en ambos lotes, así como el peso de las aves y los niveles y clases de hongos presentes en el alimento de ambos lotes, se sacrificaron aves al azar de ambos lotes para estudio histopatológico.

Resultados:
En el análisis de varianza de ganancia de peso de ambos lotes durante las 9 semanas, solo se obtuvo una diferencia estadísticamente significativa ($p < 0.05$) en el lote 2 (tratado) con respecto al lote 1 (no tratado) durante la 2a. 8a. y 9a. semana (10.83, 29.8 y 6.026 respectivamente), el análisis de la eficiencia (índice de conversión) entre los 2 lotes no mostró diferencias significativas a lo largo del esperimento ($p < 0.05$).

Al analizar el alimento par la detección de los niveles de micotoxinas, solamente durante la 7a. semana se detectaron algunas bandas con un RF mayor y menor al de la Aflatoxina B_1.

El estudio histopatológico reveló que las aves de ambos lotes presentaron lesiones principalmente del hígado, riñón y bolsa de Fabricio, siendo mucho más intensas y severas en las aves no tratadas.

Current Management Problems in Small Poultry Flocks in New Mexico

D. W. Francis, Ph.D.
Department of Animal and Range Science
New Mexico State University
Las Cruces, NM 88003

In New Mexico, we receive many more disease problems from small flock owners than from large commercial growers, mainly because the commercial flock owners have their own disease specialists or consultants who handle their problems. This leaves small producers and backyard flock owners in need of help. Many people with backyard flocks today have returned to rural living with the thought of producing their own food but with little or no knowledge of how to go about it. So, they have problems.

Seven cases are described all caused by poor nutrition due to flock owners not knowing flock nutritional requirements or trying to economize with feed. The cases came from flocks of 15 to 200 birds and involve poor egg production, calcium-phosphorus deficiency, poor nutrition complicated with coccidiosis, high mortality, poor growth, unthriftiness, and vitamin D deficiency.

Reasons for the poor nutrition, obtained from backyard flock owners, are given, along with related problems that exist and how we try to solve them in New Mexico.

A fowl pox outbreak is also discussed and how it was prevented the following year.

PROBLEMAS COTIDIANOS DE MANEJO EN GRANJAS CON PARVADAS PEQUEÑAS EN EL ESTADO DE NUEVO MEXICO, E.U.A.

Resúmen.

En el Estádo de Nuevo México, recibimos un número mayor de problemas de enfermedades en parvadas pequeñas que de granjas donde se explotan grandes parvadas a nivel comercial, principalmente porque los propietarios de dichas parvadas comerciales poseen su propio servicio de especialistas y consultores que solucionan sus problemas, Esto hace que los pequeños productores requieran de ayuda técnica.

Mucha gente que maneja parvadas de traspatio han retornado a la vida rural con la idea de producir sus propios alimentos pero con muy pocos conocimientos de como hacerlo y consecuentemente tienen problemas.
Se describen siete casos, todos debido a una mala nutrición por desconocimiento de los requerimientos Nutricionales de las aves y por intentos para economizar dinero en el alimento. Dichos casos proceden de parvadas compuestas de 15 a 200 aves y que presentaban una producción de huevo bajas a causa de deficiencias de Cálcio, Fósforo, Mala Nutrición complicada con coccidiosis, alta mortalidad, crecimiento lento y deficiencias en Vitamina D.
Las causas de éstos estados de desnutrición son descritos y se detallan los métodos con los cuáles les intentamos resolverlos en Nuevo México.
Un brote de viruela aviar es igualmente discutido y los métodos para su prevención al año siguiente.

Traducción: Cortesía del Dr. Miguel A. Márquez R.

The number of chickens in New Mexico has increased to more than 2,100,000 from the 800,000 in the middle 1960's. They are concentrated on three farms, each of which employs its own disease consultant. Except for emergencies such as in the early 1970's when exotic Newcastle disease broke out, I do not see large outbreaks of diseases. Instead, I see isolated cases of common diseases every year. With health food faddism and the trend toward rural living, there has been a large increase in the number of backyard flocks. These flocks for the most part contain 25 to 50 chickens.

You would think that people starting a backyard flock would use some common sense and seek out help before they start, if they know nothing about birds. Some do, but from the histories I get when birds are submitted for necropsy or from the telephoned descriptions of conditions, it is obvious that many solicit no help. It is strange that despite the care and emphasis many of these people place on their own food, they neglect their poultry flock.

Here are a few observations regarding mismanagement in backyard flocks that leads to undernourished broilers, layers or turkeys. These may appear elementary, but they are the type of thing one is apt to see frequently in a diagnostic laboratory as the number of backyard flocks increase.

Case 1. In May, 1975, I visited the home of a prospective poultry student who had 14 miscellaneous hens and one male. The birds were poorly feathered and thin, and altogether they produced one or two eggs a day. This was during the time egg production should have been increasing as day length increased. Questioning revealed that the prospective student fed some whole yellow corn at night, kept a bale of alfalfa in the pen for the chickens to pick at, and fed all the trimmings from the vegetables he brought home from the store where he worked in the produce department. I suggested that he change his feeding program, to feed a complete layer ration while phasing out the produce, corn and alfalfa. I also suggested the layer ration be kept before the birds at all times, and that fresh water be given daily after the waterer was washed out. About a month later, he sent a letter reporting that production was 12 or 13 eggs per day and the birds were looking better as new feathers appeared.

Case 2. In December, 1976, two 6.5 month-old Rhode Island pullets from a flock of 200 were presented with the following history: There had been a gradual death loss over the past two months. The birds were allowed to run loose in an area that provided some oat pasture and weeds along the edges of the oat pasture. A small amount of laying mash was fed in the morning, and scratch grain was available all the time. Postmortem examination revealed gizzards filled with fiber and the crops filled with grain. A green stick fracture was demonstrated in the metatarsus of both chickens. The diagnosis was calcium-phosphorus deficiency, based upon the feeding history, condition of the crop, gizzard and metatarsus. The owner was advised to change to a laying mash and forego the use of grain, pasture and other items. I never received any information as to the outcome. This often happens.

Case 3. May, 1977. A flock of 36 layers, ranging from 10 to 18 months of age, had lost their feathers and looked droopy for more than a month. The second bird that died was submitted for postmortem examination. The birds were being fed a ration of half scratch and half 16-percent lay pellets, along with discarded produce from stores. They had been treated for external parasites with Black Leaf 40. Examination revealed a large crop filled with corn and alfalfa. The gizzard contained a small amount of mash. The G.I. tract was hemorrhagic and thickened with mucus. Coccidial oocysts were demonstrated in gut scrapings. Again, this was a case of improper nutrition complicated with coccidiosis.

Case 4. In September, 1977, half a bag of laying pellets was brought to the lab because, the flock owner said, the laying chickens had begun to die "almost immediately" after they had been fed feed from the bag, which was new, from a feed store. A small test was set up with NMSU birds to compare the suspect feed with the standard NMSU ration. The test lasted until the feed was exhausted - two weeks. There were no deaths, and egg production was 7 percent higher in the group receiving the questionable feed than in the group receiving the NMSU ration. It was obvious the problem was due to another cause than the feed.

We had a similar problem come to us from the feed control section of the State Department of Agriculture. This involved some grain that had been reported to kill chickens. We fed it to young chickens for 10 days, until it was used up, but none of the chickens died. It so happened we had a tour visit from our regents during that test, and it made quite an impression.

Case 5. October, 1979. Two 12-week-old turkeys from a flock of 15 were submitted for diagnosis. The history was minimal. The poults had been started on chicken starter and changed at 6 weeks to a chicken grower and grain mix (50/50). In addition, melons, corn stalks, hay and careless weeds were available most of the time. The birds were underweight and unthrifty. One exhibited an excellent case of perosis. The second bird had a broken leg. In addition, the crop and proventriculus were empty. The gizzard was full of stones and the G.I. tract was void of feed. The presence of white feathers indicated a lysine deficiency. In this case, I suggested the owner try to obtain a turkey starter and then change to turkey grower. The problem in New Mexico is that specialty feeds, such as for turkey and game birds, are not available everywhere, if at all.

Case 6. In January, 1980, one Rhode Island Red hen and two bantams were submitted, with a telephoned history that the owner had bought some chickens about a month before and they had started dying. All the birds were light in weight and unthrifty. All three birds presented the same picture at postmortem. The crop was filled with corn, milo, oats and some straw. The gizzards were severely impacted with fiber. Evidence of a few kernels of grain were present, along with a few pebbles. It was another case of failure on the part of the owner to obtain sound advice about feeding the chickens properly.

Case 7. In October, 1977, four 6-week-old S.C. White Leghorn females were presented for diagnosis by a member of the biology department of a neighboring university. They had been raised in a battery. Mortality had been only three. They had been receiving a 15-percent protein layer feed, which was 6 months old. No drugs or vaccines had been used. The birds were unthrifty. Postmortem revealed a green stick fracture of the metatarsus. In addition, the beaks bent easily, bending and indentation of the ribs were observed, and the sternum bent easily. A recommendation was made to go to a starter mash and not use old feed because the birds appeared to have a vitamin D deficiency. The professor was cautioned to always start his chicks on fresh starter feed and store it in refrigerator if it would not be used up within a month.

These are the types of problems that are brought to our diagnostic facility. An attempt has been made to determine why so many nutritionally-oriented problems have developed. While obtaining information from or explaining the problem to the client, I usually hear one or more of the following comments:

1. "I didn't know you had to be so fussy about what chickens ate."

2. "I remember all my mother did was throw out some grain and collect the eggs."

3. "Oh! The laying pellets were too expensive, so I went to grain, which was cheaper."

4. "I thought chickens needed grain. They like it better than the pellets."

5. "I didn't know the starter mash (layer mash) was a complete feed."

6. "No, I never read the label on the bag."

7. "My chickens aren't doing well. It must be something in the feed."

Thus, there is a lot of educational work to be done, to develop an understanding among people with backyard flocks about how they should feed and handle those flocks.

The other problem lies among the feed dealers and county agents. They have more interest in horses, cattle, sheep and hogs than in poultry. So, they know very little about poultry. Some feed dealers refer people to us at the University, but considering the size of the state many people feel it isn't worth contacting us. Once in a while, we get letters thanking us after we've solved a problem. County agents call us from time to time.

In September, 1978, a number of cases of fowl pox were diagnosed in the small flocks in Dona Ana County, where NMSU is located. Chickens and turkeys were both infected. These were observed sporadically through October. I received numerous phone calls requesting information on the disease. It was so widespread that in spite of close inspection, cases appeared at the poultry show at the Southern New Mexico State Fair. These were disqualified and removed as soon as they were observed. I gave the usual explanations regarding fowl pox and suggested that a vaccination program be used the following year. Incidentally, the inspection for health was made by the people in charge of the poultry section. It was their first time and they had no knowledge of fowl pox or lice, which were also observed on a few of the birds. The veterinarian who was checking the large animals came to speak with me about the fowl pox situation as he did not know about the disease in the county. This was after we had the diseased birds removed.

During 1979, I received no calls about fowl pox. Instead the reports were that most people had vaccinated their birds to prevent the disease. The 4-H club members' interest in poultry, at least in Dona Ana County, aided in accomplishing the vaccination. Each leader undertook a neighborhood project of fowl pox vaccination. This way, they used the vaccine efficiently and spread the cost around.

We do see Marek's disease, external and internal parasites, an occasional case of coccidiosis, blackhead, sinusitis and other common diseases. The largest group of problems falls within the management-nutrition group. There are probably several reasons for this. Poultry is not considered important by county agents, vocational agricultural teachers or even the animal science people at the universities in those states where poultry departments have been consolidated with animal sciences departments. Fortunately, in New Mexico, we have fared well. The trend in the future may not be predicted as administrators change.

Many of our problems come to me over the phone, because New Mexico is a large state. The biggest problem is to obtain a good history. Each year, I write at least one news release about how to obtain a diagnosis, and I put the instructions in a monthly newsletter. Again, these are ignored by those in the counties who are in a place to help. From time to time, I have sent out sample history forms, but these are seldom used. Since 1968, there has been no full-time extension poultry specialist. My time is split between teaching, research and extension (15%). Prior to the 1970-71 exotic Newcastle disease outbreak, 10 percent was allotted for extension work. The addition of bureaucratic paperwork has reduced this time.

In this gloom, there is one ray of hope. Should we experience another serious outbreak of some disease in New Mexico, time will be provided to aid in solving the problem.

During the past year or so, there has been much information appearing relative to the quality of education. Perhaps we should look at our own curricula in agriculture and begin to require more solid courses. Those in college training for county agent or vocational agriculture work seem to by-pass subject matter courses and wind up with method courses but lack knowledge of anything to teach. This is a change from earlier days when all agricultural students had to take at least one course in every field. In order to enable our students to meet minor problems face to face or at least provide adequate information to those interested in raising a few chickens or turkeys a broader base of more solid courses should be required.

ENCEPHALOMALACIA -- A REEMERGING FIELD PROBLEM

R.E. Messersmith, DVM and C.R. Adams, Ph.D.
Department of Agriculture and Animal Health
Roche Chemical Division
Hoffman-La Roche Inc.,Nutley, N.J. 07110

The role of vitamin E is attributed primarily to its activity as a tissue antioxidant. Vitamin E deficiency diseases of chickens are encephalomalacia, late embryonic mortality, erythrocyte hemolysis, and steatitis (all attributed to presence of excessive polyunsaturated fat in the diet), exudative diathesis and muscular dystrophy. The latter two diseases also are interrelated with selenium deficiencies. The majority of the naturally occurring active form of vitamin E, alpha tocopherol, is provided in the diet of broilers by corn. This is supplemented by the addition to broiler feeds of dl alpha tocopherol acetate to meet or exceed the NRC requirement of 10IU/kg diet. Because of instability of naturally occurring alpha tocopherol, it is common practice to over-fortify broiler feeds with added vitamin E.

Beginning in early summer, 1979, encephalomalacia became an unusually severe problem in broilers in the southeast and mid-south broiler rearing areas of the U.S. The encephalomalacia generally responds to a combination of vitamin E and selenium administered in the drinking water. Unlike previous years, cases of encephalomalacia continued to occur through late fall and mild early winter '79-80. When feed samples were collected from affected houses and analyzed, vitamin E concentrations are well above the requirement.

Encephalomalacia is a disease of young chickens felt to be produced by polyunsaturated fatty acids, such as linoleic acid, present in unstabilized fats added to the diet. It can be prevented experimentally by vitamin E, ethoxyquin (an antioxidant), and of course by correcting the feed ingredient quality problem. Investigations into proper qualify control of the fat sources have met with varied success, and few broiler producers have increased the amount of vitamin E added to broiler feed. A "pale bird" syndrome has occurred, often without signs of encephalomalacia. The cause of this problem is unknown, but it is probably related to the causes of encephalomalacia. Both conditions are expected to be continuing problems in 1980.

LA ENCEFALOMALACIA - UN PROBLEMA EN EL CAMPO QUE APARECE DE NUEVO

Se atribuye principalmente el papel de la vitamina E a su actividad como un tisú antioxidante. Las enfermedades por carencia de la vitamina E en los pollos son la encefalomalacia, la tarde mortalidad embriónica, el hemolisis eritrocito, y el steatitis (todas son atribuídas a la presencia del graso poliunsaturado en la dieta, el diatesis exudado y la distrofía muscular. Estas últimas dos enfermedades también tienen correlación con las carencias del selenium. La mayoría de la forma activa de la vitamina E que ocurre naturalmente, alfa tocoferol, se encuentra en el maíz en la dieta de las parrillas. Los alimentos de las parrillas son suplidos por la adición de dl alfa tocoferol acetato para conformar a o exceder el requisito del Concilio Nacional de Investigación de 10IU/kg en la dieta. A causa de la instabilidad de alfa tocoferol que ocurre naturalmente, es común fortificar los alimentos de las parrillas con la vitamina E adicional.

La encefalomalacia empezó a hacerse un problema más grave que nunca en la primera parte del verano en 1979 en las parrillas criadas en el sudeste y el medio sur de los E.U. La encefalomalacia responde generalmente a la combinación de la vitamina E y al selenium que son administrados en el agua de los pollos. Desemejante de los años anteriores, los casos de encefalomalacia seguían a ocurrir por la última parte del otoño y la primera parte del invierno de tiempo moderado de 78-79. Cuando se coleccionaron y se analizaron los ejemplos de los alimentos de casetas afectadas las concentraciones de la vitamina E exceden bastante el requisito.

La encefalomalacia es una enfermedad de los pollitos que se siente que sea el resultado de los ácidos grasos y poliunsaturados, como el ácido linoleíco que está presente en los grasos inestables que son añadidos a la dieta. Esto puede ser impedido experimentalmente con la vitamina E, etoxiquin, (un antioxidante) y por supuesto por corregir el problema de la cualidad del contenido del alimento. Las investigaciones que enfocan en el control de la cualidad de los orígenes de los grasos han tenido éxito mezclado y pocos de los reproductores de los pollos engordos han aumentado la cantidad de la vitamina E que ellos añaden al alimento de los pollos. El síndrome del pájaro que se hace pálido ha ocurrido muchas veces sin las indicaciones de la encefalomalacia. La causa es desconocida pero es probable que relacione a las causas de la encefalomalacia. Las dos condiciones deben ser problemas que persigan en 1980.

Encephalomalacia--A Re-emerging Field Problem

R. E. Messersmith, D.V.M., Manager, Technical Services for Animal Health and
C. R. Adams, Ph.D., Manager, Technical Services for Animal Nutrition
Roche Chemical Division, Hoffmann La Roche Inc., Nutley, N. J. 07110

The major broiler raising areas of the United States are the Delmarva peninsula, the Southeast and mid-South. In recent years, producers have been reporting an increasing incidence of a "pale bird" syndrome in these broiler producing areas. In early summer, 1979, severe cases of encephalomalacia ("crazy chick") were being reported in increasing numbers from the Southeast and mid-South broiler areas. The most frequent and most severe cases were reported from the northern Alabama/ Georgia area.

General Observations on Encephalomalacia in Broiler Farms (1979) are (1) The total alpha tocopherol (vitamin E) content of feed by chemical assay is usually adequate (ranging from 30 to 68 mg/kg feed which is 3 to 6.8 times the NAS-NRC requirement[3]); (2) Many plasma tocopherol assay values are very low ($<$0.3 mg/100 ml) and in some plasma samples no tocopherol was detected; (3) Liver vitamin E values from affected birds were low; (4) Rickets or vitamin A deficiency was not evident; (5) Males and females were equally affected; (6) The problem first appeared in early summer and usually disappeared or diminished during the winter months. However, during the winter of 1979-80 the problem persisted; (7) The appearance of encephalomalacia may be preceded by an oily or white, pasty diarrhea by 1-3 days. At this time, pink discoloration of shanks may occur; (8) It may hit consecutive flocks, skip a flock, or not recur; (9) It does not occur in every house on a farm; (10) It is not usually seen in breeder flocks.

Other diseases which may be similar to encephalomalacia which are characterized by symptoms resulting in damage to the nervous system must be differentiated from encephalomalacia, i.e.: avian encephalomyelitis, Newcastle disease, Mareks disease, riboflavin deficiency, and "malabsorption syndrome."

The diagnosis of encephalomalacia can be confirmed by: (1) Age. Birds at 3-5 weeks of age are most often affected with encephalomalacia although it has been observed as early as 2-1/2 weeks of age; (2) A characteristic oily or white pasty diarrhea may precede appearance of "crazy chick" disease by 1-3 days. At this time, there is usually a pink discoloration of the shanks; (3) There are typical signs of "crazy chick" disease; (4) Gross lesions of the cerebellum are generally observed with swol-

len, edematous or necrotic areas and convolutions may be flattened. Small hemorrhagic areas also may be seen; (5) The absence of rickets or vitamin A deficiency lesions differentiate this problem from "malabsorption syndrome"; (6) Final confirmation of encephalomalacia can be made by the histopathology of the brain. In some cases, lesions of the pancreas may be seen.

To treat flocks affected with encephalomalacia, the following additions to the drinking water should be made: 640 I.U. vitamin E/gallon (170 ppm)

The vitamin E should be provided as a water dispersible, (emulsified) dry formulation or a dispersible (emulsifiable) liquid concentrate for optimum absorption. (The emulsified droplets of vitamin E should be in the sub micron range to allow for optimum absorption. Hence, the dispersible liquid concentrate is the product of choice).

To prevent further occurrence of field encephalomalacia, the role of vitamin E in the nutrition of the growing chicken should be understood. Vitamin E has been recognized as an essential dietary nutrient for chickens for many years. However, its fundamental biological role is uncertain. One theory suggests that vitamin E functions as an antioxidant in the protection of tissue lipids against peroxidation. Another theory is that it has a metabolic role(s) in addition to its antioxidant properties. The antioxidant theory has been supported by two types of experiments. First, it has been shown that unsaturated fat and/or specific polyunsaturated fatty acids (PUFA), particularly linoleic acid, exacerbate vitamin E deficiency conditions. Secondly, it has been shown that chemical antioxidants such as ethoxyquin can spare vitamin E activity in chickens. Selenium has been shown to spare vitamin E in preventing certain conditions such as exudative diathesis in poultry, but not encephalomalacia. Vitamin E is associated with the lipid phase of cell walls and constituents and protect the cell by preventing the initial phases of fatty acid oxidation. Evidence suggests that vitamin E and selenium are cofactors as cellular antioxidants but they do not replace each other. The selenium content of feed ingredients is variable. Broiler feeds are almost universally supplemented with 0.1 ppm Se. The natural tocopherols (vitamin E) found in feed ingredients are highly variable in content and bioavailability and are quite unstable. In chickens, dietary levels of vitamin E above the requirement for growth have been shown to increase shelf life of chicken meat (160 IU/kg for 5 days)[1] and to improve the response to experimental E. coli infection (300 IU/kg).[2] Vitamin E deficiency diseases and the interaction of other nutrients is illustrated by the following table:

	Prevented by		
Disease	Vitamin E	Selenium	Ethoxyquin
Encephalomalacia	Yes[a]	--	Yes[c]
Late Embryonic Degeneration	Yes[a]	--	Yes
Male sterility	Yes	--	--
Erythrocyte hemolysis	Yes[a]	--	Yes[c]
Exudative diathesis	Yes	Yes	--
Steatitis (yellow fat disease)	Yes[a]	--	Yes[c]
Muscular dystrophy	Yes	(P)[b]	--

a=Severity partially dependent on presence of polyunsaturated fatty acids
b=Incidence and severity partially related to selenium
c=Partially effective at approved 150 ppm level in feed

<u>Characteristics of Vitamin E</u> - Vitamin E is represented by a group of organic alcohols known as tocopherols. At least seven are known to occur as natural constituents of feedstuffs: One has very little vitamin E activity, five have moderate activity and one, d-alpha tocopherol is the most biologically active form of vitamin E. The vitamin E product which is most commonly added to feed is dl alpha tocopheryl acetate which contains 1 I.U. of vitamin E activity per milligram. The natural tocopherols are not very stable in feed. Chemically synthesized vitamin E, dl-alpha tocopheryl acetate, is very stable in feed. For proper utilization it must be hydrolyzed to the free alcohol--this is postulated to occur within the lumen of the intestine, in the intestinal wall, or (to a lesser extent) in the chicks body tissues.

<u>Vitamin E Content of Feeds</u> - Naturally occurring alpha-tocopherol content varies widely among feed ingredients and also among different samples of one feed ingredient. Its average concentration ranges from 108 mg/kg (alfalfa meal), 29 mg/kg (corn) to 4.4 mg/kg (soybean meal) among major feed

ingredients. From a practical standpoint, corn contributes the largest portion (>50%) of the naturally occurring vitamin E activity to broiler feeds in the United States because of the large amount of corn in such feeds. The NRC reouirement for vitamin E of broilers is 10 mg/kg[3] of feed. Nutritionists in various areas of the United States add from 1.1 to 27.5 mg/kg supplemental vitamin E to broiler feeds to supplement the naturally occurring alpha tocopherol.[4] Obviously, there are considerable differences in recommendations for vitamin E fortification by various nutritionists. The level of supplementation is determined by field experience:

		Vitamin E Concentration in Broiler Feed (I. U. /kg)
NRC requirement (starting chickens)		10.0
Roche recommended added fortification		6.6
U. S. added fortification:[a]	Southeast	1.1 - 27.5
	Midsouth-Southwest	2.2 - 16.6
	Mid Atlantic	4.4 - 11

a=from Roche survey of broiler industry

It is common practice to fortify broiler feeds with vitamin levels above the NRC requirements. This is not a whimsical practice, but is based on industry experience and the need to minimize the effect of many factors which may influence the vitamin needs of broilers.

Encephalomalacia has been experimentally produced in chicks by: (1) Feeding purified ration containing lard; (2) Feeding low vitamin E rations with safflower oil; (3) Feeding ration containing stripped corn oil, cottonseed oil, or soybean oil. In these cases, the presence of the PUFA, linoleic acid, contributed to a high incidence of encephalomalacia. Supplemental vitamin was effective in preventing encephalomalacia induced by the addition of 5% safflower oil to the diet as follows:

	Vitamin E (I. U. /kg feed)		
Ration	Added[a]	Total	% Incidence of encephalomalacia[b]
Basal (low vit E)	0	4.2	0
Basal + 5% safflower oil	0	4.2	90
"	0.5	4.7	70
"	1.0	5.2	50
"	2.0	6.2	30
"	4.0	8.2	10
"	8.0	12.2	0

a=dl alpha tocopheryl acetate
b=incidence of encephalomalacia over 5 week test period

The antioxidant, ethoxyquin, was also effective in preventing encephalomalacia,[6] but at high levels. However, selenium and cystine were not effective for this purpose. There is some suggestion that sulfur-bearing amino acids may be involved in development of muscular dystrophy. In a field outbreak of encephalomalacia in broilers at one location samples of plasma were collected from encephalomalacia affected and normal (unaffected) birds on the same farm. Plasma alpha tocopherol concentrations were:

	Unaffected	Affected with encephalomalacia
Farm 1	0.75 mg/100 ml	No measurable amount
Farm 2	0.80 mg/100 ml	0.10 mg/100 ml
Farm 3	0.68 mg/100 ml	0.27 mg/100 ml

Serum alpha tocopherol concentration range from 0.3 - 0.70 mg/100 cc in normal, healthy broilers. Treatment of the birds in all houses with a selenium-vitamin E combination (feed or drinking water?) was initiated, and plasma alpha tocopherol concentrations were considerably higher 24 hours later in samples from all but one house.

The factors believed to be responsible in the pathogenesis of encephalomalacia are: (1) The vitamin E consumed is not being absorbed efficiently; (2) Alpha tocopherol in the intestine is being destroyed by peroxides present in the dietary fat or from other ingredients. This alpha tocopherol would include natural (from diet) or that provided by hydrolysis of the acetate ester; (3) High intake of unsaturated fat may increase the tissue requirement; (4) Possibility of intestinal disease condition interfering with conversion of alpha tocopherol acetate to free tocopherol and/or the absorption of vitamin E.

Another condition which may be related to the vitamin E-encephalomalacia problem is the "pale-bird syndrome". This condition involves broilers which appear perfectly normal, except for a total lack of deposition of pigment (xanthophylls) in the skin, shanks and beak. No consistent pathology can be found, but pale birds often recur on the same farms. It is possible that oxidation of xanthophylls in the feed exacerbates the pale bird problem. The same conditions that result in pale birds may be involved in pathogenesis of encephalomalacia (i.e., destruction of fat-soluble ingredients).

To prevent encephalomalacia epidemics, a multidisciplinary approach should be persued: (1) Ignore the problem--it will go away next winter, anyway. Note: it would be prudent to let someone else go on record with this recommendation--what were isolated flock problems last year may turn out to be more numerous this year; (2) Thoroughly investigate all cases of encephalomalacia to determine if there is some underlying enteric disease process hindering utilization of vitamin E; (3) Analyze samples of starter feed actually fed to the bird during the period prior to development of encephalomalacia for vitamin E. Collect pooled plasma samples of affected and nonaffected birds in the house and analyze for vitamin E. Correlate these assays with similar assays from normal houses. Assay samples of vitamin E source used to fortify the feeds. Since the premix and feed may have been used up at the time the problem is investigated, a quality control procedure may have to be designed and implemented. Use of premix manufactured by a quality-conscious manufacturer is helpful; (4) Evaluate feed mixing procedures to determine if the vitamin E and selenium are actually being added and thoroughly mixed; (5) Check feed ingredients likely to contribute rancid or oxidized fat if improperly manufactured or stored: fishmeal, fat, meat meal or poultry by-products, etc. These ingredients should be of suitable quality and should be properly stabilized with an effective antioxidant such as ethoxyquin; (6) If finished feed is not fed quickly (for example, bagged feed), it may be necessary to add ethoxyquin to it; (7) Deliver smaller quantities of starter feed during warm months to insure freshness; (8) Add a water dispersible form of vitamin E to the drinking water. This will help assure a build-up at the cellular level prior to the time of disease incidence; (9) Avoiding the feeding of damaged or moldy grain; (10) If all else fails, it may be necessary to increase the amount of supplemental vitamin E in the feed.

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WET LITTER SYNDROME IN BROILERS AND HEAVY BREEDERS IN ARGENTINA

Mateo H. Delamer
San Sebastián S. A.
Belgrado 347
Buenos Aires

This syndrome consists of a change in the normal condition of the flock's litters as a consequence of watery dejections.

Signs

Birds show an abnormal thirst, consuming even up to three times the quantity of water normal for their age. The feathers below the cloaca are sticky and dejections are often noisy.

Lesions

The only lesion macroscopically observed,has been a hypertrophy of duodenal and intestinal epitheliums, with very protruding vill

On occasions, an intensification of leg problems and epiphisiolisis of the phemur head hasbeen observed.

Type of birds affected and age

As from three years ago it has very frequently been observed in broilers. Up until then, it had been observed in broiler breeders in the rearing and/or production periods. It has not been oberved in commercial layers on floor. It has been observed in birds as from the age of one week - in broilers it is more evident after the seventh week.

Diffusion

In mos of the country's operations, either continually or at intervals. The syndrome is more intense during the winter months. There in no information of a similar problem in other countries.

Economic importance

This problem is not of a dramatic nature as in general it is not the direct cause of death, but it can reduce growth in up to 10% and deteriorate feed conversion in a similar figure.

Ethiology

Still very unclear. Bacteriological studies of the intestinal content of the affected birds show a normal flora. A large quantity of medical treatments have been carried out with no result. Many tests have been made by excluding different feed components, with no positive result. Circumstantial evidence would seem to indicate the participation of a virus of vertical an horizontal transmission in interaction with nutritional and climatic factors.

SINDROME DE CAMAS HUMEDAS EN POLLOS PARRILLEROS Y REPRODUCTORES PESADOS EN ARGENTINA.

Consiste en una alteración del estado normal de las camas de las aves como consecuencia de deyecciones acuosas.

Signos

Las aves muestran gran avidez de agua, llegando a consumir hasta tres veces la cantidad normal para su edad. Las plumas debajo de la cloaca se encuentran pegoteadas y en muchas ocasiones las deyecciones se efectúan con un fuerte chasquido.

Lesiones

La única lesión observada es una hipertrofia de los epitelios duodenal e intestinal, con sus vellosidades muy sobresalientes. En ocasiones se intensifican los problemas locomotrices y la epifisiolisis de la cabeza del fémur.

Tipo de aves afectadas y edad

Desde hace tres años se lo observa con elevada frecuencia en pollos parrilleros.
Anteriormente, se lo había observado en aves reproductoras pesadas en crianza y/o producción. No ha sido observado en ponedoras comerciales a piso.
Se lo ha observado en aves de una semana de vida en adelante. En pollos parrileros es más evidente luego de la 7o semana.

Difusión

En casi todas las explotaciones del país en forma contínua o intermitente.
El trastorno se intesifica durante el invierno.
No se tiene información de un cuadro similar en otros países.

Importancia económica

En general este trastorno no acarrea mortandad en forma directa, lo que le quita dramatismo, pero puede reducir el crecimiento hasta en un 10% y desmejorar la conversión alimenticia en el mismo valor.
quita dramatismo, pero puede reducir el crecimiento hasta en un 10% y desmejorar la conversión alimenticia en el mismo valor.

Etiología

Aún muy oscura. Los estudios bacteriológicos del contenido intestinal de las aves afectadas muestran una flora normal. Se han efectuado gran cantidad de tratamientos medicamentosos sin ningún resultado. Se han realizado numerosos ensayos de exclusión de componentes de la ración, también sin resultados positivos.
Evidencias circunstanciales indicarían la participación de un virus de transmisión vertical y horizontal en interacción con factores nutricionales y climáticos.

Traducción: Cortesía del Dr. Carlos Lóez Coello.

SOME OBSERVATIONS AND COMMENTS ON ROTAVIRUSES IN TURKEY POULTS

Nigel E. Horrox, B.V.M.S., M.R.C.V.S.
Garth Veterinary Group "Willowgarth",
Beeford, Nr. Driffield, N. Humberside YO25 8AY, England

SUMMARY

In recent years rotaviruses have been implicated as a cause of scour in the young of mammalian species.

During the winter of 1978-79 a very mild scour was observed in flocks of young turkey poults. The less severe outbreaks were almost clinically nonapparent. Noticeable mortality only occurred if a secondary condition, e.g. vent pecking, ensued. Abnormalities were centered on the lower digestive tract in which the contents were abnormally fluid.

Virus could only be detected by electronmicroscopy between days two and six. In poults from a hatchery showing a similar postmortem picture, rotavirus could not be demonstrated. The majority of affected flocks were derived from breeder flocks that were early into lay.

RESUME

UNAS OBSERVACIONES Y UNOS COMMENTARIOS SOBRE EL ROTAVIRUS EN PAVITOS

En años recientes el rotavirus ha sido implicado como la causa del vientre flojo en los jóvenes de la especie mamífera.

Durante el invierno de 78-79 se observó el vientre flojo muy moderado en las parvadas de pavitos. Los casos menos graves fueron apenas evidente clinicalmente. La mortalidad notable sólo ocurrió si una condición secudaria siguió, p.j., el picoteo de la cloaca. Las abnormalidades se enfocaron en el bajo sistema digestivo en el cual el contenido fue abnormalmente fluído.

El virus sólo podía ser notado por electromicroscopía entre 2-6 días. En los pavitos de una incubadora que enseñaron un esquema semejante posterior a la muerte, el rotavirus no podía ser demonstrado. La mayoría de las parvadas afectadas venían de parvadas de reproductores que empezaron a empollar temprano.

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INTRODUCTION

In recent years rotavirus has been implicated as an aetiological agent of scour in many mammalian species (4). Such rotavirus related conditions have mainly been observed in the young of the species. At the present time rotavirus related scour in piglets appears to be increasing in importance in Britain.

In 1977 an enteritis in turkey poults that was probably caused by a rotavirus was reported in the U.S.A. (1).

In 1978 an outbreak of profuse scouring in 2-5-233k-old turkeys in Britain was reported (3). Rotaviruses were observed in the intestinal contents of these birds.

In 1979 an outbreak of diarrhoea in commercial layers that were in lay was reported (2). Rotavirus-like particles were observed in faecal samples from these birds. In addition to this serological investigations have shown rotavirus infection to be common in Northern Ireland (3).

CLINICAL PICTURE

During the winter of 1978-79 a very mild scour was observed in several flocks of young turkey poults. All of these flocks were intensively reared in controlled environment houses. Three different breeds of turkey were involved.

Clinically the situation on the ground varied from being non-apparent to a mild scour. The less severe outbreaks were only observed by the most observant of stockmen. Affected birds showed slight huddling and a slight loss of activity. The only visual finding indicative of scour was a dampening of the vent.

Mortality did not always occur, but when it did, the majority of the losses were as a result of secondary lesions such as those ensuing from vent pecking.

POST MORTEM FINDINGS

Poults showing the above clinical picture were submitted for post mortem examination.

The quality of these poults was variable and the only visible abnormalities were centered on the lower digestive tract. The common finding was one of abnormally fluid intestinal and caecal contents. In many instances the caecal contents were also frothy and/or contained large gas bubbles. There were no visible abnormalities in the intestinal or caecal walls.

Histopathological examinations failed to reveal any lesions.

Bacteriological studies revealed normal mixed floras. No salmonella was isolated. No parasites were observed in wet smear preparations.

When dead poults were examined, the above picture was observed but the cause of death could be attributed to other lesions e.g. severe anaemia as a sequel of vent pecking.

ELECTRONMICROSCOPY

Samples of caecal and intestinal contents were examined under the electronmicroscope.

Table 1

Age (Days)	Clinical Picture	Post Mortem	Virology
2	Poor poult quality Wet vents	Enteritis/Caecal Enteritis	ROTAVIRUS 3/4
3	Poor poult quality Wet vents	Enteritis/Caecal Enteritis	ROTAVIRUS 5/5
9	Huddling	Caecal Enteritis	--
6	Huddling Wet vents	Caecal Enteritis	ROTAVIRUS 2/3
6	Poor poult quality Wet vents	Caecal Enteritis	ROTAVIRUS and ENTEROVIRUS 1/1
1	Mild huddling	Mild Caecel Enteritis	--
28	Huddling. Scour	Enteritis/Caecal Enteritis	--

Bergeland reported a similar syndrome but in two-week-old poults (12-21 days) showing a mortality of 3.3-7.5%. The mortalities seen in this survey were less than this--the highest total (from all causes) 14-day mortality figures being 4.45%.

This difference is apparent when 21-day mortality percentages are compared.

Table 2

21-day Mortality Figures (%)	
Bergeland	Table 1 flocks
11.20	2.38
7.98	4.69
	3.20

Although the syndrome seen in Humberside was basically in poults less than one week old a similar syndrome was occasionally seen in older poults but the presence of rotavirus could not be demonstrated.

Examination of poults at a hatchery in which there was mild scouring in the hatchers revealed a similar post mortem picture but electronmicroscopy failed to demonstrate the presence of rotavirus.

Also noted in this study was that the majority of affected flocks in which rotavirus could be demonstrated were derived from breeder flocks that were early into lay.

COMMENTS

The syndrome that was observed was a very mild one and did not appear to cause mortality in its own right. In the examinations undertaken, rotavirus could only be demonstrated between two and six days of age.

If rotavirus infection occurred at the point of hatching it is probable that there would be an insufficient number of virions in the digestive tract contents for detection by electronmicroscopy until the second day of infection.

The findings to date are suggestive of poults being infected in the first few hours after hatching and/or rotavirus being transmitted on or in the egg and thereby infecting the poult.

Lowered poult quality and/or stress appear to be involved in the syndrome being manifested slightly more severely.

To date the syndrome on its own has not been of much economic significance but this is not the case should vent pecking ensure.

The work done to date is still in its infancy, but the indications are that the scour syndrome described is related to rotavirus infection.

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UPDATE ON LEG WEAKNESS IN BROILER CHICKENS

C. Riddell
Western College of Veterinary Medicine
Saskatoon, Saskatchewan
Canada S7N OWO

Leg weakness in broiler chickens may be divided into two major categories, infectious and non-infectious disorders. Avian encephalomyelitis, viral arthritis, infectious synovitis and staphylococcosis are well recognized infectious disorders causing leg weakness as a prominent part of the clinical disease they produce in broiler chickens. Other infectious agents will also produce leg weakness in broiler chickens. The list of agents possibly capable of causing leg weakness in broiler chickens has recently been expanded by the isolation of adenoviruses from tendinitis, the contamination of Marek's disease with reticuloendotheliosis virus and the association of a yet to be identified infectious agent with osteodystrophy in Holland. It is difficult to establish how widespread and important infectious diseases are in leg weakness but they do produce regional problems. Non-infectious disorders causing leg weakness are more widespread in the broiler industry and may be of greater overall importance. Common non-infectious disorders are spondylolisthesis, tibial dyschrondoplasia and long bone distortion. Spondylolisthesis has been of minor economic importance. Tibial dyschondroplasia has been of greater economic significance primarily due to downgrading of product due to the enlarged and misshapen tibial head. Research suggests that the defect is the result of an interaction between genotype and diet. Long bone distortion is the commonest disorder and causes the greatest loss

due to the resulting cull birds and poor quality finished product. It includes several types of deformity variously described as perosis, twisted leg, chondrodystrophy and rotated tibia. The difference between these deformities are poorly defined and understood. Research indicates that genotype, nutrition, and environment can influence the incidence of long bone distortion. The relative importance of these different etiological factors in the field has not been well defined.

ACTUALIZACION SOBRE LAS CLAUDICACIONES EN POLLO DE ENGORDA.

Las causas de claudicación en el pollo de engorda pueden ser divididas en dos grandes categorías, problemas infecciosos y no ifecciosos. La encefalomielitis aviaria, la artritis viral, la sinovitis infecciosa y la estafilococosis son problemas infecciosos bien conocidos, en los que la claudicación forma una parte importante de su presentación clínica en pollo de engorda. Otros agentes infecciosos tambien producen claudicación. La lista de agentes capaces de causar claudicación en pollo de engorda ha sido recientemente ampliada con el aislamiento de adenovirus en casos de tendinitis, la contaminación de vacunas contra la enfermedad de Marek con virus de la reticuloendoteliosis y la asociación de un virus aún no identificado con casos de oteodistrofia en Holanda. Es dificil determinar cuan extendidas estan y que importancia tienen las enfermedades infecciosas en la claudicación, pero si se sabe que pueden producir problemas regionales. Los problemas no infecciosos como causa de claudicación están mas extendidos en la industria del pollo de engorda y pudieran tener mayor importancia global. Los problemas no infecciosos mas comunes son la espondilolistesis, discondroplasia tibial y deformación de los huesos largos. La espondiololistesis tiene una menor importancia económica. La discondroplasia tibial tiene mayor importancia -- principalmente debido al depreciación de la canal por el aumento de tamaño y deformación de la cabexa de la tibia. Las investigaciones indican que este defecto es causado por la interacción entre el genotipo y la dieta. La deformación de los huesos largos es el problema mas común y causa las mayores pérdidas por el número de animales que se desechan y la mala calidad de la canal. En ella se incluyen varias deformaciones descritas como perosis, piera torcida, condrodistrofia y rotación de la tibia. La diferencia entre estas deformaciones no esta bien definida y está mal entendida. Las investigaciones indican que el genotipo, la nutrición y el medio ambiente pueden influir sobre la incidencia de la deformación de los huesos largos. La importancia de estos factores etiológicos no se ha determinado en el campo.

Traducción: Cortesía del Dr. Benjamín Lucio Martinez.

UPDATE ON LEG WEAKNESS IN BROILER CHICKENS

C. Riddell
Department of Veterinary Pathology
Western College of Veterinary Medicine
University of Saskatchewan
Saskatoon, Saskatchewan Canada S7N OWO

Leg weakness in broiler chickens may be divided into two major categories, infectious and non-infectious disorders. Avian encephalomyelitis, Marek's disease, viral arthritis, infectious synovitis and staphylococcosis are well recognized infectious disorders causing leg weakness as a prominent part of the clinical disease they produce in broiler chickens. It is difficult to establish how widespread and important these infectious diseases are in leg weakness but they do produce regional problems. Avian encephalomyelitis, Marek's disease and infectious synovitis can cause significant losses, but the first two are commonly controlled by vaccination while the last is controlled by eradication. The importance and control of viral arthritis by vaccination is complicated by the possibility that tendinitis in broilers may also be caused by adenoviruses (6,10) and by the possibility that rupture of the gastrocnemius tendon may not always be a sequel to viral

arthritis. In Western Canada I recognize little clinical tendinitis in commercial broilers though a small number of birds with ruptured tendons may be found when birds are processed. Staphylococcosis in my experience is not commonly a major cause of lameness in broiler chickens but will cause losses near marketing time if birds are overcrowded and/or if litter conditions are poor. Field observations of Staphylococcosis in turkeys, broiler breeders and leghorn chickens suggest it is often associated with stress or management errors. An important question is whether or not viral arthritis predisposes to Staphylococcosis. Tendon sheath lesions due to viral arthritis could aid in localization of bacterial infection (9). The list of agents possibly capable of causing significant leg weakness in broiler chickens has recently been expanded by the contamination of Marek's disease with reticuloendotheliosis virus (5) and the association of a yet to be identified infectious agent with osteodystrophy in Holland (7).

In my opinion, non-infectious disorders causing leg weakness appear to be more widespread in the broiler industry and may perhaps be of greater overall importance. Common non-infectious disorders are spondylolisthesis, tibial dyschrondroplasia and long bone distortion (17). Rickets is also still encountered as a cause of leg weakness in some broiler flocks. Degeneration of the femoral head has been reported as a significant problem in some areas.

Spondylolisthesis, though widespread, has been of minor economic importance. It is caused by deformation of the spine pinching the spinal chord and producing paralysis. Tibial dyschondroplasia has been of greater economic significance than spondylolisthesis primarily due to downgrading of product due to the enlarged and misshapen tibial head. This deformity is due to abnormal development of cartilage in the proximal tibia. The abnormal development occurs in a minor form in many broilers with no clinical significance. In some birds, however, the abnormal cartilage is very massive, birds have bowed legs and are reluctant to move. The incidence of this defect can be increased rapidly by genetic selection and the incidence can be decreased by slowing down the growth rate of susceptible birds. It has also been shown that the incidence and severity of the defect can be increased by feeding rations containing a high level of chloride (8). Recently, Poulos (14) has suggested that tibial dyschondroplasia is part of a generalized skeletal disorder similar to osteochondrosis in mammals and is related to failure of cell differentiation in growing cartilage. French workers have indicated that acidosis due to high chloride may impair renal transformation of 25 Hydroxycholecalciferol into 1,25 Dihydroxycholecalciferol (18). Though experimental work has differentiated rickets from tibial dyschondroplasia it is possible that mild rickets may make tibial dyschondroplasia more severe. Often in field cases of tibial dyschondroplasia bone strength is poor. Veltmann and Jensen (19) have recently suggested that factors in litter may predispose chicks to tibial dyschondroplasia. It has also been suggested that certain factors may interfere with vitamin D utilization. Recent studies in Germany suggested that toxins produced by _Fusarium moniliforme Sheldon_ might influence the availability of vitamin D in broiler chickens (3). Degeneration of the femoral head has only been briefly reported from America (13), Australia (1) and Holland (11) and in the first two countries has been associated with scabby hip syndrome. Studies in Australia (12) have demonstrated that both scabby hip and femoral bone syndrome may be prevented by molybdenum supplementation but this has not, to my knowledge, been confirmed elsewhere.

The largest economic loss due to leg weakness in broiler chickens may at present be due to various types of long bone distortion. These deformities in the field are described by many names, including twisted leg, rotated tibia, chondrodystrophy and perosis. Their cause is poorly understood. Experimental studies have shown that similar long bone distortion can result from changes in cartilage of the growth plate (21), poor development of the shaft of long bones (2) or changes in tendons (16). The relative importance of these different mechanisms in field problems is unknown. Nutritional factors have not been implicated in the latter two mechanisms but have been shown to be important in the first mechanism. Several B vitamins and trace minerals including choline, biotin, pyridoxine, nicotinic acid, folic acid, manganese and zinc have been shown to be important in proper development of the growth plate and deficiencies have resulted in chondrodystrophy and long bone distortion or what has commonly been called perosis (15). Further research may be needed to determine whether adequate levels of these B vitamins are being added to present day poultry rations or whether utilization of these vitamins is being prevented. There is no evidence to date to indicate that Mycoplasma infection may interfere with B vitamin utilization as has been demonstrated for the turkey (20). Genetics has been shown to influence the incidence of long bone distortion as has environment (4). The increased incidence of this type of deformity in birds reared in batteries may be attributed to lack of exercise resulting in poor bone development. Current management practices of "end brooding" and limited floor space may be contributing to the problem.

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LEG PROBLEMS IN BROILERS AND THE DEVELOPMENT OF A DAY-OLD CHICK VACCINE AGAINST VIRAL ARTHRITIS/TENOSYNOVITIS

Louis van der Heide
Department of Pathobiology
University of Connecticut
Storrs, Connecticut 06268 U.S.A.

Leg problems in broilers and broiler breeders are discussed and their causative factors, infectious as well as non-infectious, determined.

Genetic and nutritional factors play a role, as well as bacteria (Staph., E. Coli etc.), Mycoplasma (M. synoviae), and viruses (reovirus and possible adenovirus).

Unfortunately, many conditions such as viral arthritis, tibial dyschondroplasia, femoral head necrosis/brittle bone disease and Staph. infections can be found together in the same bird, which is a highly confusing situation. Frequent misdiagnosis are the result and disappointing results of treatment and/or control are found.

Vaccination of broiler breeder flocks against viral arthritis/tenosynovitis has performed well in regard to protecting broiler progeny against viral arthritis, but it does not seem to protect progeny against the development of femoral head necrosis or brittle bone disease.

A day-old chick vaccine has been developed at the University of Connecticut which offers protection against viral arthritis challenge infections. The vaccine is totally apathogenic when given subcutaneously at one day of age in chicks with or without maternal immunity against viral arthritis.

TRASTORNOS LOCOMOTORES EN POLLOS DE ENGORDA Y EL DESARROLLO DE UNA VACUNA CONTRA ARTRITIS/TENOSINOVITIS EN AVES DE 1 DIA DE EDAD.

Los trastornos locomotores en pollos de engorda y reproductoras pesadas - son discutidos, así como los factores infecciosos y no infecciosos que los producen.

Factores genéticos y nutricionales, así como bacterias (Staph., E. coli, - etc.), Micoplasma (M. synoviae) y virus (reovirus y posiblemente adenovi-- rus) representan causas a considerar.

Desafortunadamente varias condiciones como es la artritis viral, condrodis plasia tibial, necrosis de la epífisis del fémur, fragilidad ósea, e infec ción por Staph. pueden ser encontrados simultáneamente en la misma ave oca sionando situaciones confusas, y provocando frecuentemente controversias o diagnósticos erroneos.

La vacunación de las parvadas reproductoras contra la artritis-tenosinovi- tis viral se ha desarrollado para dar protección a la progenie contra la - artritis viral, pero aparentemente no protege a la progenie contra la ne- crosis de la epífisis femoral o la fragilidad ósea.

Una vacuna para pollitos de 1 día de edad ha sido desarrollada en la Uni-- versidad de Connecticut la cual ofrece protección contra la artritis viral. La vacuna es totalmente apatógena cuando es administrada por vía subcuta-- nea al 1er.día de edad en aves con o sin inmunidad materna contra la artri tis viral.

Traducción: Cortesía de la Dra. Luz Ma.Charles de López

AISLAMIENTO DEL VIRUS DE LA ARTRITIS VIRAL EN MEXICO

M.V.Z. Juan Carlos Morales L.
M.V.Z. J. Genaro Huerta V.
M.V.Z. Francisco Reséndiz S.
Mezquital del Oro
Hermosillo, Son. México

RESUMEN

Se aisló el virus de la artritis viral (.A..V.) de un brote de campo en pollonas de reposición de 20 semanas de edad en el area avícola de Hermosillo, Sonora.

Se utilizó la técnica de inoculación en embriones S.P.F. y se logró la reproducción de la enfermedad en pollos de engorda y reproductoras pesadas de 4 y 16 semanas de edad respectivamente.

ISOLATION OF VIRAL ARTHRITIS VIRUS IN MEXICO

ABSTRACT

Isolation of the virus causing an outbreak of Viral Arthritis (V.A.) in replacement pullets 20 weeks of age was accomplished in the Hermosillo, Sonora area.

The SPF embryo inoculation technique was used in reproducing the disease in broiler chicks and heavy breeders pullets of 4 and 20 weeks of age, respectively.

INTRODUCCION:

La artritis viral es una enfermedad de las gallinas causada por un reovirus; ésta se caracteriza por las lesiones que produce a nivel de la articulación tibio-tarsiana, afectando el tendón flexor digital, las vainas tendinosas - y la membrana sinovial, en donde causa inflamación, necrosis y ruptura del tendón; así como las lesiones en el miocardio (2,4,5,6,7,12,14).
Los primeros reportes de brotes fueron descritos por Olson en 1957 en los - Estados Unidos a partir de aves de raza pesada y en Inglaterra por Dalton y Henry; posteriormente se ha aislado el virus en otras partes del mundo (5, 6,15).
La literatura menciona que esta enfermedad afecta principalmente a aves de raza pesada; informes posteriores reportan la presencia de ésta en aves de raza liviana (1).
En México, en el año de 1978 se realizó una encuesta serológica, demostrándose la presencia de anticuerpos contra dicha enfermedad en algunas áreas, sin haber efectuado el aislamiento del virus (5).
En Hermosillo, Sonora, se presentaron los primeros brotes en el año de 1974, afectando a parvadas de raza pesada, con mayor incidencia en los machos, -- confirmándose la enfermedad por métodos serológicos e histopatológicos de - muestras enviadas a los Estados Unidos. Recientemente hemos observado brotes de campo en gallinas Leghorn (13).
Este trabajo se llevó a cabo para intentar el aislamiento del agente causal de la artritis viral a partir de un brote de campo en pollas leghorn.

MATERIAL Y METODOS:

a) Historia de la parvada- Las aves en las cuales se encontró este problema procedían de una parvada de 115,000 pollonas de reemplazo de 20 semanas de edad.
Los signos que mostraban estas aves fueron; resistencia a cualquier movi--- miento, postración, incoordinación, parálisis y posición clásica de apoyo - sobre los tarsos. La morbilidad alcanzaba el 0.5%.
Las lesiones observadas en el campo se limitaban a la región de la articulación tibio-tarsiana, en la cual era notoria la inflamación de ésta, con la característica de ser unilateral en la mayoría de los casos.
A la necropsia se encontraron las siguientes lesiones: inflamación del tendón flexor digital y extensor metatarsal además de exudado mucoso de color amarillento a nivel de la articulación tibio-tarsiana.
b) Obtención del Virus- El exudado que se encontraba a nivel de la articula

Leg problems in broilers and broiler breeders are a major problem in the broiler industry today. In addition to genetic and nutritional causes, infectious agents have been identified, that can cause leg problems. The most widely recognized infectious agents are bacteria (Staphylococcus, E. Coli etc.), Mycoplasma synoviae (M.S.) and viruses (Avian reovirus and possible adenovirus).

Interaction between these various causes has made the situation confusing. In some broilers one can find viral arthritis/tenosynovitis, Staph. infection, tibial dyschondroplasia, twisted legs and femoral head necrosis/brittle bone disease all at the same time. Such situations frequently lead to misdiagnosis and disappointing results of treatment and control measures.

Mycoplasma synoviae eradication has been largely accomplished in broiler breeding stock. If an M.S. break occurs in a broiler breeder flock, the progeny appears to have more air sac problems these days than synovitis. Heat or antibiotic treatment (dipping or inoculation) of hatching eggs appears successful to eliminate egg transmission of M.S.

Staphylococcus infections are a major problem today. Swollen hock joints and tendons frequently yield Beta Hemolytic staph bacteria, many of which are resistant to most antibiotics. A sensitivity test is very helpful in those cases to insure a successful treatment.

Viral arthritis/tenosynovitis as a disease entity has been the subject of vaccination efforts (1,2). Breeder vaccination protected broiler progeny but could not always protect the breeders themselves, when vaccine was given at recommended age of 10-17 weeks. Vaccination at an earlier age (e.g. 3 weeks) has been reported as successful in many cases. However, a safe, apathogenic day-old chick vaccine was also needed and has now been developed at the University of Connecticut. Early tests have shown its safety and protective ability against viral arthritis after day-old application in chicks with or without maternal immunity to viral arthritis. (Table 1).

The appearance of femoral head necrosis and brittle bone disease in broilers and broiler breeders has confused the picture even more. Reoviruses and also adenoviruses have been isolated from chickens with femoral head necrosis. Tests are being performed to see, if the day-old chick vaccine will also protect against the development of femoral head necrosis/brittle bone disease.

Table 1

A.G.P. Reactions and Gross and Microscopic Lesions of Tenosynovitis in SPF Chickens After Day-old Vaccination with $10^{6.8}$ $TCID_{50}$ of P235/P66 of S1133 Strain Reovirus and Footpad Challenge at 14 Days of Age.

	A.G.P.	GROSS LESIONS 9 days p.c.	GROSS LESIONS 21 days p.c.	MICR. LESIONS 21 days p.c.
DAY-OLD	0/19 (N.I. = 0.2)	N.D.	N.D.	N.D.
VACC/NO CHALL.	7/7	N.D.	0/7	0/14
VACC/CHALL.	9/9	1/9(±)	2/9(±)	3/18(±)
NO VACC/CHALL.	10/10	10/10(1+,2+, 3+,4+)	9-10(±)	8/20(1+,2+,3+)
NO VACC/NO CHALL.	0/9	N.D.	0/9	0/18

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ción tibio-tarsiana fue colectado en forma estéril con una jeringa que contenía caldo nutritivo; así mismo tendón e intestino los cuales fueron macerados, centrifugados, adicionándoseles antibióticos (Penicilina-Estreptomicina), almacenándose en congelación con el fin de utilizarlos posteriormente.
Para el aislamiento del virus se utilizaron embriones libres de patógenos - específicos (S.P.F.).
El material sospechoso (exudado sinovial, tendón e intestino) fue descongelado e incubado .5 c.c. en embriones de 3 días de edad, cuando fue utilizada la vía saco vitelino y de 7 días de edad cuando se empleó la vía membrana corioalantoidea(cámara falsa). Dichos embriones fueron incubados a 37°C con una humedad relativa de 70 a 80% y ovoscopeados diariamente.
c) Estudios Bacteriológicos, Serológicos e Histopatológicos- Se realizaron siembras de la articulación tibio-tarsiana en medios de cultivo como McConkey u Staph. 110., con el objeto de determinar contaminación bacteriana.
Otra prueba realizada fue la de aglutinación en placa contra Mycoplasma --- gallisepticum y Mycoplasma synoviae con antígeno específico.
Con los sueros de las aves problemas se efectuaron pruebas de difusión en agar con antígeno de artritis viral.
Por último se tomaron muestras en formol al 10% de tendón y corazón para el estudio histopatológico.
d) Reproducción de la enfermedad - Para confirmar la presencia del virus se utilizaron 5 aves reproductoras pesadas y 10 pollos de engorda de 16 y 4 - semanas de edad respectivamente.
La vía de inoculación en las aves fue directamente por cojinete plantar; depositándose por medio de jeringa 0.5 c.c. de material sospechoso colectado - de embriones inoculados; estas aves se mantuvieron alojadas en baterías en - cuarto de aislamiento.

RESULTADOS:

La mortalidad de los embriones al primer día de inoculados fue desechada por considerarse causada por traumatismo.
La inoculación de los embriones por la vía saco vitelino, dió como resultado la mortalidad total de éstos a los 4 días postinoculación.
Estos embriones se encontraban marcadamente hemorrágicos y presentaban además congestión del hígado, riñón y bazo.
La presencia de placas pustulosas de color gris-amarillento, así como edema en la membrana corioalantoidea fueron encontrados a los 5 días postinoculación.
Todos estos embriones fueron checados bacteriológicamente, realizándose además aglutinaciones con glóbulos rojos al 2% para descartar enfermedad de -- Newcastle, dando resultados negativos a ambas pruebas.
Posteriormente fueron cosechadas muestras de saco vitelino, líquido y membrana corioalantoidea para utilizar en la posterior reproducción de la enfermedad.
Varios de los embriones inoculados por vía membrana corioalantoidea se dejaron incubar 21 días, naciendo algunos y otros solo picaron el cascarón sin poder eclosionar; de los pollos vivos, la mayoría presentaban debilidad y dedos torcidos, teniendo estos una viabilidad de sólo 11 días.
Las siembras realizadas a partir de la articulación tibio-tarsiana dieron -- por resultado el aislamiento de colonias de Staphylococus aurus.
Las pruebas serológicas de aglutinación en placa practicadas a los sueros de estas aves nos mostraron 2 de ellas positivas a Mycoplasma gallisepticum y - una a Mycoplasma synoviae.
Se encontraron bandas de precipitación en la prueba de difusión en agar,determinandose anticuerpos contra artritis viral.

En lo que respecta a la reproducción de la enfermedad, los primeros indicios de ésta al primer día postinoculación en el pollo de engorda se iniciaron -- con la inflamación del cojinete plantar, presentándose posteriormente un marcado edema de las vainas tendinosas.
Las aves mostraban claudicación, incoordinación, apoyo sobre los tarsos y rechazo al movimiento al 2o. día de realizada la inoculación.
Por lo que respecta a las aves reproductoras pesadas, los signos de inflamación en el tendón fueron observados hacia el octavo día en la mayoría de las aves; de igual manera que en el pollo de engorda las aves presentaban postración, marcha rígida y rechazo al movimiento.

Se les practicó la necropsia encontrándose exudado de color amarillento,inflamación del tendón flexor digital y formación de tejido fibroso en la vaina de éste.
Las aves inoculadas experimentalmente fueron sangradas realizándose con el suero de ellas las pruebas de difusión en agar y aglutinación contra Mycoplasma gallisepticum y Mycoplasma synoviae; dando como resultado la presencia de anticuerpos precipitantes contra A.V. y aglutinaciones negativas a Mycoplasma gallisepticum y Mycoplasma synoviae.

DISCUSION:

Los primeros trabajos publicados en el mundo mencionan que este problema se presenta principalmente en raza pesada(6,8,10,11,14); sin embargo, no se había hecho mención en raza liviana (exceptuando un reporte de campo en los E.U. (1) como nosotros lo hemos obervado).
Las lesiones encontradas en los embriones S.P.F. inoculados con las muestras sospechosas nos indican la presencia del virus de la artritis viral.
Los resultados de la inoculación e infección experimental de las aves son similares a aquellas obtenidas por Olson y colaboradores (9).
El estudio histopatológico nos revela que son lesiones similares a las producidas por el virus de la artritis viral (6,9,12).
La ausencia de anticuerpos contra Mycoplasma synoviae en los sueros de las aves experimentales, nos indica que las lesiones a nivel de la articulación tibiotarsiana, no corresponden a sinovitis infecciosa; por el contrario, el hallazgo de anticuerpos contra artritis viral reconfirma el diagnóstico.
Considerando los resultados obtenidos durante el presente trabajo podemos llegar a la conclusión de que el virus aislado corresponde a las características señaladas para el agente causal de la artritis viral.

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LYMPHOID LEUKOSIS INCIDENCE IN LAYING TYPE BIRDS AND A METHOD FOR CONTROL OF THE DISEASE IN THE NETHERLANDS.

H. J. L. Maas

DEPT. OF VETERINARY PATHOLOGY
UNIVERSITY OF UTRECHT
Yalelaan 1 Eastwing Rm 373
3584 CL UTRECHT THE NETHERLANDS

Lymphoid leukosis is wide spread in flocks of chickens throughout the Netherlands and the World. In the first an estimate of its importance in the Netherlands was obtained from an analysis of the mortality results in nine successive Random Sample Tests in Wissenkere, performed between 1968-1979. The average mortality of the flocks during lay (420 days) was 14.4 percent and lymphoid leukosis was responsible for 2.8 percent. The majority of the commercial egg laying flocks participating in these Tests were of American origin, since most stocks used in this country are basically from the U.S.A. If the average lymphoid leukosis mortality rate were applied to the total number of hens used by the Poultry Industry (approximately 25 million) it can be estimated that yearly more than 0.5 million mature birds died of the disease. In the field heavy losses occurred incidentally; between 1970-1976 two rather large breeder organizations were forced to quit business due to very heavy losses from lymphoid leukosis in their flocks and with clients. The leukosis mortality in those flocks ranged between 0.5 - 2.5 percent per month.

In 1970 in the Netherlands, a first attempt was undertaken to develop a method for control of the disease. In 1978 the method has been developed in a program for control of lymphoid leukosis. The program consists of (1) the selection of those dams in a breeder flock which produced leukosis virus-free eggs (embryo examination), (2) only eggs from these mothers were used for progeny, (3) the chicks were reared in isolation for two months, (4) subsequently inoculated with 10^5 median tissue culture infective doses of leukosis viruses of subgroups A and B, and (5) transferred to conventional poultry houses. Under field conditions the program - exclusively for breeder birds - has given very satisfactory results.

Both, incidence of the disease and results of the control will be outlined.

INCIDENCIA DE LA LEUCOSIS AVIARIA EN GALLINAS DE POSTURA Y UN METODO PARA EL CONTROL DE LA ENFERMEDAD EN HOLANDA.

La leucosis linfoide se encuentra muy difundida entre las parvadas de gallinas de Holanda y del mundo.En un principio se stimó su importancia en Holanda mediante un análisis de la mortalidas en nueve pruebas de muestreo al --- azar sucesivas,llevadas a cabo entre 1978 y 1979 en Wissenkere.La mortalidad promedio de las parvadas durante la postura (420 días)fué del 14.4%,del que el 2.8% fue atribuible a la leucosis linfoide.La mayoría de las parvadas comerciales de ponedoras que participaron en las pruebas al azar son de origen americano, ya que la mayoría de los pies de cría usados en este país provienen de los E.U.A.Si el promedio de mortalidad por leucosis linfoide se traspolara al número total de gallinas usadas por la industria avícola (aproximadamente 25 millones) se puede pensar que más de 0.5 millones de aves adultas mueren por leucosis.En el campo hay, en ocasiones mortalidad elevada entre - 1970 a 1976 dos grandes organizaciones de reproductoras quebraron debido a - que tuvieron pérdidas serias por leucosis linfoide en sus propias parvadas y en las parvadas de sus clientes.La mortalidad debida a leucosis linfoide fue de 0.5-2.5% al mes.

El primer intento de desarrollo de un método de control de la enfermedad en - Holanda se realizó en 1970.En 1978 el método evolucionó a un programa para el control de la leucosis linfoide.Este programa consiste en: (1) la selección de madres que produzcan embriones libres del virus (examen embrionario),(2) el uso exclusivo de esos huevos para incubación,(3) crianza en aislamiento - durante dos meses de los pollitos obtenidos (4) inoculación posterior con -- 10^5 dosis infectantes para cultivos celulares de los virus de la leucosis, subgrupos A y B y (5) traslado a gallineros convencionales. Bajo condiciones de campo, el programa usado exclusivamente para reproductoras ha dado resultados satisfactorios.

Tanto la incidencia de la enfermedad como los resultados del control se presentarán.

Traducción: cortesía del Dr. Benjamín Lucio Martínez

LYMPHOID LEUKOSIS IN LAYING-TYPE BIRDS AND A
PROGRAM FOR CONTROL OF THE DISEASE IN THE NETHERLANDS

H. J. L. Maas

DEPT. OF VETERINARY PATHOLOGY
UNIVERSITY OF UTRECHT
Yalelaan 1 Eastwing Rm 373
3584 CL UTRECHT THE NETHERLANDS

Lymphoid leukosis (LL) is wide spread in chickens throughout the world. In the Netherlands an estimate of its importance was obtained from an analysis of the mortality in nine successive random sample tests performed between 1968 and 1979 in Wissenkerke. The average overall mortality in the flocks during lay (420 days) was 14.5% and LL was responsible for 2.8%.

If the average LL mortality rate were applied to the total number of hens used by the Dutch Poultry Industry (approximately 25 to 30 million), it can be estimated that yearly more than 0.5 million mature birds died from the disease.

The majority of the stocks in these tests participating with their so-called "end-product" was of American origin. In the field heavy losses occurred incidently, however, between 1970 and 1976, two rather large size breeder organizations were forced to quit the poultry business due to very heavy losses from LL in their flocks. LL in these flocks ranged from 0.5 to 2.5% per month.

It is not an overstatement to say that there are no poultry breeders, in the United States or elsewhere, who have not suffered heavy financial losses within a certain period due to LL in their flocks.

In the 1960's an observation of great importance was made, i.e., clinical leukosis is almost exclusively seen in birds derived from leukosis virus (LV) infected dams (1,2). This implies that a chicken born free of LV only very rarely developes the disease, notwithstanding the fact that it may become infected by natural exposure. Another very intriguing observation was that chicks born free of LV develop an age-dependent resistance to LL. This resistance becomes almost fully effective between 4 and 8 weeks of age (3). The development of this property is shown in two experiments (Table 1) in which different age groups of LV-free chickens were inoculated simulataneously with rather high doses of LV (4.5). In the older age groups LL mortality is evidently lower than in the groups of chickens exposed at younger age; it should be noted that LL mortality is expressed as

$$\frac{\text{the number of birds with LL}}{\text{the number of birds with LL + the number of survivors}} \times 100$$

TABLE 1. Percent lymphoid leukosis and congenital transmission of leukosis virus in groups pf chickens inoculated at various ages.

		Percent Lymphoid leukosis[1]		Congenital LV transmission[2,3]	
Group	Age at injection	Experiment 1	Experiment 2	Experiment 1	Experiment 2
1	1 day	54.3	62.5	11/13[4]	13/19
2	2 weeks	40.0	37.9	3/9	1/19
3	4 weeks	9.5	3.8	10/20	0/25
4	6 weeks	6.9	8.0	2/21	0/26
5	8 weeks	7.4	4.3	0/16	0/25
6	10 weeks		0.0	0/44	0/26
7	Control	0.0	0.0	0/48	0/54

[1]Percent LL = $\frac{\text{number of birds with LL}}{\text{number of survivors + number of birds with LL}} \times 100$

[2]In Experiment 1, 12 egg collection periods of 2 weeks per hen; in Experiment 2, 5 collection periods per hen.

[3]Virus assay; NP activation test.
[4]Number of positive hens/number of hens tested.
Source: Modified from Maas et al. (1980).

In these experiments is also observed that challenge infection with high doses of LV during lay did not change the incidence of LL in the various age groups. The observations mentioned before indicated that the prevention of clinical leukosis in a flock was feasible, provided (a) LV-free 1-day-old chicks could be hatched, (b) the chicks could be kept free from an infection with LV during the first 8 weeks of live, and (c) a way could be found to prevent the (breeder) birds from virus shedding into their eggs. The first two purposes could be solved by the selection of dams which only produced LV free embryos and by the rearing of their progeny in isolation (the selection was performed by virologic examination of pooled embryo homogenates of each dam; to this end the COFAL test, the NP test, or the PM test are applied). The third purpose, however, i.e., to prevent a bird from shedding LV into her eggs, appeared not so easily to be unraveled. However, in the experiments already discussed the shedding of LV into the embryos of the birds in the various age groups was also studied for obvious reasons. It was then detected that in the age groups exposed to LV after 6 weeks of age the birds did not only show a low LL mortality, but that they did not transmit LV into their eggs also (Table 1). This characteristic did not change after a challenge infection during lay (4,5). To our further surprise, the non-LV shedding embryos were also observed in the eggs from LV free control birds (not vaccinated) after a challenge infection with LV during lay (4,5).

These results show that congenital shedding of LV in LV-free born chickens depends on the age of the first infection. It is clear that for reasons of economy the rearing of commercial chicks (i.e., end product) in isolation units -- even for 2 weeks -- is out of the question. However, omitting this rather short period of isolation in rearing may result in a very early infection with LV of the chicks which occasionally ends in clinical leukosis when adult age is reached. The possibility for this undesirable effect disappeared almost entirely if the "end product" chicks were derived from dams, which were born free of LV, kept in isolation for 0 to 8 weeks of age, and then inoculated with LV. The explanation of this phenomenon is most probably that these "vaccinated" dams passively protect much better the progeny via the transfer of much higher amounts of maternal antibodies (6,7) than the natural contact exposed dams do (Table 2). Consequently end product chicks do not need to be reared in isolation.

TABLE 2. Neutralizing antibody titers in yolks.[1]

Treatment of hens	No. eggs tested	Mean NT[2]	Titer range
Leukosis vaccination at 8 weeks of age	33	10^5	10^3 to 10^6
Naturally exposed to leukosis virus	15	$10^{2.6}$	10^1 to 10^3

[1]90% reduction of 100 FFU BH RSV (RAV-1). [2]NT = meutralizing titer; mean titer in yolks from vaccinated hens was 250 times higher than in yolks from contact-exposed hens.
Source: Modified from deBoer *et al.* (1978).

The various features of avian leukosis discussed above led to the development of a program for the control of LL in breeder birds (8) as follows:

Control of Lymphoid Leukosis in Breeder Birds:

1. Select from the grandparent flock (female line) dams that produce only leukosis virus (LV)-free eggs (by examination of embryo-extracts with the NP test [other appropriate virologic tests are the COFAL and PM tests]).
2. Hatch only eggs from the selected dams.
3. The hatched chicks and later the adult birds have to be:
   a) reared in isolation (FAPP house) for 2 months;
   b) subsequently inoculated with a LV containing suspension (10^5 $TCID_{50}$ of subgroup A and B;
   c) then transferred to a conventional chicken house;
   d) examined for the presence of neutralizing antibodies in sera taken prior to and after inoculation with LV; and
   e) examined during lay for the presence of LV in embryo extracts.

An investigation was performed to examine whether the program for the control of LL also influenced egg production in a negative way. The production of groups of birds treated according to the program for control was compared to that of non-treated groups of birds serving as controls. The results did not reveal significant differences between both groups (unpublished data).

Under field conditions in the Netherlands, the program, for reasons given above, exclusively applied to breeder birds, has proved satisfactory. It appeared to be possible to free most breeder stocks under test from vertical LV transmission within a few generations and consequently from the disease (Table 3).

TABLE 3. Congenital leukosis virus transmission[1] in chickens before and after inclusion in the program for LL control

Generation	Strains[2]			Grandparent flock[2] W PL R	
	WL-A	WL-B	WL-98A	Test 1	Test 2
I	7/43[3]	8/33	15/39	9/500	9/437
II	11/41	2/22	7/32	0/368[4]	0/359[4]
III[4]	5/94	0/39	0/38	0/552	0/175
IV[4]	0/35	0/41	0/22	0/118	0/106
V[4]	2/73	0/15	0/20		

[1]Virus assay: NP activation test; per generation applied once for the White Leghorns and twice for the White Plymouth Rocks, i.e., egg collection periods of 2 weeks.

[2]WL = White Leghorn; W Pl R = White Plymouth Rocks

[3]Number positive hens/number hens tested.

[4]Generation reared in isolation and inoculated with LV at 8 weeks of age.

Source: Modified from deBoer et al. (1978).

Disadvantages of the Program for the Control of Leukosis:

1. Obligation to use isolation units for breeder chicks from 0 to 8 weeks of age.
2. Need for carefully instructed personnel.
3. Collection of serum samples (during rearing) and trapnested eggs.

Advantages of the Program for the Control of Leukosis:

1. Suppression of clinical leukosis in breeder birds and in egg product as well.
2. Presence of an age-dependent resistance to an as yet unknown number of diseases.
3. Various vaccinations - given usually between 0 and 4 weeks of age - are now applied after 4 weeks of age and result in higher levels of neutralizing antibodies (no interference with maternal antibodies; better immune response).
4. End product chicks do not require housing in isolation nor LV vaccination.

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Growth and Characterization of, and Immunological Response of Chicken to, a Cell Line Established from JMV Lymphoblastic Leukemia

Martin Sevoian and David Munch
Department of Veterinary and Animal Sciences
University of Massachusetts
Amherst, Massachusetts 01003

ABSTRACT

Continuous *in vitro* passage of JMV-1 tumor cells of Marek's disease resulted in adaptation of the cultured cells as evidenced by faster growth and increased cell viability. With increasing time in continuous passage, lethality declined. Antigenicity was retained, since chickens that survived inoculation with JMV-1 of lowered pathogenicity survived challenge with the more virulent wild type of JMV.

Immunization of S- and K-line chickens with these partially attenuated JMV-1 cells significantly reduced tumor incidence in chickens infected with Conn B MDV below that of unimmunized MDV-infected chickens. Immunization of chickens with JMV-1 cells also significantly reduced the level of rescuable MDV from infected chickens. Analysis of serum samples from these chickens also revealed an increase in precipitin antibodies against MDV. The titers were not due to JMV-1 cell immunization alone, since immunized non-MDV-infected chickens had no detectable MDV precipitin antibodies. These experiments have shown that JMV-1 immunization can significantly alter the pathogenesis of MDV infection and tumor development.

CRECIMIENTO, CARACTERIZACION Y RESPUESTA INMUNOLOGICA DE POLLOS A UNA LINEA CELULAR ESTABLECIDA A PARTIR DE LA LEUCEMIA LINFOBLASTICA JMV.

Pasajes contínuos *in vitro* de células tumorales JMV-1 de la Enfermedad de --- Marek (EM) produjeron una adaptación de las células cultivadas,que se hizo evidente por un crecimiento más rápido y un incremento en la viabilidad celular.
Al incrementar el tiempo en pasajes contínuos, decreció la letalidad.La antigenocidad fue retenida ya que los pollos que sobrevivieron a la inoculación con JMV-1 de menor patogenicidad,sobrevivieron al desafío con el tipo JMV de campo más virulento.
La inmunización de los pollos de las líneas S y K con las células JMV-1 parcialmente atenuadas, redujo significativamente la incidencia de tumores en los pollos que fueron infectados con el virus de la Enfermedad de Marek -- Conn B (Conn B MDV), en comparación con lo que ocurrió en los pollos que no fueron inmunizados y que se incularon con virus de la EM.
La inmunización de los pollos con celulas JMV-1 también redujo en forma significativa el nivel del virus de la EM obtenido a partir de los pollos infectados. El análisis de las muestras de suero obtenidas a partir de estos pollos reveló también un incremento en los anticuerpos precipitante contra el virus de la EM.
Los títulos no se debieron solamente a la inmunización con las células JMV-1, ya que en los pollos inmunizados que no fueron infectados con MDV no --- presentaron anticuerpos precipitantes detectables contra el virus de la EM. Estos experimentos han mostrado que la inmunización con celulas JMV-1 puede alterar significativamente la patogenésis de la infección por el virus de la EM y el desarrollo de tumores.

Traducción: Cortesía de la Dra. Luz Ma. Charles de López.

VIRAL AND TUMOR CELL IMMUNITY OF CHICKENS TO MAREK'S DISEASE

Martin Sevoian and David Munch
Department of Veterinary and Animal Sciences
University of Massachusetts
Amherst, Massachusetts 01003

Introduction

Marek's disease (MD) is a common malignant lymphoproliferative disease of poultry caused naturally by a herpesvirus. Most lymphoma cells are nonproductively infected, so that such tumors contain little or no infectious virus or viral antigens (1). Nevertheless, MD tumor cells transmit viral infections and induce the disease when inoculated into susceptible chickens. Rapid serial passage of the JM strain of MD cells in young chicks has resulted in a reduced incubation period and increased mortality (5). This strain, designated JMV, has been used effectively as a challenge strain to determine the immunity of chickens vaccinated against MD (3), has been developed as an *in vitro* cell line (2,4) for characterization studies, and has been used in investigations of MD (5,6,7).

Continuous *in vitro* passage of JMV-1 tumor cells of Marek's disease resulted in adaptation of the cultured cells as evidenced by faster growth and increased cell viability. With increasing time in continuous passage, lethality declined. Antigenicity was retained, since chickens that survived inoculation with JMV-1 of lowered pathogenicity survived challenge with the more virulent wild type of JMV.

Immunization of S- and K-line chickens with these partially attenuated JMV-1 cells significantly reduced tumor incidence in chickens infected with Conn B MDV below that of unimmunized MDV-infected chickens (Tables 1 and 3). Immunization of chickens with JMV-1 cells also significantly reduced the level of rescuable MDV from infected chickens (Table 2). Analysis of serum samples from these chickens also revealed an increase in precipitin antibodies against MDV. The titers were not due to JMV-1 cell immunization alone, since immunized non-MDV-infected chickens had no detectable MDV precipitin antibodies. These experiments have shown that JMV-1 immunization can significantly alter the pathogenesis of MDV infection and tumor development.

Table 1

Incidence of Marek's disease lymphomas in S- and K-line chickens immunized with 2×10^3 viable JMV-1 cells.

	JMV-1 immunized controls	JMV-1-immunized MDV-infected	Unimmunized MDV-infected	Unimmunized uninfected controls
S-line	0/20 (0.0%)a[A]	5/19 (26.3%)b	13/16 (81.2%)c	0/20 (0.0%)a
K-line	0/20 (0.0%)b	1/16 (6.3%)b	9/21 (42.9%)c	0/20 (0.0%)a

A. Treatment means followed by the same letter are not significantly different at $P > 0.05$ when tested by Duncan's multiple-range test.

Table 2

Viremic status of S- and K-line chickens measured at 2, 5, and 7 weeks postinfection with Conn B isolate of MDV

		Chicken[B]	MDV plaque-forming units/2 x 10^6 kidney cells[A] Weeks postinfection 2	5	7
S-line	Unvaccinated MDV-infected	1	1.5	107	122
		2	1.0	83	46
		3	1.4	138	38
		4	1.7	80	80
		$\bar{x}$	1.4	102	72
	JMV-1-vaccinated MDV-infected	1	0	6	0
		3	0	0	30
		4	0	0	44
		$\bar{x}$	0	2	25
	Isolated uninfected	1	0	0	0
		2	0	0	0
		$\bar{x}$	0	0	0
K-line	Unvaccinated MDV-infected	1	0	7	8
		2	0	48	13
		3	0	8	50
		4	0	0	226
		$\bar{x}$	0	16	76
	JMV-1-vaccinated MDV-infected	1	0	2	0
		2	0	1	0
		3	0	0	0
		4	0	0	0
		$\bar{x}$	0	1	0
	Isolated untreated controls	1	0	0	0
		2	0	0	0
		$\bar{x}$	0	0	0

A. Figures given represent the average number of PFU for 2-3 petri plates. Counts were made 6-7 days after initiation of cultures.

B. Four chickens from each treatment group were sacrificed at each test time, and their kidneys were cultured individually.

Table 3

Relative time of Marek's disease lymphoma incidence in vaccinated S- and K-line chickens sacrificed at specific intervals following exposure to Conn B isolate of MDV

		Incidence of MD lymphomas (weeks postexposure to Conn B isolate of MDV)		
		2[A]	5	7
S-line chickens	Unvaccinated MDV-infected	0/4	4/4	4/4
	JMV-1-vaccinated MDV-infected	0/4	0/4	1/4
K-line chickens	Unvaccinated MDV-infected	0/4	1/4	1/4
	JMV-1-vaccinated MDV-infected	0/4	0/4	0/4

A. Figures represent the number of MD lymphomas found at each time/the total number of chickens examined at that time.

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LIMITED PATHOGENIC POTENTIAL OF MAREK'S VACCINE (HVT) FOR CHICKENS AND TURKEYS

J. M. Sharma, BVSc, PhD, and A. K. Elmubarak, BVSc, PhD
U. S. Department of Agriculture
Science and Education Administration, Agricultural Research
Regional Poultry Research Laboratory
East Lansing, Michigan 48823

Pathogenicity of herpesvirus of turkeys (HVT) for chickens and turkeys was examined. HVT is antigenically and morphologically related to Marek's disease (MD) virus and is widely used as a vaccine against MD in the field. In several experiments, chickens lacking maternal antibody to MD virus and HVT were immunosuppressed by neonatal thymectomy followed by total body sublethal -irradiation. This regimen of immunosuppression adversely affected normal T-cell responsiveness because lymphoid cells of thymectomized chickens had reduced blastogenic response to in vitro stimulation with the mitogen phytohemagglutinin. Immunosuppressed chickens and intact hatchmates were inoculated with a high dose of HVT and subsequently examined for HVT viremia and gross and microscopic lesions. Immunosuppressed and intact chickens remained persistently viremic through the observation period of 5 weeks. The immunosuppressed chickens had a slightly higher frequency of microscopic lesions in peripheral nerves than the intact chickens although the lesions in both groups were very mild and no gross lesions or clinical disease was detected.

Herpesvirus of turkeys also induced persistent viremia in turkey poults but lymphoproliferative lesions were not detected during the observation period of 8 weeks. Immunosuppression by treatment with 14 mg of cyclophosphamide did not enhance the susceptibility of turkeys to lesion development by HVT.

This study indicated that pathogenic potential of HVT for chickens and turkeys is quite limited.

POTENCIAL PATOGENICO LIMITADO DE LA VACUNA CONTRA LA ENFERMEDAD DE MAREK (HVT) PARA POLLOS Y PAVOS.

Se examinó la patogenicidad del herpesvirus de los pavos (HVT) para los pollos y los pavos.
El HVT esta antigénica y morfológicamente relacionado con el virus de la Enfermedad de Marek y es ampliamente utilizado como vacuna contra esta enfermedad en el campo.
En algunos experimentos, pollos que carecían de anticuerpos maternos contra el virus de la Enfermedad de Marek y el virus HVT fueron sujetos a una inmunosupresión mediante una timectomía neonatal seguida de una irradiación subletal corporal total. Este régimen de inmunosupresión afectó adversamente la capacidad de respuesta de las células T,consecuentemente reduciéndose en las células linfoides de los pollos timectomizados su respuesta blastogénica hacia la estimulación in vitro con la Mitógeno Fito hemaglutinina.
Los pollos inmunosuprimidos y aquellos que nacieron junto con ellos pero sin ser sometidos a tratamiento fueron inoculados con una dosis alta de HVT y posteriormente examinados en busca de una viremia con HVT y lesiones macro y microscópicas.
Los pollos con inmunosupresión y los intactos permanecieron persistentemente virémicos a través de la observación realizada durante un periodo de 5 semanas.
Los pollos con inmunosupresión mostraron una frecuencia ligeramente mayor de lesiones microscópicas en los nervios periféricos que los pollos normales, no obstante que las lesiones en ambos grupos fueron muy leves y no se detectaron lesiones macroscopicas ni enfermedad clínica.
El herpesvirus también indujo en los pavos una viremia persistente pero no se detectaron lesiones linfoproliferativas durante el período de observación de 8 semanas.
La inmunosupresión con un tratamiento de 14 mg de ciclofosfamida no motivó la susceptibilidad de los pavos hacia el desarrollo de la lesión producida por el HVT.
Este estudio indicó que el potencial patogénico del virus HVT para los pollos y los pavos es bastante limitado.

Traducción: Cortesía de la Dra. Luz. Ma. Charles de López

LIMITED PATHOGENIC POTENTIAL OF MAREK'S VACCINE (HVT) FOR CHICKENS AND TURKEYS

J. M. Sharma, BVSc, PhD, and A. K. Elmubarak, BVSc, PhD
U. S. Department of Agriculture, SEA
Regional Poultry Research Laboratory
East Lansing, Michigan 48823

Herpesvirus of turkeys (HVT) is widely used as a vaccine against Marek's disease (MD), a disease syndrome in chickens that is characterized by lymphoid cell proliferation, tumor formation, loss of body weight, drop in egg production, immunodepression, and death. Immunization with live HVT minimizes the disease-inducing potential of virulent MD virus and associated economic losses.

Herpesvirus of turkeys is ubiquitous and infects most turkeys raised under field conditions. Chickens are refractory to natural infection with HVT, although they can readily be infected by inoculation. Horizontal or vertical transmission of HVT does not ordinarily occur in chickens. Further, HVT by itself has been considered harmless for chickens, turkeys, and mammals including man. The apparent lack of pathogenicity of HVT has no doubt contributed to the wide acceptance of this virus as a live vaccine.

A few years back, Witter et al. (1976) reexamined the effect of HVT on chickens. These workers found that when newly hatched chicks, particularly those lacking maternal antibody to HVT, were inoculated with HVT, the chickens developed microscopically detectable lymphoproliferative lesions in peripheral nerves and gonads. The lesions were generally quite mild, occurred soon after infection, and invariably disappeared within 2-3 weeks. Subsequently, several laboratories reported that chickens vaccinated with HVT also developed cells that expressed the tumor antigen of MD on their surface. This antigen, popularly known as MATSA (for Marek's disease tumor associated surface antigen), is considered by some to be expressed by cells that have become cancerous (transformed) as a result of virus infection, although there is some controversy on the relationship of MATSA to cancer. In any case, presence of MATSA-bearing cells and lymphoproliferative lesions in vaccinated chickens suggested to us that the pathogenic potential of HVT for chickens needed further examination. In this study, we examined the response of newly hatched, maternal-antibody-negative chickens to HVT. The chickens were immunosuppressed before virus inoculation so that any lesions would be maximally manifested. We also studied the response of intact and immunosuppressed turkey poults to HVT.

Chickens of lines P and 15x7, highly susceptible to tumor formation by MD virus were from our specific-pathogen-free flocks and lacked maternal antibody to HVT and MD virus at hatching. Chickens were divided into 2 groups. In one group chickens were surgically thymectomized (TX) within 24 hours of hatching and then irradiated with 600 rad of γ-rays. The chickens in the second group were left untreated. Peripheral blood cells from TX and intact chickens were examined for in vitro blastogenic response to the mitogen phytohemagglutinin (PHA). Chickens in both groups were inoculated with $2.0-3.2 \times 10^4$ plaque forming units of HVT and chronologically examined for viremia and gross and microscopic lesions. Viremia was quantitated by inoculating cell cultures of chicken embryo fibroblasts with peripheral blood leukocytes.

Turkey poults were a gift of Nicholas Farms, Sonoma, California.* Poults pretreated with 14 mg of cyclophosphamide (4 injections of 3.5 mg each given at days 2-5 after hatching) or untreated were injected with $4.0-8.0 \times 10^4$ plaque forming units of HVT. At intervals after inoculation, turkeys were examined for HVT viremia and gross and microscopic lesions. Chickens were included for comparison.

Peripheral blood cells of TX chickens were severely deficient in their response to PHA at 7, 11 and 21 days after thymectomy; the response returned to normal levels by day 35. These results suggested that neonatal thymectomy accompanied by γ-radiation caused immunosuppression of the T-cell function. Inoculation of immunosuppressed and intact chickens with HVT resulted in viremia that was detectable as early as 7 days postinoculation and persisted until 35 days postinoculation, the last time tested. In general, the immunosuppressed chickens had higher levels of circulating HVT than did the intact chickens.

The lesion response to HVT of line P chickens is given in Table 1. Microscopic lesions were slightly more frequent in TX chickens than in intact chickens, although lesions were invariably mild and no clinical disease or gross lesions were detected in any chicken. The response of line 15x7 chickens was similar to that of line P chickens.

Table 1

Response of immunosuppressed and intact line P chickens to herpesviruses of turkeys (HVT)

Days after HVT Inoculation	Lesions[a]			
	Thymectomized chickens		Intact Chickens	
	Gross	Microscopic[b]	Gross	Microscopic
7	0/7[c]	0/7	0/7	2/7 (1.0)
11	0/4	1/4 (1.0)[d]	0/7	2/7 (1.0)
21	0/7	5/7 (1.0)	0/6	1/6 (1.0)
35	0/15	5/15 (1.2)	0/23	1/23 (1.0)
7-35 (combined)	0/33	11/33 (1.1)	0/43	6/43 (1.0)

[a] 3 to 25 uninoculated control chickens examined at each interval lacked detectable gross or microscopic lesions.

[b] Lymphoproliferative lesions were detected primarily in peripheral nerves.

[c] No. of chickens with lesions/total no. examined.

[d] Numbers in parenthesis indicate mean lesion scores in positive chickens. Lesions in each chicken were scored on a scale of 1-4 on the basis of increasing intensity of lymphoproliferation.

Turkey poults also developed persistent viremia after inoculation with HVT. The lesion response shown in Table 2 indicated that turkeys were refactory to lymphoproliferative lesions of HVT despite pretreatment with 14 mg of cyclophosphamide. The cyclophosphamide treatment in turkeys abrogated antibody response to two serial injections of sheep erythrocytes and Brucella abortus.

Table 2

Response of immunosuppressed and intact turkeys and chickens to herpesviruses of turkeys (HVT)

Experiment	Inoculum		Observation period (weeks[b])	Lesions[a]			
	14 mg Cyclophosphamide	HVT		Turkeys		Chickens	
				Gross	Micro.	Gross	Micro.
1	-	+	8	0/32[c]	0/32	0/32	4/32
2	-	+	4	0/10	0/10	0/17	7/17
3	+	+	3	0/6	0/6	Not done	
	-	-		0/8	0/8	Not done	

[a] Peripheral nerves, visceral organs and skin were examined. Lesions in chickens were lymphproliferative in nature and were noted primarily in the peripheral nerves.

[b] 2-5 turkeys and an equivalent number of chickens were examined at weekly or biweekly intervals. Data from all observations were pooled.

[c] No. of animals with lesions/total no. tested.

The above results with chickens and turkeys indicated that immunosuppression did not substantially enhance the pathogenicity of HVT for these species. This and previous evidence suggestive of the limited disease-inducing potential of HVT is of practical significance, because live, infectious HVT is used the world over as a vaccine against MD.

Reference

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EFICACIA DE LA VACUNA HERPES DEL PAVO EN POLLOS DESAFIADOS CON CEPAS DE MAREK AISLADAS RECIENTEMENTE

C. S. Eidson, M. N. Ellis y S. H. Kleven
Department of Avian Medicine
Poultry Disease Research Center
College of Veterinary Medicine
University of Georgia
Athens, Georgia 30605

Cuatro cepas de virus Herpes de Marek fueron obtenidas a partir de reproductoras de 21 semanas de edad que mostraban lesiones y mortalidad debidas a la enfermedad de Marek. Estas aves habían sido vacunadas correctamente contra la enfermedad. Las cepas aisladas fueron identificadas como Ala-7, 8, 9 y 10.

Estas cuatro cepas fueron comparadas frente a la cepa GA del virus Herpes de Marek en pollos vacunados con dosis variables de vacuna preparada con virus Herpes del pavo. El desafío fué hecho con el virus Herpes de Marek a dosis variables.

Todas las 5 cepas de Marek aisladas demonstraron virulencia similar. Sin embargo, la protección producida por el virus vacunal Herpes del pavo frente al desarrolo de lesiones de Marek fué significantemente menor en las cepas Ala. 7, 8 y 9 que la protección obtenida con las cepas Ala. 10 y GA.

Las cepas Herpes de Marek Ala. 7, 8 y 9 indujeron la producción de placas de tamaño pequeño en monocapas de fibroblasto de embrión de pollo; la cepa Ala. 10 indujo la presentación de placas grandes y pequeñas, y la cepa GA produjo predominantemente placas pequeñas con unas pocas placas de tamaño mayor.

EFFICACY OF THE TURKEY HERPESVIRUS VACCINE IN CHICKENS CHALLENGED WITH RECENTLY ISOLATED MAREK'S DISEASE VIRUSES

Four isolates of Marek's disease herpesvirus (MDV) were obtained from 21 week old broiler breeders with lesions and mortality from Marek's disease (MD). They had been properly vaccinated with turkey herpesvirus (HVT). The isolates were designated Ala-7, 8, 9, and 10.

These 4 MDV strains were compared with the GA isolate of MDV in chickens vaccinated with graded doses of HVT. Challenge with the MDV strains was also with graded doses. All 5 isolates of MDV were similar in virulence. However, protection afforded against MD lesions by HVT was significantly lower in the Ala-7, 8, and 9 isolates than with the Ala-10 or GA isolates.

The Ala-7, 8, and 9 isolates of MDV induced small-cell plaques in chick embryo fibroblasts, the Ala-10 isolate induced both large and small cell plaque types, and the GA isolate produced predominantly small cell plaques with a few plaques of the large cell type.

Abstract

DIFFERENCES BETWEEN MAREK'S DISEASE VIRUS STRAINS

B. W. Calnek
Department of Avian and Aquatic Animal Medicine
New York State College of Veterinary Medicine
Cornell University, Ithaca, New York 14853

Marek's disease virus (MDV) strains vary in degree of oncogenicity based on the incidence of neoplasms they induce. Factors such as genetic strain of the host, age at infection, and presence or absence of maternal antibodies influence the degree to which oncogenic potential is expressed. Thus, low virulence MDVs might cause MD only in young, genetically susceptible strains while high virulence MDVs might induce lymphomas even in older, resistant strains. There is presently some concern that some MDVs present in commercial flocks are sufficiently virulent to overwhelm the protection normally afforded by MD vaccines. The reason(s) for differences in oncogenicity are essentially unknown.

To compare 3 viruses considered to be of moderate, high or very high virulence (JM-10, GA-5 and ALA-8, respectively), young genetically susceptible P-line or resistant N-line birds were infected and then examined at 5 and 20 days postinfection (DPI). Virus isolated from spleen, cytolytic infection in lymphoid organs and gross changes in the bursa and spleen were used to assess the host's initial response and ability to curtail infection. Early infections were severe and similar in all groups. Bursal atrophy resulted from JM-10 and ALA-8, but not JM-10 infection in P-lines and was especially marked with ALA-8. Only ALA-8 virus caused bursal atrophy in N-lines. Levels of virus infection and VIA expression also were greater in ALA-8 than JM-10 or GA-5 infections at 20 DPI. Prior vaccination with turkey herpesvirus prevented early cytolytic infections with all 3 MDV strains. Splenic enlargement, normally observed at 20 DPI, was less pronounced in vaccinated N-lines than in all other groups.

It appears probable that the degree of oncogenicity is directly related to the extent of immunosuppression induced; this warrants further study.

DIFERENCIAS ENTRE VARIAS CEPAS DEL VIRUS DE LA ENFERMEDAD DE MAREK.

Resúmen.

Las cepas del virus de la enfermedad de Marek (V.E.M.) varían en su grado de oncogenicidad basado en el índice de inducción de neoplasmas. Factores táles como las características genéticas del hospedador, edad al momento de la infección y presencia o ausencia de anticuerpos maternos, determinan el potencial oncogénico.
Un V.E.M. de baja virulencia puede causar la enfermedad solamente en aves jóvenes genéticamente susceptibles. Un V.E.M. de alta virulencia puede inducir linfomas aún en animales de edad madura o en razas resistentes. Existe actualmente la preocupación de que algunos V.E.M presentes en parvadas comerciales sean suficientemente virulentos para rebazar la protección normalmente conferida por la vacuna contra la enfermedad de Marek (E.M.). Las razones de la diferencia en la oncogenicidad son desconocidas.
Se comparáron 3 virus considerados como de virulencia moderada, alta y muy alta (JM-10, - - GA-5 y ALA-8 respectivamente). Aves jóvenes geneticamente suceptibles Línea P y Línea N - fuéron infectados y examinados a los 5 y 20 días post-infección (D.P.I). Se aislaron virus a partir del Bazo. La infección citolítica en órganos linfoides y cambios macroscópicos en la Bolsa y en el Bazo se emplearon para medir la respuesta inicial del hospedador y su habilidad para bloquear la infección. Las infecciones tempraneras fueron severas y similares en todos los grupos.
La infección por JM-10 y ALA-8 produjo atrofia bursal, mientras que la JM-10 no la produjo en la Línea-P pero fué especialmente marcada con la ALA-8. Unicamente la ALA-8 causó atrofia de la Bolsa en la Línea-N. Los niveles de infección viral y la expresión V.I.A. fuéron igualmente más grandes en ALA-8 que en JM-10 o GA-5 a los 20 D.P.I. La vacunación previa con virus herpes de pavo previno infecciones citolíticas tempraneras con las 3 cepas del - V.E.M.
Esplenomegalia se observó a los 20 D.P.I. aunque fué menos severa en las aves vacunadas de la Línea-N que en los otros grupos.
Es probable que el grado de oncogenicidad esté directamente relacionado con el grado de - inmunosupresión inducida, lo cuál justifica la continuación de ésta investigación.

Traducción: cortesía del Dr. Miguel A. Márquez R.

DIFFERENCES BETWEEN MAREK'S DISEASE VIRUS STRAINS

B. W. Calnek
Department of Avian and Aquatic Animal Medicine
New York State College of Veterinary Medicine
Cornell University, Ithaca, New York 14853

Marek's disease virus (MDV) strains fall into 2 serological groups which are related, but distinct from one another. Group 1 viruses, such as JM, GA, HPRS-16, CU, etc., are all oncogenic, although to varying degrees. Group 2 viruses, as represented by HN, HPRS-24, and SB are characterized as avirulent or nononcogenic. Turkey herpesvirus (HVT) constitutes the 3rd related serological group and, like group-2 MDVs, is avirulent. Both avirulent virus types are suitable vaccines against the oncogenic strains.

SB-1 and HVT share certain features which distinguish them from oncogenic MDVs. In vitro, they grow readily in CEF cultures, in contrast to strains like JM or GA which have a marked preference for CK cultures (6). In vivo, all 3 virus types display a common pattern of early localization in the lymphoid organs, but HVT and SB-1 cause very little or no cytolytic infection. Therefore, there is no bursal or thymic atrophy, and immunocompetence is retained (3). This is a key point which could be the reason for their failure to induce tumors (i.e., the bird remains able to destroy transformed cells), although it is also possible that they lack transforming genes. The reasons for the marked differences in oncogenicity among the viruses in serotype-1 are still largely unknown.

Vaccination against MD with either HVT or SB-1 protects against the early cytolytic infection normally associated with challenge by oncogenic virus strains (2). It has been known for some time that vaccination is less efficacious in chickens which are genetically susceptible to MD than in those genetically resistant (7). Also, some of the oncogenic strains of MD are better able than others to override genetic resistance. Since vaccinal protection is immunological and genetic resistance is apparently related to immune competence (5), then it is plausible that protection from vaccination might be especially poor in certain combinations of genetic strains and virus strains.

Recently, there have been reports of vaccine "breaks" in commercial flocks of broilers and layers. So-called "variant" strains of MDV have been isolated and characterized as especially immunosuppressive, and better able than other strains to induce MD in vaccinated birds (4, 8, 9). In an attempt to gain some insight into possible mechanisms to explain this exceptional virulence, 2 experiments were conducted. Variables were genetic strain and virus strain. Susceptible P-line and resistant N-line chickens were challenged at about 2 weeks of age with viruses of moderate (JM-10), high (GA-5) or reportedly very high (ALA-8, kindly supplied by Dr. C. Eidson). Some of the challenged birds in the 2nd experiment had been vaccinated with HVT at 2 days of age. Chicks were examined at 5 and 20 days post infection (DPI). Virus isolated from the spleen, cytolytic infection detected by the fluorescent antibody test in lymphoid organs and feather follicle epithelium (FFE) and pathological changes in the bursa were used to assess the host's initial response and ability to curtail infection. Results are in Table 1.

The data from these experiments illustrate several points which are important in assessing the pathogenicity of a given MD virus and the degree of protection offerred by HVT. From the results of virus isolation attempts and the FA tests to detect cytolytic infection, it can be seen that, as reported before (1), the early pathogenesis does not readily distinguish among virus strains or genetic strains. Major differences were not observed at 5 DPI. However, both the virus strain and the genetic strain were important in determining: 1) The extent to which these infections were curtailed by 20 DPI, and 2) the degree of damage inflicted upon organs like the bursa. JM infection, which is the least oncogenic of the 3 tested, was markedly reduced by 20 DPI in N-lines based on the failure to isolate virus from spleen and the presence of very little or no antigen in the FFE. This suggests a strong host immune response. Further, no bursal damage was observed at that time. P-lines, in contrast, had marked infection in the FFE and some bursal atrophy from necrosis.

At the other end of the scale was the ALA-8 virus infection, which was not effectively curtailed in either N-lines or P-lines and caused the most severe bursal damage. GA virus infection was perhaps intermediate but had the unusual and unexplained property of not inflicting severe changes on the lymphoid organs. Witter et al. (9) reported the same observation.

Table 1

Effects of Virus Isolate, Genetic Strain and Vaccination on Pathogenesis of Marek's Disease In P-line (P) and N-line (N) chickens examined at 5 and 20 days post MDV infection (DPI).[a]

Exper. no.	HVT vacc.	MDV chall.	Virus isolations-spleen: FFU/10^6 cells				Viral antigen: Average FA test score (maximum 4.0)[b]				Bursal changes 20 DPI:			
			5 DPI		20 DPI		Lymphoid organs 5 DPI		FFE 20 DPI		Rel. bursa weight		% follicles damaged	
			P	N	P	N	P	N	P	N	P	N	P	N
1	-	-	0	0	0	0	0	0	0	0	(1.0)	(1.0)	0	0
		JM	4	4	6	0	1.9	1.5	3.1	0	0.83	0.82	2.5	0
		GA	...[c]	3	...	12	...	1.8	...	2.4	...	0.79	...	0
		ALA	21	10	74	42	2.8	3.2	3.8	3.7	0.41*[d]	0.51*	50.4	5.0
2	-	-	0	0	0	0	0	0	0	0	(1.0)	(1.0)	0	0
		JM	42	28	9	0	2.3	2.4	3.5	0.5	0.66*	0.89	0	0
		GA	8	3	21	1	0.5	1.6	2.8	2.4	0.84	1.16	0	0
		ALA	22	7	22	1	2.8	2.1	4.0	3.0	0.48*	0.61*	5.6	24.8
	+	-	0	0	0	0	0	0	0	0	(1.0)	(1.0)	0	0
		JM	0	0	0	0	0	0	1.6	0	0.80*	1.19	0	0
		GA	0	0	0	0	0	0	1.9	2.2	0.86	1.05	0.3	0
		ALA	0	0	4	0	0	0	2.9	1.0	0.93	1.07	3.3	0

[a] Averages from groups of 3-8 birds.

[b] Lymphoid organs = bursa, spleen, thymus; FFE = feather follicle epithelium.

[c] ... = not done.

[d] * = P<0.05

Vaccination prior to challenge effectively prevented early cytolytic infections by all challenge strains of MDV, although it appeared to be least effective in the case of ALA-8 challenge in P-lines and most effective with JM-10 challenge in N-lines.

These data support the conclusion that both genetic constitution and MD virus strain are important factors in determining the efficacy of field vaccination against MD. It seems probable that "breaks" may relate to both of these factors and perhaps to others as well. Other infections, management factors, maternal antibodics, etc. may contribute to expression of the oncogenic potential which is determined by the virus and the host. The reason why some MD virus strains override the immunity induced by HVT still needs definition.

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MAREK'S DISEASE VACCINE BREAKS

J. M. Sharma, BVSc, PhD
U. S. Department of Agriculture, SEA
Regional Poultry Research Laboratory
East Lansing, Michigan 48823

Marek's disease (MD) has been a problem of long standing. Although wide usage of herpesvirus of turkeys (HVT) as a vaccine has substantially reduced MD losses, the reports of vaccine breaks and associated economic losses continue to surface. What causes these breaks and how can they be prevented? Unfortunately this question may not have a simple and clear answer. As we make inroads into the understanding of the mechanism of protection by HVT, we are realizing that protection may be mediated through a chain of events and a break at any of the various links in this chain may interfere with protection. Let us briefly examine how HVT works and what are the possible sources of vaccine failure.

There is clear evidence that immunity plays an important role in vaccine protection. When HVT is injected into a chicken, the vaccine stimulates the development of antiviral and antitumor immunity. Because HVT is closely related to MD virus, the immunity generated by HVT cross reacts against MD virus and provides protection against MD. The antiviral immunity keeps MD virus from replicating to high levels and antitumor immunity minimizes tumor formation by MD virus. There seems to be a critical balance between vaccinal immunity and protection: if vaccinal immunity does not develop or is compromised, there is a breakdown of protection. Following are some of the parameters that must be considered while evaluating success or failure of a vaccination program:

1. Vaccine virus must infect chickens.

The process of vaccination must result in introduction of sufficient quantity of live infectious HVT in the chickens. Without good infection, the immune system will not respond. Using a vaccine of questionable quality, using improper diluent, not following manufacturers instructions and faulty vaccination procedures may result in lack of proper infection of chickens with HVT. Maternal antibody may also, to a certain extent, interfere with proper infection with the vaccine virus, particularly if cell-free vaccine of low titer is used.

2. There should be sufficient interval between vaccination and challenge.

Vaccine does not provide immediate protection. The vaccinated chicken needs time to first establish infection with HVT and then mount an immune response before a full measure of protection is developed against the challenge virus. Perhaps certain breaks in broiler flocks may occur because chickens are moved into heavily contaminated brooder houses too soon after vaccination. The challenge in these chickens is not only massive, but also occurs before vaccine has had a chance to establish solid immunity. Exactly how much lead time is necessary for the vaccine to induce immunity needs to be determined. Some people feel that holding vaccinated chicks a few hours or overnight in the hatchery and thus delaying challenge has beneficial effect, although experimental data are needed to evaluate this possibility.

3. Vaccinal immunity must persist through the life of the chicken.

Under ordinary circumstances vaccination at hatching can be expected to immunize chickens for life. However, if, at a certain stage, vaccinated chickens undergo stress, the immunity may break. The types of stress that most commonly result in vaccine breaks are not known, although theoretically, physiological stress (egg-laying), environmental stress (too cold or too hot) or disease outbreaks (IBD, REV) may immunodepress chickens. It has been difficult under laboratory conditions, to reproduce stressful situations that would result in vaccine breaks with a predictable regularity. As a general rule, good management practices that would minimize stressful situations should be followed.

4. Vaccine immunity must cross-react against challenge virus.

Recent evidence indicated that HVT may not be equally effective against all field viruses. Certain MD viruses isolated from break flocks were poorly protected against by conventional HVT vaccination procedures. Considerable effort is underway in our laboratory and in others to study the nature of these MD isolates and to determine the extent to which such isolates may be involved in vaccine breaks. If research shows that MD isolates that escape protection by HVT are widespread, then other suitable vaccines may have to be developed to protect against these viruses.

Considerable progress has been made within the last few years in understanding vaccine breaks, but there is an obvious need for continued research in this area. A free flow of information between the industry and the research laboratories must be maintained to sustain the extent of research effort needed.

ANTIBODY TITER TO ADENOVIRUS 127 IN LONG ISLAND DUCKLINGS FROM ONE WEEK TO SIX WEEKS OF AGE

G. M. Schloer, Ph.D.
Plum Island Animal Disease Center
Greenport, NY 11944

S. Haider, D.V.M., Ph.D.
Cornell University, Duck Research Laboratory
Eastport, NY 11941

SUMMARY

Commercial duck flocks in the United States show a high frequency of hemagglutination-inhibition (H.I.) antibody to adenovirus 127. Five flocks on Long Island, NY, including the flock at the Duck Research Laboratory (DRL), were examined in the spring of 1978. Ducklings were bled at weekly intervals beginning at one week of age until the ducklings were six weeks of age. Random samples were taken from ten ducklings from each of five flocks per week. All of the flocks showed evidence of H.I. antibody at one to two weeks of age. The number of positive sera in each flock of ducklings at one to two weeks of age ranged from 40 to 90%. None of the flocks had demonstrable H.I. titlers at three weeks of age. All flocks showed some rise in H.I. titer at four to six weeks of age. The increase in the number of positive sera in ducklings from four to six weeks of age differed considerably among the five flocks tested. Only three of ten sera from six-week-old ducklings of the DRL flocks had positive titers compared to 70 to 100% positive in four-to-six-week-old ducklings in three of the flocks. Sera from the DRL breeder ducks bled in February and August showed a similar distribution of H.I. titer, with 87% of the sera in February and 74% of the sera in August having H.I. titers ranging from four to 128. The results show that ducklings one to two weeks of age have maternal antibody which is not detectable in three-week-old ducklings. The rise in antibody in four-to-six-week ducklings appears to be associated with the acquisition of active infection with an adenovirus 127-like agent.

TITULOS DE ANTICUERPOS CONTRA EL ADENOVIRUS 127 EN PATOS DE 1 a 6 SEMANAS DE EDAD EN LONG ISLAND

RESUME

Parvadas comerciales de patos en los E.U.A. presentan una elevada frecuencia de anticuerpos inhibidores de la hemoaglutinación (I.H.) contra el adenovirus 127. Cinco parvadas en Long Island, NY (incluyendo la parvada localizada en el laboratorio de investigaciones de patos (LIP) fueron examinadas en la primavera de 1978. Se tomaron muestras sanguineas semanales desde la la. hasta la 6a. semana de edad. Muestras al azar se obtuvieron semanalmente de 10 patos en cada una de las parvadas. Todas las parvadas demonstraron la presencia de anticuerpos I.H., entre la la. y 2a. semana de edad.

El porcentaje de sueros positivos en cada parvada de patos de 1 a 2 semanas de edad varió desde 40% hast 90%. Ninguna de las parvadas mostró títulos I.H. a la 3a. semana de edad todas las parvadas mostraron un aumento en los títulos I.H. entre la 4a. y 6 a. emana de edad. El incremento en el número de sueros positivos en patos de 4 a 5 semans de edad, difirió considerablemente entre las 5 parvadas muestreadas. Solamente 3 de 10 sueros de patos de 6 semanas de edad de L.I.P. tuvieron títulos positivos comparados con 70% a 100% positivos en 3 parvadas de patos de 4 a 6 semanas de edad. Suero de reproductoras del L.I.P. sangradadas en febrero y agosto mostraron una distribución similar de títulos de I.H. con 87% de los sueros en febrero y 74% en agosto, teniendo títulos entre 4 y 128. Los resultados señalan que los patos entre la la. y 2a. semana de edad tienen anticuerpos maternos que no son detectados en aves de 3 semanas de edad. El aumento de anticuerpos en patos de 4 a 6 semanas parece estar asociado con la infeción activa por un adenovirus 127.

Traducción: Cortesía del Dr. Carlos López Coello.

Introduction

Egg drop syndrome 1976 (EDS 76) of chickens is associated with a new hemagglutinating adenovirus, adenovirus 127, which may have originated from ducks (1,3). Most commercial duck flocks in the United States have antibody to adenovirus 127 (2,4), and a virus similar to the EDS agent has been isolated from a number of duck flocks (1,5 and Calnek, personal communication). Although adenovirus 127 may be of duck origin, little is known about the epizootiology of adenovirus 127 in ducks.

A seriological survey of young ducklings from one to six weeks of age was done on five commerical duck flocks on Long Island. In general duckling production begins in the spring and terminates in the fall. The survey of duck flocks began in the spring of 1979. At weekly intervals random samples of blood were taken from ten ducklings at each farm. Table 1 lists the size of the flocks and the source of the ducklings. Flocks B and C were obtained from the same hatchery, while the remaining flocks were obtained from breeder ducks at the individual farms. Serum from breeder ducks from Flock D were obtained from 23 ducks bled in February and 37 ducks bled in August, 1979.

TABLE 1. Origin and Size of Duck Flocks Used in Survey

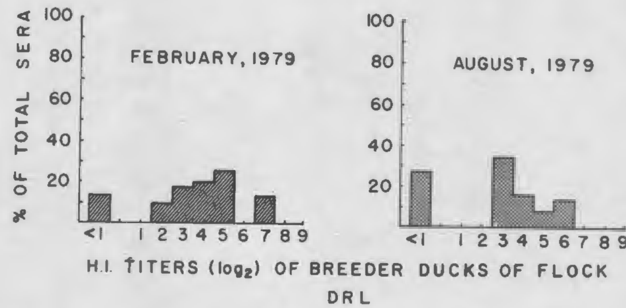
Flock	Size of Flock[a]	Source of Ducklings	Condition of Premises
A	2,000	own breeders	poor
B	2,000	hatchery	good
C	10,000	hatchery	fair
D	500	own breeders	good
E	2,000	own breeders	fair

[a]Number of ducklings processed per week.

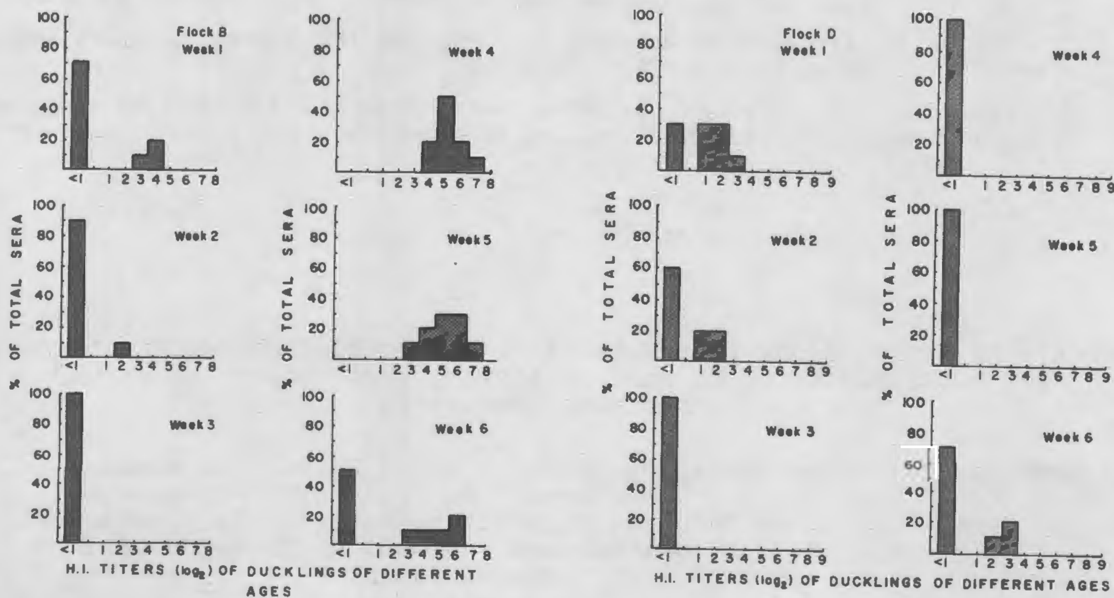
Serum samples were assayed for hemagglutination inhibition (H.I.) antibodies to adenovirus 127 using a standard test described previously (4). A precipitin test was done on glass slides using 0.6% agarose in 8.0% NaCl and 0.1M phosphate buffer pH 7.4. The antigen was obtained from infective allantoic fluid, which was concentrated by centrifugation at 20,000 rpm for 1 hr. Soluble antigen was obtained by disrupting the virus pellets with 1% SDS.

Results

Fig. 1 shows the distribution of antibody in the breeder ducks from Flock D bled at two different intervals. The distribution of titers shows no significant difference between February and August. Moreover, it is not different from the titers from the same flock bled the previous year (4).



All of the five flocks examined showed a similar distribution of H.I. titers from sera of ducklings bled at weekly intervals from one to six weeks. All had evidence of maternal antibody at one to two weeks of age, which was not detectable at three weeks of age. This was followed by an increase in titer in ducklings at four to six weeks of age. Figures 2 and 3 show the distribution of titers in Flocks B and D. The distribution of titers are similar, both flocks showing positive sera at six weeks. However, the rise in titer in Flock B began at four weeks. In Flock B all sera were positive from ducklings at four and five weeks of age.



A comparison of the precipitin with the H.I. antibodies shows some correlation between the two tests, Table 2. The correlation is not absolute, some sera were positive in the precipitin test and negative in the H.I. assay and vice versa.

TABLE 2. Comparison of Precipitin Test and Hemagglutination-Inhibition (H.I.) Test for the Detection of Antibodies to Adenovirus 127 in Ducklings of Different Ages.

Flock	Duckling Age in Weeks	Number Positive/Total	
		H.I.	Precipitin
A	1	9/10	4/10
	2	1/10	2/10
	3	0/10	0/10
	4	0/10	0/10
	5	1/10	0/10
	6	7/10	2/10
B	1	7/10	6/10
	2	4/10	7/10
	3	0/10	0/10
	4	0/10	1/10
	5	0/10	0/10
	6	3/10	1/10

Conclusion

Results of the survey show that many week-old ducklings have maternal antibody to adenovirus 127 which declines to undetectable levels at three weeks of age. The gradual use in antibody in ducklings from four to six weeks of age indicates that the ducklings acquired an active infection after the decline in maternal antibody. Acquisition of infection varied among the flocks. Cleanliness of the premises was not directly related to the rise in H.I. titer to adenovirus 127.

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REPORTE EN MEXICO SOBRE LA PRESENCIA DE ANTICUERPOS CONTRA EL ADENOVIRUS CAUSANTE DEL SINDROME DE LA BAJA EN POSTURA (CEPA BC-14) EN PARVADAS DE GALLINAS DOMESTICAS

G. Rosales, M.V.Z., A. Antillón, M.V.Z., M.S., Ph.D. y C. Morales, M.V.Z.
Departamento de Producción Animal:Aves
Facultad de Medicina Veterinaria y Zootecnia.
Universidad Nacional Autónoma de México
México 20, D. F.

DETECTION OF ANTIBODIES AGAINST EDS - VIRUS (BC - 14 STRAIN) IN DOMESTIC HENS IN MEXICO

One thousand one hundred blood samples were obtained from 67 chicken flocks throughout Mexico. Four hundred and six samples were positive to Adenovirus antibodies using the agar gel precipitin (AGP) test.

Hemagglutination inhibition (HI) tests were performed on the 406 AGP-positive samples; 318 were negative, 36 had titers of less than 1/40, and 52 were considered positive (titers of 1/40 or higher).

Most of the positive samples came from commercial medium-size layers and heavy breeders.

En un estudio serológico que abarcó 67 parvadas de gallinas domésticas, se estudiaron 1,100 muestras de sueros procedentes de distintas partes de la Republica Mexicana.

Por medio de la prueba de inmunodifusión en agar (IDA), se encontraron 406 muestras positivas a la presencia de anticuerpos contra Adenovirus; en todas las muestras positivas se corrieron pruebas de IH resultando 318 sueros negativos, 36 tuvieron un título menor de 1/40 y 52 sueros fueron considerados positivos por alcanzar títulos de 1/40 o superiores.

Los hallazgos de títulos IH elevados se circunscriben casi exclusivamente a parvadas comerciales de gallinas semipesadas y reproductoras de raza pesada.

INTRODUCCION

Actualmente la avicultura comercial se enfrenta a una nueva enfermedad (1), que ha sido identificada y estudiada a partir del año de 1976 en países del continente europeo principalmente (3,9,12,13); este padecimiento se conoce con el nombre de "Síndrome de la Baja de Postura", "Síndrome de la Baja de Huevos", o "Síndrome de Caída de la Puesta 76" (3,9,12,13), cuyo agente etiológico es un virus considerado hasta el momento como perteneciente a la Familia de los Adenovirus (3,5,8,10).

En nuestro país, se han venido observando en los últimos años, una serie de problemas relacionados con bajas de postura en la etapa de mayor producción, o bien con una inexplicable y pobre curva estadística de la postura. En algunos casos, se ha establecido la presencia de enfermedades de sobra conocidas en nuestro medio, mientras que en otros no ha sido posible llegar a conclusiones satisfactorias.

La fuerte sospecha de que existe la enfermedad denominada "Síndrome de la Baja en Postura" en el cono sur del continente americano, aunada a la observación de casos clínicos similares a esta adenovirosis en nuestro país,han creado inquietud en el medio avícola por conocer si esta nueva enfermedad está presente en la República Mexicana.

Por tal razón, el objetivo primordial de este trabajo, es realizar una encuesta serológica en parvadas comerciales de gallinas domésticas procedentes de distintas regiones de la República Mexicana, para determinar si existen anticuerpos contra el Adenovirus BC-14 (Baxendale, 1976), causante del "Síndrome de la Baja en Postura".

MATERIALES Y METODO

El estudio tuvo una duración de 10 meses durante los cuales se obtuvieron sueros de 67 parvadas de gallinas domésticas comerciales procedentes de diferentes Estados de la República Mexicana. Se analizaron 1,100 muestras divididas en 220 sueros de 21 parvadas de aves de raza ligera, 111 sueros de 8 parvadas de gallinas de raza semipesada productoras de huevo café, 114 sueros de 20 parvadas de reproductoras semipesadas y ligeras y 625 sueros de 20 parvadas de reproductoras de raza pesada.

La edad de las aves varió entre las 28 y las 60 semanas, habiendo 8 parvadas de menos de 28 semanas y 7 parvadas de más de 60 semanas de edad.

Para las pruebas serológicas de inmunodifusión en agar (IDA), así como la correspondiente para la de inhibición de la hemoaglutinación (IH), se utilizaron antígenos a base de la cepa BC-14, obtenidos gentilmente por el Dr. Baxendale*.

RESULTADOS

De las 1.100 muestras de sueros de gallinas domésticas procedentes de 67 parvadas comerciales localizadas en distintos lugares de la República Mexicana, 406 resultaron positivos a la presencia de anticuerpos contra la cepa BC-14, por medio de la prueba de inmunodifusión en agar (Cuadro 1).

Las aves semipesadas productoras de huevo rojo, tuvieron una mayor incidencia (43.2%) observandose anticuerpos en 6 de las 8 parvadas muestreadas; en este orden siguieron las reproductoras pesadas (40.0%) con presencia de anticuerpos en 20 de 29 parvadas;ponedoras de huevo blanco (32.7%)en 16 de 21 parvadas;reproductoras de raza semipesada (18.7%)en 2 de 4 parvadas, y por último en reproductoras ligeras (13.7%)en 3 de 5 parvadas.Se encontró que las parvadas positivas serológicamente estuvieron distribuidas casi en todos los estados muestreados.

*W.Baxendale, Intervet Laboratories Ltd.,Huntington,Cambridgeshire,Inglaterra

Dado que el número de sueros era elevado y no se contaba con la suficiente cantidad de antígeno, se decidió realizar pruebas de inhibición de la hemoaglutinación, exclusivamente en las 406 muestras de sueros que habían resultado positivas a la prueba de inmunodifusión en agar.

Los resultados de estas 406 muestras de sueros fueron los siguientes:318 resultaron negativos, 36 muestras alcanzaron un título menor de 1/40 y 52 sueros fueron considerados positivos al alcanzar títulos de 1/40 o más (Cuadro 2). En estos últimos sueros positivos se encontraron títulos que variaban desde 1/40 hasta 1/320, observándose la mayor cantidad de sueros positivos en 1/80 (Cuadro 3).

En cuanto a la incidencia de animales positivos a la prueba de IH, en relación a su tipo zootécnico, se observaron resultados similares a los expresados en el Cuadro 1, es decir, las aves ponedoras semipesadas productoras de huevo rojo tuvieron el porcentaje más elevado de sueros positivos con el 16.6%, encontrandose niveles IH en dos parvadas; las reproductoras pesadas con el 15.9% correspondiente a 6 parvadas; las ponedoras de huevo blanco con 27% obtenido de una sola parvada; mientras que las reproductoras de raza semipesada y ligera no presentaron ningún suero con niveles IH (Cuadro 4).

En cuanto a la distribución geográfica de las parvadas en las que se encontraron niveles elevados de IH, se observó que las ponedoras de huevo café provenían del Estado de Puebla, mientras que las reproductoras pesadas provenían en su mayoría del Estado de Nuevo León y una parvada del Estado de Morelos. Finalmente, los sueros positivos de ponedoras de huevo blanco procedían también del Estado de Puebla.

DISCUSION Y CONCLUSIONES

Existe información científica que demuestra la presencia de cierto grado de antigenicidad cruzada entre la cepa BC-14 y otros adenovirus (7,11).En el presente experimento se encontraron bandas claras de precipitación en 406 de un total de 1,100 muestras de sueros, lo cual sugiere que en las parvadas muestreadas hubo infecciones por adenovirus en alguna etapa de su vida.

Este dato nos indica que las infecciones por adenovirus son frecuentes,aunque no se pueda establecer una relación directa con presentaciones clínicas bien definidas.

Los resultados también indicaron que hay una mayor incidencia de anticuerpos IH en las parvadas de gallinas semipesadas productoras de huevo café, seguida por las reproductoras de raza pesada, y en un nivel más bajo en las ponedoras de raza ligera. Estos hallazgos concuerdan con las observaciones clínicas e investigaciones hechas en Europa (6,13).

Por otro lado, las pruebas de IH tienen un importante significado, ya que indican la presentación localizada de infección activa con títulos elevados de anticuerpos en parvadas de gallinas semipesadas productoras de huevo café en el Estado de Puebla, y en parvadas de reproductoras de raza pesada en el Estado de Nuevo León. Este dato tiene gran importancia desde el punto de vista sanitario ya que permitiría su rápido control por medio de programas de vacunación.

Desafortunadamente en los experimentos a nivel de campo en los cuales se obtiene material de parvadas heterogéneas, sin datos anamnésicos, de diferentes procedencias y tipos de manejo, hacen en ocasiones de este tipo de encuestas serológicas, un material difícil de evaluar. En el presente experimento, de desconoce si las parvadas sufrieron infecciones por otros adenovirus, además del BC-14, a diferencia de otros experimentos controlados en los que se han utilizado aves libres de anticuerpos contra adenovirus(2,4).

Debe considerarse que, el hecho de haber encontrado anticuerpos precipitantes en 406 de 1,100 sueros, aunado a la presencia de niveles de anticuerpos IH en 9 de 67 parvadas con 52 sueros positivos, en aves de razas semipesadas y pesadas que procedían de regiones avícolas delimitadas, hacen suponer que en la República Mexicana existen parvadas comerciales de gallinas que se han infectado con el virus causante del Síndrome de la Baja en Postura, u otros adenovirus relacionados antigénicamente con éste.
Se considera que el presente trabajo puede servir de pauta para futuras investigaciones, en las que deberán incluirse necesariamente el aislamiento y la identificación del agente causal, así como la reproducción del cuadro clínico patológico, de tal manera que pueda confirmarse la presencia de esta enfermedad en nuestro país.

CUADRO NUM. 1

RESULTADO DE LAS PRUEBAS DE INMUNODIFUSION EN AGAR

Tipo de Ave	No. de parvadas	Total de sueros	Sueros Positivos	Parvadas Positivas	%
Productoras de huevo café	8	111	48	6	43.2
Reproductoras pesadas	29	625	263	20	42.0
Productoras de huevo blanco	21	220	72	16	32.7
Reproductoras semipesadas	4	64	12	2	18.7
Reproductoras ligeras	5	80	11	3	13.7
T o t a l	67	1,100	406	47	

CUADRO NUM. 2

RESUMEN DE LOS RESULTADOS DE LAS
PRUEBAS DE INHIBICION DE LA HEMOAGLUTINACION.

Resultados	No.de Sueros.
Positivos	52
Título Mín. 1/40	36
Negativos	318
T o t a l	406

CUADRO NUM. 3

TITULOS DE LOS SUEROS CON ACTIVIDAD
INHIBIDORA DE LA HEMOAGLUTINACION.

Título de la inhibición	No. de sueros
1/320	6
1/160	14
1/80	20
1/40	12
T o t a l	52

CUADRO NUM. 4

RELACION ENTRE EL TIPO ZOOTECNICO DE AVE Y EL PROCENTAJE DE SUEROS POSITIVOS A LA PRUEBA DE LA HEMOAGLUTINACION.

Tipo de Ave	No.de sueros	Sueros Positivos	Parvadas positivas	%
Productoras de huevo café	43	8	2	16.6
Reproductoras pesadas	263	42	6	15.9
Productoras de huevo blanco	72	2	1	2.7
Reproductoras semipesadas	12	-	-	-
Reproductoras ligeras	11	-	-	-
T o t a l	406	52	9	

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EGG DROP SYNDROME (EDS'76): ETHIOPATOGENESIS, EPIDEMIOLOGY, IMMUNOLOGY AND CONTROL OF THE DISEASE

A. Zanella, A. Nigrelli, and G. Poli
Institute of Animal Infectious Diseases and Institue of Microbiology and Immunology
University of Milano--Research Laboratories
EUROBIO, Brescia, Italy

ABSTRACT

In 1976 a new condition, Egg Drop Syndrome (EDS'76), became widespread in West Europe causing very high economic loss. Isolation of an haemoagglutinating adenovirus and experimental reproduction of the syndrome with various isolates showed that this virus is the causative agent. This paper reports the results of three years' research into the etiology, pathology, immunology, and control of the disease.

Strain E-77, isolated in Italy, had biological, physico-chemical, and morphological characteristics of adenovirus, high pathogenicity and antigenicity, and serological identity with the strains 127, BC-14, 3877, isolated elsewhere. The virus grows well in duck-embryo fibroblast and in duck and chicken hepatocytes, with cytopathic effect (rounded, enlarged, refractile cells and nuclear inclusion bodies). By allantoic route, it kills duck-embryos in 7-10 days, whereas the chick-embryos, infected by yolk-sac, are stunted with poor hatchability. In one-day-old chicks, without maternal antibody, the virus multiplies inducing active antibody, whereas in those with maternal immunity it appears not to multiply, is not reisolated, and does not induce HI antibody; it may be latent until sexual maturity or lay.

Many avian species are susceptible, at least experimentally, with lateral infection spread in ducks, chickens, turkeys, with rapid and high antibody response; in geese, pheasants, guinea fowls, with slow and lower antibody response; in quail, without HI response. Differing egg production occurred in different chicken breeds.

A very efficient dead vaccine in oil emulsion has been prepared to control the disease; many millions of pullets have been treated. This permitted control of the disease and could be an approach to future eradication.

EL SINDROME DE LA BAJA DE POSTURA (SBP '76) ETIOPATOGENESIS, EPIDEMIOLOGIA, INMUNOLOGIA Y CONTROL DE LA ENFERMEDAD.

Resúmen.

En 1976, una nueva condición, el Síndrome de la Baja de Postura (SBP '76), se difundió en Europa Occidental causando grándes pérdidas económicas. El aislamiento de un adenovirus - hemoaglutinante y la reproducción experimental del síndrome con el aislamiento de varias cepas se demostró que éste virus es el agente causal. Se reportan los resultados de 3 años de investigación en la etiología, patología, inmunología y control de la enfermedad.
La cepa E-77 aislada en Italia posee características biológicas, de los adenovirus, y una similitud serológica y antigénica con las cepas 127, BC-14 y 3877. El virus crece adecuadamente en fibroblastos de embrión de pato y en hepatocitos de pato y de pollo, produciendo efectos Citopatogénicos (células redondeadas, engrosadas y refractiles con cuerpos de inclusión intranucleares). Mata por vía alantoidea a embriones de pato de 7 a 10 días así como 1 baja de incubabilidad en embriones de pollo inoculados por vía intra-saco vitelino. En pollitos de un día de edad carentes de anticuerpos maternos el virus se multiplica induciendo anticuerpos activos, mientras que en pollitos de la misma edad pero con inmunidad materna, el virus no se multiplica aparentemente, ni se puede reaislar y no produce anticuerpos I.H., y puede permanecer latente hasta la madurez sexual o la postura.
Muchas especies aviares son susceptibles, al menos experimentalmente por medio de una infección horizontal en patos, pollos y pavos con una rápida y alta respuesta de anticuerpos. En gansos, faisanes, gallinas de guinea con una respuesta lenta y baja de anticuerpos. En la codorniz no ocurre la respuesta de I.H. La producción de huevos se altera de manera variada en las diferentes razas de gallinas.
Una vacuna inactivada en vehículo oleoso ha sido preparada para controlar la enfermedad. - Varios millones de pollas han sido vacunadas . Esta permitiría el control de la enfermedad y podría ser considerado como un acercamiento para una futura erradicación de la enfermedad.

Traducción: Cortesía del Dr. Miguel A. Márquez R.

EGG DROP SYNDROME (EDS'76): ETHIOPATHOGENESIS, EPIDEMIOLOGY, IMMUNOLOGY AND CONTROL OF THE DISEASE

A. Zanella, A. Di Donato, A. Nigrelli and G. Poli
Institute of Animal Infectious Diseases-Institute of Microbiology and Immunology, University of Milano - Research Laboratories "EUROBIO", Brescia (Italy)

Introduction

In 1976 a new pathological condition, called "Egg Drop Syndrome '76" (EDS'76), became rapidly widespread in hen's farms throughout West Europe (Mc Ferran et al., 1977; Baxendale, 1978; Zanella, 1978; Picault, 1978); however it seems that a similar condition was apparently present in some parts of Netherlands for a few years (Van Eck et al., 1976). The condition was characterized almost exclusively by failure to reach predicted egg laying targets or, more frequently, by quite sudden and severe falls in egg laying (20-50%), together with high and long lasting production of egg with soft shell or without shell. The great scientific interest of the phenomenon, but overall the considerable economic importance of the condition, pushed to carry out wide and sudden researches into its ethiology, epidemiology and control. The isolation of haemoagglutinating adeno-like viruses, antigenically distinct from known fowl adenovirus (FAV)(McFerran et al., 1978), in various outbreaks of this syndrome and in different countries, the extensive serological investigations and the experimental reproduction of the disease with the various isolates (Mc Cracken & Mc Ferran, 1978; Baxendale et al., 1978; Zanella, 1978 pers. comm.), showed that EDS'76 is caused by this virus.

The purpose of the present paper is to report the results of our three years intensive research into the different and interesting aspects of the disease.

Ethiopathogenesis

During virological investigation on EDS'76 in Italy, a virus (strain E-77) was isolated from cloacal swabs of hens suffering from depressed egg production, with poor egg shell quality (Zanella, 1978). The virus was isolated in chick-embryo hepatocites after 3 blind passages. The strain E-77 showed serological features indistinguishable from strain 127 (Mc Ferran et al., 1978), BC14 (Baxendale, 1978) and 3877 (Picault, 1978), isolated in other countries; it was characterized by a very high pathogenicity and antigenicity. The biological, phisico-chemical and morphological characteristics permitted us to classify the isolate E-77 as an avian haemoagglutinating adenovirus. Electron microscopic examination of pelleted virions revealed particles with a clear hexagonal outline, typical of adenovirus, with a range in size between 70 and 80 nm. The virus showed to be stable over a wide range of pH, in monovalent but not in divalent cations, resistent to ether, chloroform and heat (50°C) treatment; inhibition tests by IUDR indicated the presence of DNA.

The virus grew well in duck- and chick-embryo hepatocytes and in duck-embryo fibroblast, with a clear cytopathic effect (rounded, enlarged, refractile cells and nuclear inclusion bodies). By allantoic route, it grew well in and killed duck-embryos in 7-10 days, with a very high haemoagglutinating (HA) titer of allantoic fluid, that could reach also 2^{18}. Whereas the chick-embryos, above all if infected by yolk-sac at 5-7 days, developed stuntly, with very poor and delayed hatchability; the HA titer of their allantoic fluid appeared absent or very low. Only a part of chicks, hatched from eggs inoculated with virus, developed haemoagglutination-inhibiting (HI) antibody within 10-14 days; also only a part of chicks, contemporarely hatched from not infected eggs and reared with the previous chicks, developed antibody within 14 days, beeing the remainder serologically negative for at least two months.

In experimental infections of one-day-old SPF chicks, by eye or cloacal route, the virus was reisolated from intestine and bursa of Fabricius in 8/8 birds after 7 days, but not after 14 days; no isolation at these times from respiratory tract, tymus, liver and blood leucocytes (!). All chicks developed HI antibody rather rapidly (7days), without symptons and mortality. On the

contrary,no virus reisolation and no development of active antibody occurred, at least apparently, in chicks with maternal antibody. In these last birds, the isolation of the virus and the development of HI antibody occurred gradually, starting from 27th week of age in a first trial and from 14th week of age in a second trial; strangely, two birds of the last trial excreted virus for a long time (at least from 23th to 31st week of age),without developing HI antibody and continuing to lay normally. In chicks with maternal antibody the virus might have been not fully neutralized, hide in some places of the body and rouse up during the stress of sexual maturity or of laying, as it seems generally to happen in the field.

Attemps to isolate virus from the last part of oviduct (uterus) of hens challenged during laying period succeded 3 and 7 days after (3/3 birds and 1/3 birds respectively),but not 10 and 14 days after challenge. Virus isolation from albumen and yolk of 20 eggs layed without shell in various trials of experimental infection, always failed. This means that, if the vertical transmission of infection occurs, it could be only for a very short time,probably in the first seven days after infection. Also Baxendale (1979) reported that chicks hatched from hens in the acute fase of EDS'76 failed to develop antibody and he was unable to isolate the virus from them.Test of virus isolation from eggs layed in the first 8-9 days after infection (when the egg shell is still normal but virus is present in the oviduct)are in course.

As regard the mechanism by which the virus could cause egg shell changes, it is not yet known.Histological examinations of oviducts from field cases showed no profound changes (Mc Cracken,pers.comm.)or oedema with moderate infiltration of plasmacells, lymphocytes and heterophils in the _lamina propria_ and decrease or disappearance of the secretory granules of the uterine epithelium (Asdrubali et al.,1978).The analysis of sera revealed only a decrease of alkaline phosphatases (Picault,1978),enzymes which intervene in the complex mechanisms of shell formation.Complete histological examination of oviduct and biochemical tests at subsequent steps after experimental infection are still in course in our laboratories. However the first observations of the uterus showed: at 3 days=normal tissue; at 7 days=inclusion bodies in epithelium of plicae; at 10 days=inclusion bodies in and necrosis of epithelium with phlogosis and arteritis; at 15 days=deep phlogosis and necrotic-proliferative arteritis (Mandelli and Zanella,pers.observ.).

Epidemiology

It is almost ascertained that origin of the agent of EDS'76 is from ducks for the following reasons:1)the _in vitro_ preference of the virus for duck-embryo and cells;2)the widespread of the infection (virus isolation and presence of specific antibody) in different breeds and in many flocks of ducks,also in age as early as 3-6 weeks,in different countries (Baxendale,1979;Zanella, pers.comm.);3)the limited lateral spread of infection in chicken;4)the very rare cases of serological positivity in chicken before laying period (Zanella,pers.comm.);5)the apparent first appearance of the disease in the Netherlands, a country well known for duck breeding (Van Eck et al.,1976);6)the presence of antibody in ducks of North America where the syndrome in chicken is unknown (Calnek,1978).

We carried out serological and receptivity tests in some avian species, also in order to determine the potential reservoirs and spreaders on infection.Besides in chicken, natural HI antibody has been found only in different breeds of ducks;Mandelli et al.(1978) evidenced antibody, very rarely and at very low titer (max.1:20),in sparrow living in chicken infected farms,which we are unable to detect,in spite of numerous tests done also in sparrows of duck infected farms. Receptivity to and transmission of infection by contact have been demonstrated by us, besides in different breeds of ducks and chicken, also in geese, pheasants,guinea fowls,turkeys and quails, with reisolation of virus and development of antibody at different degree and fastness; shedding of virus,but not HI antibody development have been demonstrated in quails,also one month after infection.

The susceptibility of the various chicken breeds to the disease resulted quite different.The brown egg layers seemed to be the most susceptible,as re

gards either the entity and the period of drop in laying or the egg shell quality;broiler breeders and White Leghorn (W.L.) followed in order.For some time, at least in our country, the W.L. hens appeared rather resistent to the disease. In fact in many farms or houses with brown and W.L.hens,the ones but not the others suffer of EDS'76; afterwards a few cases of syndrome have been observed also in W.L., but with losses comparatively lower (Zanella, pers.comm.).

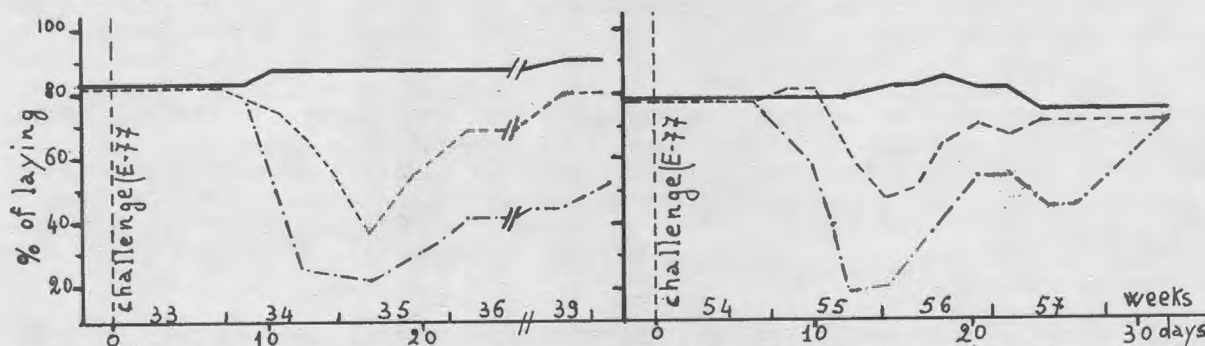
The fastness in the lateral spread of infection resulted higher on the litter than in cage; in this last type of rearing the spread seems to occur overall by contiguity.The most probable way of transmission of infection seems to be the digestive apparatus; on the contrary, the respiratory apparatus would seem to be less important, at least in regard to the transmission from house to house or from farm to farm.

Control of the disease by vaccination

For convenience of use, a bivalent vaccine against EDS'76 and Newcastle disease has been prepared.Two strains (E-77 and 127) of adenovirus,propagated firstly on tissue-culture of chick-embryo hepatocytes and, in a second time, of duck-embryo fibroblast and four higly immunogenic strains of Newcastle disease virus (NDV), propagated on 11 days embryonated eggs have been used. The HA and infecting titres of fluids with adenovirus have been not less than 2^{14} HA units and $10^{8,5}$ $TCID_{50}$/ml;the titres of fluids with NDV have been not less than 2^{10} HA units and $10^{9,5}$ EID_{50}/ml, respectively. After inactivation with betapropiolactone, the viral fluids have been mixed together, in equal parts, and then emulsified with 70% of Freund's incomplete adjuvant.

The vaccination has been done in pullets of different breeds at 16-20 weeks of age (0,5 ml/bird);the degree and lenght of immunity has been controlled and evaluated by the HI test and by challenge with adenovirus strain E-77 ($10^{6,5}$ $TCID_{50}$/bird), at different stages of laying.

The results of numerous experimental and field trials,carried out on different breeds,showed:1)high levels of HI antibody (2^7 to 2^{10})to EDS'76 and NDV,with a single dose of vaccine;2)complete protection of layers to the challenge against EDS'76, already ten days after vaccination and for at least one year of laying; 3)high levels of maternal antibody in the progeny.Tipical results of challenge against EDS'76 in vaccinated and unvaccinated hens are reported in graph 1-2.



Graph.1 - 2: rates of laying in vaccinated(——)and control(total - - - and normal—·—eggs)hens ,challenged at 32 and 53 weeks of age.

Many millions of doses of such vaccine have been used in field in West Europe during these last 2½ years and it performed excellently(sometime also in flocks vaccinated at the beginning of the syndrome),with no or very slight cases of break of immunity,that is pushing the farmers to insist in the use of this vaccine to control EDS'76. The vaccination would have also the advantage to remove or to reduce the incidental vertical transmission, as well as to prevent the lateral spread of virus not only into the same farm, but also from farm to farm in areas with highly intensive breeding.

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VACCINATION AGAINST EGG DROP SYNDROME (EDS) AND NEWCASTLE DISEASE WITH A BIVALENT INACTIVATED VACCINE IN OILY ADJUVANT

JF. BOUQUET, B. DEVAUX, D. GAUDRY, Y. MOREAU.
IFFA MERIEUX, 254 rue Marcel Mérieux 69007 LYON (FRANCE)

VACUNACION CONTRA EL SYNDROME DE LA BAJA DE POSTURA Y ENFERMEDAD DE NEWCASTLE CON UNA VACUNA INACTIVADA BIVALENTE CON ADJUVANTE OLEOSO

RESUMEN

Uno de la problemas más comúnes afectando la procduccion de huevo en reproductoras y ponedoras, son las fallas en la producción de huevo; que se manifiesta por una rápida baja en el numero de huevos producidos o parvadas que no alcanzan la produccion que se esperaba. Existe un gran numero de factores bien conocidos que puden causar baja en la produccion de huevo. Muchas de las posibles causas son conocidas, pero las mas importantes son enfermedades virales tales como bronquitis infecciosa, enfermedad e Newcastle, encefalomielitis aviar y adenovirus.

Recientemente en Europa un nuevo syndrome se ha diseminado y se considera ahora que es uno de las problemas mas importantes en la endustria avicola causando grandes pérdidas económicas en reproductoras y ponedoras comerciales.

Esta enfermedad llamada syndrome de la baja de postura (Egg Drop Syndrome) es causada por un adenovirus el cual ocasiona que se produscan huevos con cascaron debil y huevos sin cascaron y aparente bajas en la produccion.

Este adenovirus fue aislado por MacFerran que le nombro virus 127 y por Baxendale llamandole BC 14; que a differencia de otros adenovirus aislados de gallinas, este aglutina erytrocitos aviares y manifiesta una comparticion parcial de una antigeno especifico común a los adenovirus aviares.

Una vacuna inactivada preparada con virus 127 en un adjuvante oleoso, induce protección contra exposiciones en la laboratorio con el virus pategénico 127; esta vacuna se combinó con la vacuna inactivada contra el Newcastle y ha dado resultados muy satisfactorios en las ponedoras comerciales y reproductoras desde hace muchos años.

One of the commonest problems affecting the laying production of parent stock and layers is failures in production manifesting either by a sudden fall in egg numbers or a lack to reach the expected production. There are a large number of well known factors which can cause depression of egg production. Many of the possible cause are known but the most important are viral diseases in which infectious bronchitis, Newcastle disease, avian encephalomyelitis and adenoviruses.

Recently, in Europe, a syndrome has become widespread and it is now considered to be one of the most important problems in poultry industry causing high economical losses in breeders and layers.

This disease named Egg Drop Syndrome (EDS) is caused by an adenovirus which is caracterised by inducing a production of soft shelled and shell-less eggs and apparent drops in production.

This adenovirus isolated by Mac FERRAN registered on 127 virus and by BAXENDALE with BC 14 isòlate, unlike other adenoviruses isolated from chickens, agglutinates fowl erythrocytes, and shows partial sharing of a specific antigen common to fowl adenovirus.

A vaccine prepared with 127 virus inactivated in an oil adjuvant induce a protection against a severe laboratory challenge with the pathogenic 127 virus.

After these results this vaccine was combined with the Newcastle inactivated vaccine that gave satisfactory results on the commercial layers and breeders since many years.

The present communication describes the bivalent vaccine and shows the laboratory results of safety and potency tests and the field results of long lasting immunity by challenging a group of birds at different time in laboratory conditions.

1. Vaccine

127 virus : Strain 127 was obtained from Dr JB. Mac FERRAN (Vet. Research Laboratory Stormont, Belfast, N. Ireland) and propagated on chicken embryo liver cells (CELIC). The strain was then adapted on Moscovy duck embryofibroblasts (MDEF).

Newcastle disease virus : A velogenic strain of Newcastle disease (ND) was propagated on embryonnated eggs (EE) and the amnio allantoïc fluid was harvested.

Inactivation : Both virus were inactivated by bêta-propiolactone (BPL) for which the action was combined with temperature.

Antigen test : Inactivation of the antigens was tested by two subcultures on the homologous cell system. Two passages of the 127 antigen on MDEF and two passages of the ND antigen on EE during a 14 days period. After this period, any suspicion of live virus must not exist. Both antigens were tested for bacteriological and fungi contamination.

Emulsification : After these tests were satisfactory, the antigens were blended with a sterile liquid paraffin with sorbitan monooleate and polysorbate 80 to obtain a stable emulsion. Each batch of bivalent inactivated vaccine was tested for bacterial and fungi sterility safety and potency tests.

Safety test : This test consisted by inoculation of 1 dose by IM route on 10 three-week chickens, after 21 days observation period, all chickens must be healthy.

Potency test : 127 activity was appreciated by GMT of HI antibody values, 21 days after vaccination. Newcastle disease activity was tested after challenge of four groups of 10 birds whichreceived 20, 10, 5 or 2,5 µl vaccine (which represented 1/25, 1/50, 1/100 and 1/200 vaccine dose) 21 days before. With the protection results it was possible to determine PD 50 with Probits method.

2. Laboratory results

a) Toxicity (Growth rate)

The safety test was examined with a special test of toxicity on day-old chickens.

10 day-old chickens received 0,5 ml par IM route and 10 day-old chickens of the same origin were kept as controls. Each bird was weight every day during 15 days. For each day a geometric mean for both group was compared.

The observation of the growth weight curve showed no significant difference between the vaccinated group and controls.

b) Long lasting immunity

Conventional White Legorhn layers were obtained form commercial sources. These birds were maintained under field conditions during experiment.

Humoral immunity was tested by inhibition of hemagglutination test (HI) and seroneutralisation test (SN) every three weeks on 20 birds.

The HI test was conducted by conventional microtiter method. 127 antigen consisted of inactivated cell-culture supernatant from infected MDEF. Microplates used were from Linbro Chemicals. A 0,050 ml serum volume was diluted in two-fold serial dilution in the antigens volume containing 4 HA units per well. The neutralization time was conducted at room temperature during 30 mn.

Then adding 0,025 ml of 1 % young chickens red blood cells suspension to the dilutions. A positive serum control consisted of known monospecific serum with an HI titer of 640 was titrated in each test to compare the results.

The SN test was used by beta method with constant virus and serum dilution. Serum dilutions were obtained in microculture plates by using 0,025 ml diluters. Each serum was placed into 6 replicate wells and diluted in four-fold dilution in 0,075 ml containing 100 TCID 50. Plates were covered with a clear polystyrene top and incubated at 37°C for one hour. The incubation period used in the test was determined by preliminary trials to give maximal sensitivity in the shortest period of time. After incubation, cells were added on 0,15 ml volum containing 3.10^4 MDEF in F 10 - 199 with 2 % calf serum. Microculture plates were read 7 days post incubation. Serum titer was determined by Karber method.

For EDS response after vaccination, SN antibodies response was higher than HI antibodies. This result was routinely observed and can explain some poor HI antibodies responses observed on the field with a good protection simultaneously.

3. Fields results

a) Experiment 1

A conventional layers flock was vaccinated with EDS + Newcastle vaccine on the field at 18 weeks of age excepted a little part wich received the Newcastle vaccine only.

After a period of 8 weeks of vaccination, 20 layers vaccinated with bivalent vaccine and 20 layers vaccinated with Newcastle vaccine were transfered in animals facilities for EDS challenge.

The EDS challenge was performed by administration of $10^{3,7}$ TCID 50/bird of the pathogenic 127 strain by intratracheal and oral route.

b) Experiment 2

Conventional Brown Legorhn layers flock was divided in two parts. On half of the flock received the bivalent vaccine at eighteen weeks of age and the other part was not vaccinated.

20 birds of each part were transfered into animals facilities, 17 weeks and 38 weeks after vaccination.

Protection of vaccinated birds was tested by HI antibodies values and by EDS challenge in the same way that the experiment 1.

It was possible to produce experimental disease of EDS in laboratory. Layers are very affected in the earlier time of laying period but at 56 weeks-old layers gave an egg drop too.

The most interesting results was obtained with normal egg production (egg production without soft shell egg and shell less egg representing commercializable eggs). The difference between vaccinated and non vaccinated group on curve was 35 %, 60 % and 15 % for challenge at 8, 17 and 36 weeks after vaccination. For the second experiment, vaccinated group did not produce any anormal egg.

These results were obtained with only one vaccination at 18 weeks of age on birds without antibody against EDS. The association of this vaccination with Newcastle disease vaccine did not induce any disturbance in Newcastle vaccination as reported by antibodies values.

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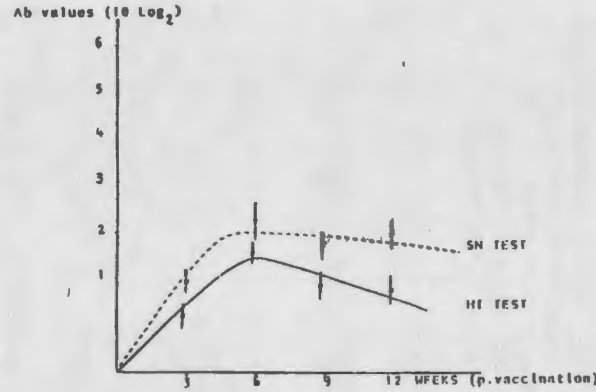
See next page for figures.

RESULTS OF POTENCY TESTS NEWCASTLE + EDS VACCINE

BATCH	POTENCY					
	EDS HI (GMT)	NEWCASTLE				
		20	10	5	2,5	PD_{50}
9 OSA 01	320	10	·10	9	9	> 200
9 OTA 01	680	9	10	10	7	> 200
9 OUA 01	100	7	8	3	3	142
9 OVA 01	170	10	9	9	7	> 200
9 OWA 01	680	10	10	8	10	> 200
9 OXA 01	520	9	10	10	10	> 200
0 OY 01	520	10	10	10	8	> 200
0 OZ 01	450	9	9	10	8	> 200
0 OA 01	480	10	10	10	8	> 200
0 OB 01	320	10	10	9	10	> 200

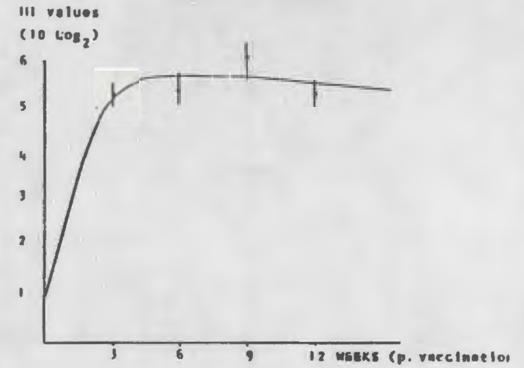
ANTIBODIES RESPONSE AGAINST EDS

AFTER NEWCASTLE + EDS VACCINATION



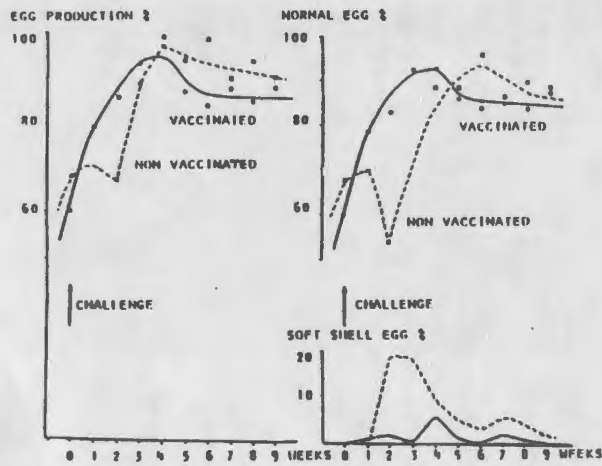
ANTIBODIES RESPONSE AGAINST NEWCASTLE

AFTER NEWCASTLE + EDS VACCINATION



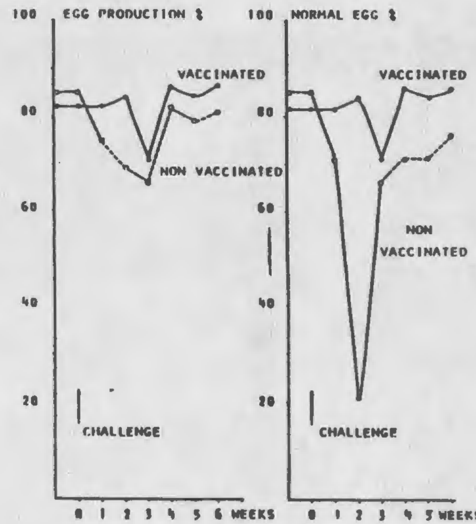
EGG PRODUCTION OF LAYERS CHALLENGED

8 WEEKS AFTER VACCINATION



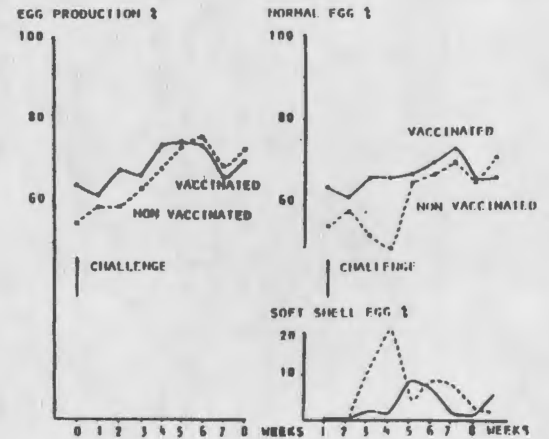
EGG PRODUCTION OF LAYERS CHALLENGED

17 WEEKS AFTER VACCINATION



EGG PRODUCTION OF LAYERS CHALLENGED

36 WEEKS AFTER VACCINATION



RECENT RESEARCH ON EGG DROP SYNDROME '76 (EDS '76)

WILLIAM BAXENDALE
INTERVET LABORATORIES LTD.
THE ELMS
HOUGHTON, HUNTINGDON
CAMBRIDGE PE 17 2 BQ, INGLATERRA

SUMMARY

EDS '76 has become widespread in the fowl population and it is a cause of considerable economic loss. Eradication of the disease may be possible from the breeding and elite stock as vertical transmission is an important means of the spread of the virus. Screening individual birds for antibodies and removing them before lay may eliminate carriers. Where resources are limited, vaccination with an inactivated vaccine that induces high antibody levels may be attempted. The results reported here would indicate that it is likely that both vertical and horizontal infection are reduced.

Of interest is the recent observation (Baxendale, unpublished) that vaccination of chicks previously experimentally infected as 1 day old embryos and which had developed only low Hl antibody, titres resulted in a considerable boost in antibody titre. If such birds were potential carriers, an antibody boost may reduce the chance of these birds excreting virus.

INVESTIGACIONES RECIENTES SOBRE EL SINDROME DE LA BAJA DE POSTURA '76 (EDS '76)

RESUMEN

El Síndrome de la Baja de Postura '76 se haya hoy día distribuido ampliamente en la población avícola, causando pérdidas económicas importantes. La erradicación de la enfermedad puede ser posible actuando sobre las parvadas de progenitoras y reproductoras puesto que la transmisión vertical del virus es al parecer el medio más importante de difusión de la enfermedad.

La estrategia de verificar serológicamente a las aves en búsqueda de anticuerpos que denuncien la presencia de la enfermedad con el objeto de eliminar a las portadoras, es un método limitado por su alto costo. La vacunación con una vacuna inactivada inductora de altos niveles de anticuerpos puede ser la opción a tomar. Los resultados reportados en este trabajo indican que, tanto la transmisión vertical como la infección horizontal fueron reducidas.

Es altamente significativa la reciente observación (Baxendale, en prensa) de que la vacunación de pollitos previamente infectados cuando eran embriones de 1 día de edad y que habían desarrollado sólamente un bajo nivel de anticuerpos Hl, dichos anticuerpos actuaron aumentando considerablemente los títulos de los anticuerpos vacunales. Si dichas aves eran portadoras potenciales, un aumento de los anticuerpos, podría reducir las posibilidades de que estas aves excretaran virus.

RECENT RESEARCH ON EGG DROP SYNDROME '76 (EDS '76)

W. Baxendale, Intervet Laboratories Ltd., Houghton, United Kingdom

INTRODUCTION

The Egg Drop Syndrome '76 (EDS '76) was first observed three years ago and first described by Van Eck *et al.* (1976). Since that time the disease has been observed in most of the Western European countries as well as Hungary, Yugoslavia, Central and South America. The disease is typified by a drop in production, the laying of soft-shelled eggs and in the case of brown egg layers, there is a loss of shell color. Often there is a reduction in food intake and a transcient diarrhea.

The disease is caused by an adenovirus (EDS virus) that is serologically distinct from the fowl adenvirus (Baxendale 1978; McFerran *et al.* 1978). The virus has been shown to be widespread in both domestic and wild ducks and it has been suggested that it was introduced into the fowl population by the use of a vaccine grown on duck tissue that was contaminated with EDS virus.

An effective inactivated vaccine against the condition was developed in 1977 (Baxendale 1977) and this has been commercially available for two years now.

The detailed epidemiology of the disease is not yet clear. However, certain facts have emerged and strong circumstantial evidence exists and these are sufficient to give some indication of the epidemiology. It is this aspect as well as the possible approaches to eradicate the disease that are the subject of this report.

EPIDEMIOLOGY IN DUCKS

As mentioned earlier the EDS virus probably is of duck origin and in this species the epidemiology would appear similar to that of the fowl adenovirus in the fowl. The virus spreads readily from duck to duck and is easily isolated from young ducklings (Baxendale 1977). It is not known if it causes any disease condition in this species.

Small scale experiments by ourselves would indicate that the EDS virus is unlikely to spread from ducks to chickens unless the two species were kept in close contact. As this is unlikely under normal management especially in elite and breeding stock it is probable that if the virus were eradicated from the fowl population, then the duck population would not reinfect them.

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EPIDEMIOLOGY IN FOWLS

In the suggested epidemiology of the EDS virus in the fowl (Fig. 1) the carrier bird and vertical transmission play an important part in perpetuating the disease. The carrier bird, possibly infected through the gg, usually does not infect the flock until the critical time of peak egg production. The number of carrier birds may not be great (less than 1%) and work by Schloer *et al.* would suggest that they may not always infect the rest of the flock. The same workers also showed such birds could have low levels of antibody. In an attempt to experimentally reproduce the carrier state we infected embryonated eggs with virus and showed that the chicks that hatched often had low hemagglutinating inhibiting (HI) antibody titers (Fig. 2).

The first isolates of EDS virus (BC14 and 127 strain) spread poorly and often did not spread to fowl in an adjacent house. Reports from the field would suggest that the virus is now spreading more rapidly. However, there is no firm data published on this aspect. Assuming that the infectivity of the virus has not changed radically and the suggested epidemiology is correct, then vertical transmission is the most important means of perpetuating the virus in the fowl population. It would, therefore, appear possible to eradicate the virus by removing the carrier or by stopping them infecting the rest of the flock. The two methods that are available are:

1. Detect carriers and remove them.
2. Vaccinate to reduce vertical and horizontal infection.

It is necessary to consider these in detail.

1. Detection of Carriers and Removal of Them.

The only possibly reliable method available for detecting carriers depends upon the assumption that infected birds respond with antibody and hence the removal of such birds results in removal of carriers. If, however, truly immunologically tolerant birds exist or the levels of antibody are below the threshold of sensitivity of the antibody test

Figure 1. Suggested Epidemiology of EDS virus in the Fowl

Infected laying flock lay low % of infected eggs for 8-10 weeks -- then the numbers of infected eggs greatly reduced.

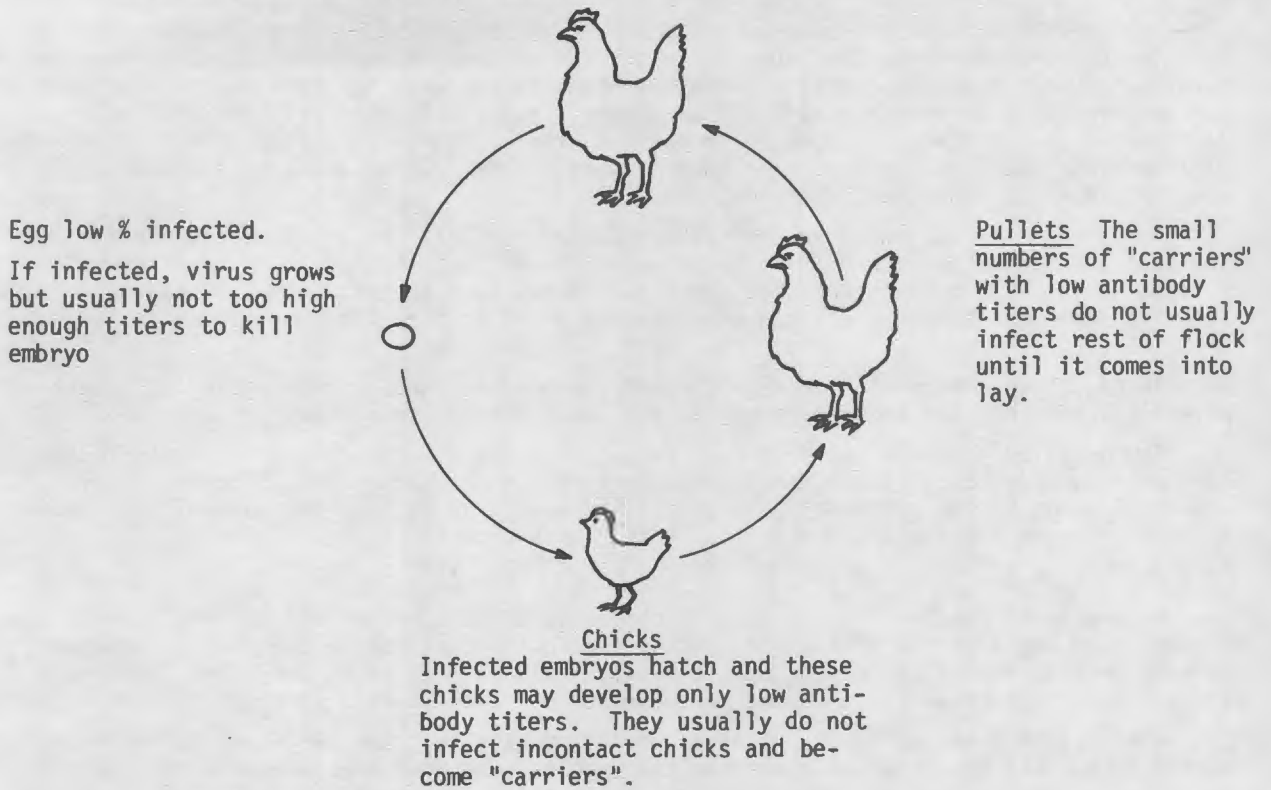
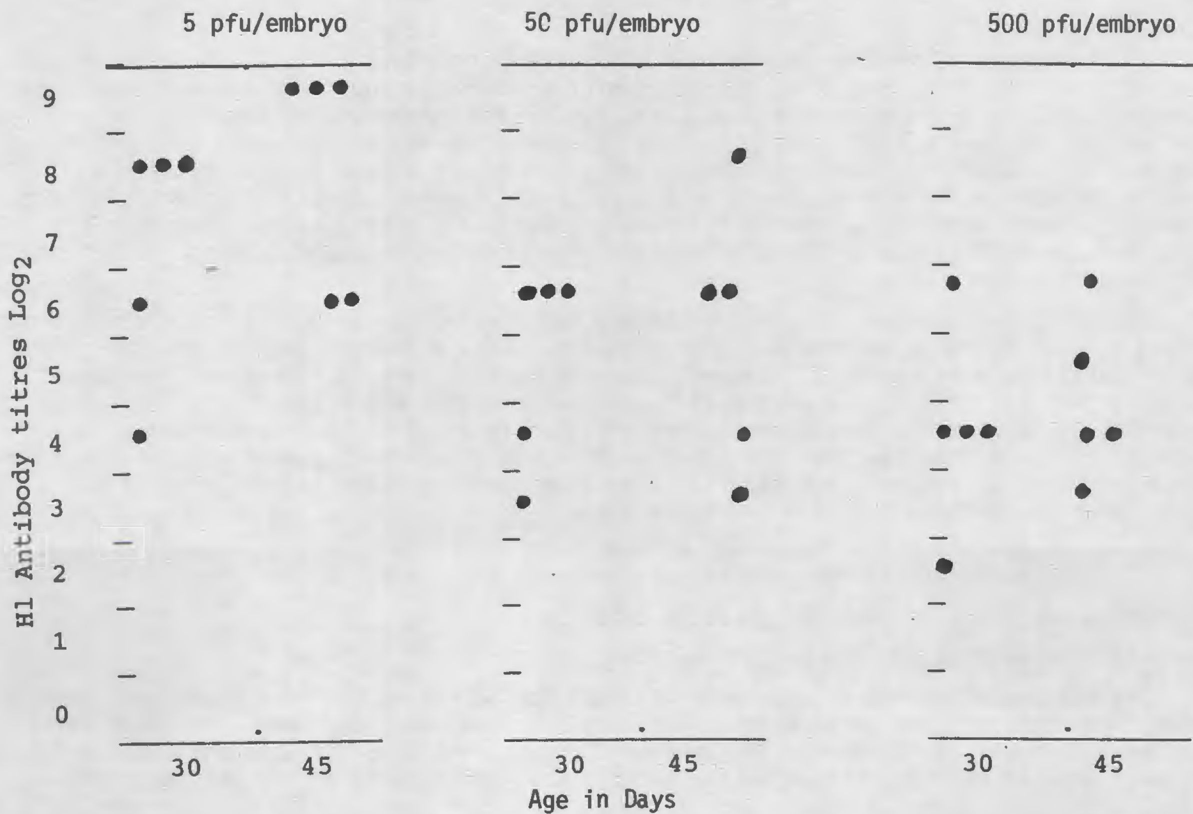


Figure 2. Antibody titers of SPF chicks infected as 9-day-old embryos with various doses of EDS virus (BC14 strain).



employed, this method is unreliable.

It is suggested that if this is to be attempted then all the birds within a flock should be tested by the HI test for antibody to EDS before coming into lay, and also that the sensitive plaque reduction test could be used (Baxendale 1979) to confirm whether sera that were suspect in the HI test were truly negative or positive.

2. Vaccine to Reduce Vertical and Horizontal Infection

The inactivated oil adjuvant vaccine is effective in protecting against the signs of the disease. However, to be of value in the eradication of the virus, it would have to reduce the amount of virus excreted by the birds as well as reduce or stop egg transmission The evidence for this is still not great and in some cases comes only from laboratory experiments.

EVIDENCE FOR VACCINATION REDUCING VIRUS EXCRETION

A. Laboratory Data. In a trial a group of 18 x 20-week-old layers were vaccinated and placed in a pen adjacent to 15 unvaccinated hens. At 27 weeks of age both groups of birds were challenged with EDS virus by intranasal installation--at a dose rate of 10^6 pfu/bird. The non-vaccinated hens developed typical EDS '76 with a drop in egg production and loss of egg shell color, whereas the vaccinated birds were immune. Blood samples, buffy coat samples and vent swabs were taken from all chickens at intervals throughout the trial and tested for antibody and virus.

In the vaccinated group no boost in vaccinal antibody was observed following challenge and no virus was isolated from vent swabs and buffy coat samples taken at weekly intervals for four weeks. In the unvaccinated group, virus was isolated from one of 15 buffy coat samples taken at seven days post-infection and four out of 15 at 14 days and from two of 15 vent swabs taken 14 days post-infection. These results show clearly that there is a reduction in virus excretion in the case of vaccinated birds. The fact that no boost in vaccinal antibodies was observed following challenge may indicate that the birds were partially or completely immune to infection with the dose of virus used.

B. Field Data. The observation has been made that where a vaccine that induces good antibody responses is used there is no boost in vaccinal antibodies despite the vaccinated birds being kept in pens adjacent to unvaccinated hens showing severe EDS '76 (Baxendale *et al.* 1979 in press) again possibly indicating that vaccinated birds show a measure of immunity to infection (Table 1).

Attempts to isolate the EDS virus from vaccinated birds in the field have only been successful once despite numerous attempts. On this successful occasion only one vent swab out of 20 proved positive and the virus was of low titer. However, as it was not possible to establish that the vaccinated birds were challenged in all cases, the significance on this observation is doubtful.

IMMUNITY INDUCED BY VACCINAL MATERNAL ANTIBODY IN THE EMBRYONATED EGG

If antibody in the egg increases the dose of EDS virus necessary to infect it and hence the chick, then it may reduce the level of vertical transmission.

In an experiment designed to observe this effect, 9-day-old embryonated eggs from SPF and vaccinated hens were infected allantoically with different doses of EDS virus (BC14). The chicks were allowed to hatch and then placed in isolators where they were tested for virus and antibodies (Table 2). It was clear from these results that vaccinal maternal antibody induced a significant level of immunity. A dose in excess of 50 pfu was necessary to infect the embryos with maternal antibodies whereas a dose of 5 pfu was sufficient to infect the SPF embryos.

IMMUNITY INDUCED BY VACCINAL MATERNAL ANTIBODIES IN YOUNG CHICKS

If, as would appear to be the case, young chicks that are infected via the egg excrete virus until they develop antibodies post-hatch, then it is important that they do not infect incontacts for the first few weeks of life.

In an attempt to observe the immunity induced by maternal antibodies groups of maternally immune and SPF day-old chicks were infected with different doses of virus intraoccularly and intranasally. The various groups of birds were kept in isolation until 10 weeks of age during which time they were tested for virus and antibodies. (Table 3)

The results show clearly that a dose in excess of 500 pfu was necessary to infect the maternally immune chicks and a dose of 250 was sufficient to infect the chicks without antibodies.

In a further similar experiment where the birds were two weeks old when challenged

with 100 pfu of EDS virus (unpublished data) 24 of 24 maternal immune birds were not infected, whereas 8 of 8 SPF birds all developed antibodies.

These experiments indicate that maternal antibody increases the does necessary to infect the chick.

DISCUSSION

EDS '76 has become widespread in the fowl population and it is a cause of considerable economic loss. Eradication of the disease may be possible from the breeding and elite stock as vertical transmission is an important means of the spread of the virus. Screening individual birds for antibodies and removing them before lay may eliminate carriers. Where resources are limited, vaccination with an inactivated vaccine that induces high antibody levels may be attempted. The results reported here would indicate that it is likely that both vertical and horizontal infection are reduced.

Of interest is the recent observation (Baxendale, unpublished) that vaccination of chicks previously experimentally infected as one-day-old embryos and which had developed only low HI antibody titers, resulted in a considerable boost in antibody titer. If such birds were potential carriers, an antibody boost may reduce the chance of these birds excreting virus.

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Table 1. Effect on Vaccinal antibody titers where a poor vaccine and good vaccine were used in field trials following natural EDS challenge (unvaccinated birds in the same house developed EDS'76).

No. of Trials	Mean HI Titre Induced by Vaccination	Mean HI Titer Following Challenge
	LOG_2	LOG_2
3	4	9
6	5	9

Table 2. Results of antibody test and virus isolation attempts made on samples taken from chicks with and without vaccinal maternal antibody that have previously been infected as 9-day-old embryos with various doses of EDS virus.

	CHICKS WITH MATERNAL ANTIBODY		SPF CHICKS	
dose pfu/embryo	virus * isolation	Hl + antibody	virus isolation	Hl antibody
5000	3/12 *	7/11	N D	N D
500	0/11	3/11	6/9	5/5
50	0/10	0/10	4/6	5/5
5	0/10	0/10	1/8	5/5

*Virus Isolation -- Vent swabs taken at day-old.
Vent swabs taken at 6 weeks negative for all groups

+HI antibody -- Sera taken 20 and 45 days

*3/12 No. positive / No. in group

Table 3. Results of virus isolation attempts and antibody tests conducted on chicks with and without vaccinal maternal antibodies and infected with different doses of EDS virus (BC14).

	SPF CHICKS		MATERNAL ANTIBODY POSITIVE CHICKS	
Dose in pfu/chick	Virus isolation	HI antibody*	Virus* isolation	HI antibody+
25,000	N D		4/25	10/24
2,500	N D		4/25	10/25
500	N D		0/25	0/25
250	4/24	24/25	0/24	0/24

*Virus isolation == Vent swabs and buffy coat samples taken at 1,2,4 and 10 weeks of age
+Ab Test == Sera tested at 10 and 12 weeks of age

FURTHER INVESTIGATIONS ON THE EGG DROP SYNDROME

M. S. McNulty and J. B. McFerran, Veterinary Research Laboratories
Stormont, Belfast, BT4 3SD, N. Ireland

This condition, first described in 1976, is characterised either by a sudden increase in the production of thin shelled, soft shelled or shell-less eggs or by a failure to achieve predicted production levels. In either case the birds remain apparently healthy.

The aetiological agent is a duck adenovirus. This virus only spreads from infected ducks to fowl if they are in very close contact. The spread appears to be due to contact with faeces and aerosol spread is minimal.

In Northern Ireland the disease was eradicated from an infected basic breeding organisation. Eradication was based on the following premises:

a) Birds infected through the embryo quite often failed to develop detectable antibody.

b) These infected birds would show EDS around peak production. Following EDS antibody was detectable.

c) Birds over 40 weeks of age, even if infected, were unlikely to excrete virus.

d) The viruses infecting fowl in 1976-1977 at least had poor lateral spreading ability.

Therefore using these findings, chicks were hatched from flocks over 40 weeks of age. They were segregated from infected birds in the hatchery and were reared in semi isolation. These flocks were tested by HI at regular intervals. If a large number of reactors were found the flock was discarded. If only a few reactors were found, these were removed. After 40 weeks, if the flock passed a 100% HI test, eggs for breeding were then collected.

INVESTIGACIONES RECIENTES SOBRE EL SINDROME DE LA BAJA DE POSTURA (SBP)

Esta enfermedad, reportada por primera vez en 1976,tiene como característi--cas clínicas la repentina aparición de numerosos huevos con cascarón delgado o carente de cascarón,o bien por una incapacidad de la parvada para alcanzar el máximo de producción esperado.En ambos casos ,la parvada se nota clínicamente sana.
El agente causal es un adenovirus de los patos, el cual solo se disemina cuando están en estrecho contacto con gallinas. La principal vía de infección es a través de las heces y en un grado menor, aerógena.
La enfermedad fue erradicada de una granja de reproductoras situada en Irlanda del Norte, siguiendo el programa descrito a continuación:
a).- las aves infectadas en forma vertical, en numerosas ocasiones fueron negativas serológicamente.
b).- estas aves mostraban el SBP cuando llegaban el máximo de producción;posteriormente, se hacián positivas a la prueba serológica.
c).- aves mayores de 40 semanas difícilmente excretaban el virus.
d).- los virus que afectaron parvadas entre 1976-1977 mostraban baja transmisión horizontal.
Por lo tanto, y considerando las anteriores observaciones, se procedió a incubar huevos procedentes de gallinas mayores de 40 semanas de edad. Se incubaron en forma separada siendo su crianza en semi aislamiento. Se corrieron pruebas de IH a intervalos regulares. Se eliminaba la parvada solamente si - el porcentaje de anticuerpos era elevado. Si la incidencia era baja, se eliminaban únicamente las aves positivas. Si después de 40 semanas, la parvada era negativa en un 100% a la prueba de IH, entonces se procedía a incubar el huevo para reproducción.

Traducción: Cortesía del Dr. Armando Antillón Rionda.

ADENOVIRUS INFECTION AND "EDS-76"

J.B. McFerran, Ph.D. B.Sc. M.R.C.V.S.
Department of Agriculture, Northern Ireland,
Veterinary Research Laboratories, Stormont,
Belfast, BT4 3SD, Northern Ireland

It is not possible to review adenovirus infections within the time allowed. Therefore I am going to concentrate on the more practical aspects of adenovirus research with special emphasis on work done at Stormont.

All known chick adenoviruses share a common group antigen. This can be detected by the double immunodiffusion (agar gel precipitation) or the immunofluorescence test. Therefore the agar gel test will indicate only that the birds have been infected by an adenovirus. If one wishes to determine which adenovirus then the serum neutralisation test must be used. This test is expensive and time consuming. Not only does one require to use cell cultures, but also there are 12 different serotypes recognised. In fact there are more, as the breakdown of adenoviruses isolated at Stormont in 1978 shows

Adenovirus serotypes isolated in 1978

Serotype	F1	F4	F5	F8	F12	F2, 3, 6, 7, 9, 10	Broad Antigenicity	Not typeable
Number of isolates	15	3	16	31	25	8	18	38

Thus 38 isolates were not neutralised by the prototype serums. Some of these are probably mixtures of 2 or more recognised serotypes, but others are undoubtably new serotypes. A second point is that F1 (ie Celo) is not the commonest isolate, even though in 1978 it was much more frequently isolated than normal.

When a bird is infected with an adenovirus it excretes virus, mainly in the faeces, for about 3 weeks. The gut or respiratory tract is then resistant to reinfection with the same serotype for about 9 weeks or so, but is not resistant to infection with a different serotype. If a laying hen is infected, she will produce eggs infected with virus and after about 6 days the yolk sac will contain antibody. Chicks hatching from these eggs will have maternal immunity, which of course decreases and is finally lost usually between 2 and 4 weeks of age. At this stage, latent virus in the chick is unmasked, the chick excretes virus and infects surrounding susceptible chicks. As in most units the chicks are coming from a number of parent farms, each with its own flora of viruses, considerable mixing occurs. Therefore a number of serotypes can be isolated from a flock and even from the same bird.

Under modern poultry husbandry adenoviruses are ubiquitous and they are often isolated from healthy birds. It is entirely wrong to conclude that if diseases birds are shown to have rising titres to adenoviruses or if adenoviruses are isolated that the adenoviruses are the cause. They may be the cause or they may just be part of the normal flora of the bird at that time.

Adenoviruses certainly have been associated with disease - eg hepatitis, respiratory disease, arthritis, falls in egg production, poor food conversion, etc. In my opinion this association is probably correct for a number of conditions. But except in a few cases the adenovirus by itself is not responsible for the pathological condition. It must work in association with some other factor.

For example adenoviruses have been associated with inclusion body hepatitis. In our hands we cannot reproduce the disease in theage of bird seen naturally affected, and even to produce a hepatitis we have to use an abnormai route of infection. Furthermore the liver cells in IBH contain eosinophilic inclusions which on electron microscopic examination show degenerating nuclei. The experimental cases have basophilic nuclei, which are full of virus particles. Again, not one, but all known serotypes of chick adenovirus have been associated with IBH and the same serotypes can be isolated from healthy birds.

A second example is the association of adenoviruses with respiratory disease. About 12 years ago we had a very severe outbreak of respiratory disease. From our studies at that time it seemed the main causes were infectious bronchitis (IB) and adenoviruses. Whilst in normal birds adenoviruses are normally confined to the upper respiratory tract and gut, in the affected birds adenoviruses were isolated in very high titres from the lower respiratory tract. However once the IB was controlled by vaccination, the adeno-

virus problem was also resolved. Similarly at present we again have respiratory problems and again find adenoviruses associated with them. But we are undergoing a severe attack of infectious bursal disease and I expect if we can control it, we will control the adenoviruses.

One clinical condition does appear to be due to an adenovirus. This is the Egg Drop Syndrome 1976 (EDS 76). It is generally accepted this is due to a duck adenovirus. The virus grows best in duck cells or duck embryonated eggs. Studies in many countries have shown that whilst fowl serum prior to 1976 did not have antibody to EDS, both wild and domestic duck sera have antibody. It is probably that it was introduced into fowl by a contaminated vaccine. Subsequent effects depended on the level of introduction. If into parent or commercial stocks the virus is quickly eliminated, whilst if into basic breeding stock the disease becomes endemic because the virus is passed through the egg.

There are no signs of disease in birds affected with EDS, except possibly a transient diarrhoea. The main effect is that the birds either fail to achieve peak production levels or else show a marked fall in production. These effects in broiler breeders are seen between 25 and 35 weeks of age, although all ages will show the syndrome if infected. The egg sheel changes are loss of colour, thinning of the shell, soft shelled and shell-less eggs. If affected eggs are removed there is no effect on fertility or hatchability.

Chickens hatched from infected eggs may develop antibody, but in many cases fail to do so. At around point of lay, the carrier birds break down and excrete virus, infecting the incontacts. Where some chicks have developed antibody there is a failure to peak whereas if no antibody has developed the fall in production is seen. Egg transmission is maximal during the period of maximal signs and normally egg transmission does not occur to any extent in birds over 36 weeks of age.

Lateral spread of virus between flocks is poor. This can be confirmed experimentally when it can be shown that even a netting wire fence will prevent spread. Spread is however more efficient between ducks. Experimentally there appears to be no breed resistance to infection, and in nature most breeds have been infected, even those not exposed to potentially contaminated vaccine. This might be due to the virus becoming better adpated to fowl, but his adaptation has not yet been shown. However virus can be transferred by needles either used for bleeding or vaccination.

If there are infected birds, then a few rules must be observed:-

a) Do not use the same apparatus (or at least the needle) to vaccinate clean and infected stock.

b) Do not allow bird movement between clean and infected (including vaccinated) stock.

c) Must clean and disinfect houses, transport and boxes between infected and clean stock.

d) Do not hatch clean and infected stock in same hatchery. If unavoidable, use separate hatchers and hatch on different days. At very minimum take off, vaccinate and dispatch clean stock before the infected stock is touched.

Although a vaccine is available, we have gone for eradication and this has been successful. It is based on:-

1. Hatching from infected flocks when over 40 weeks of age.

2. Rearing the progeny and testing at 10, 20 and 30 weeks of age. If still negative after a 100% test at 40 weeks, hatching from these flocks.

In the event of a few reactors developing, these were removed and the other birds in the pen were subjected to two 100% tests 4 weeks apart. If reactors continued to develop, or a significant number of birds had antibody, the flock was eliminated.

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AISLAMIENTO DE *Arizona hinshawii* EN POLLO DE ENGORDA, REPRODUCTORES, DETRITUS DE INCUBACION Y MATERIAS PRIMAS PARA LA ELABORACION DE ALIMENTOS DE AVES EN MEXICO.

Gloria Oralia Pacheco: Departamento de Producción Animal:Aves Facultad de Medicina Veterinaria y Zootecnia. Universidad Nacional Autónoma de México. Cd.Universitaria, 20 D.F.

Durante el año de 1979 se remitieron al Departamento de Producción Animal: - Aves, animales jóvenes y adultos clínicamente sanos o con signos similares a la enfermedad de Onfalitis o Salmonelosis. También se recibieron muestras de harina de carne, harina de hueso y detritus de incubación, los cuales procedían de los estados de Guanajuato, Morelos, Veracruz, Yucatán y Estado de -- México.

Al realizar el estudio postmortem se observó que las lesiones más notables - estaban localizadas en el hígado (hepatomegalia, friable y color bronceado).

Se procedió a analizar bacteriológicamente los órganos afectados obteniéndose el aislamiento de Arizona hinshawii en 15 casos clínicos.

Las muestras de harinas y detritus de incubación se analizaron de acuerdo a las técnicas de Gentry y de la Food and Drug Administration obteniéndose como aislamiento principal Arizona hinshawii.

THE ISOLATION OF Arizona hinshawii FROM BROILERS, BREEDING ANIMALS, INCUBATION WASTE MATERIALS AND FOOD COMPONENTS USED IN POULTRY DIETS IN MEXICO.

The Departamento de Producción Animal:Aves, received during 1979 several - cases of young and adults animal (healthy or disease). Some of which had - clinical syntoms of Salmonellosis or Omphalitis disease.

Some samples of meat meal, bone meal and incubation waste material from the states of Guanajuato, Morelos, Veracruz, Yucatán y Estado de México were - studied as well.

The most common pathological lesions were found in the liver of the affected animals (hepatomegaly, brownish decoloration and degeneration of the -- tissue).

The bacteriological analysis demostrate the presence of Arizona hinsawii in the affected tissues of 15 cases.

Arizona hinshawii was isolated from the samples of meat meal, bone meal and the incubation waste materials using the methods recomended by Gentry and - the Food and Drug Administration.

INTRODUCCION.-

La arizonosis es una enfermedad causada por Arizona hinshawii, aislada por primera vez en reptiles en el año de 1939 (1,2,4,9,11). En las aves se aisló en 1936 de pollos jóvenes que sufrieron una infección similar a la pulorosis. Esta bacteria también es la causa de enfermedad en otras aves,mamíferos y el hombre (1). Esto probablemente se debe a la ingestión de alimentos contaminados, como sucede con el huevo en polvo (3),carne y huevos de aves infectadas (1).

La importancia de este microrganismo radica en su potencial patógeno similar al de las Salmonelas que se transmiten a través del huevo (13),por lo que - representa un serio peligro para la industria avícola debido a las pérdidas económicas que ocasiona.

Aparentemente en México no se ha reportado la presencia de arizonosis en gallinas de postura y pollos de engorda. Sin embargo, el aislamiento del agen

te patógeno se ha logrado en varias ocasiones de animales remitidos para el diagnóstico de Salmonela o incluso en aves aparentemente sanas.

En el año de 1979 se recibieron en el Departamento de Producción Animal:Aves de la Facultad de Medicina Veterinaria y Zootecnia de la Universidad Nacio-- nal Autónoma de México, 1429 casos clínicos en los cuales se realizaron 596 estudio bacteriológicos,63 muestras de alimento y 15 muestras de detritus de incubación.

SIGNOS Y LESIONES.

Al revisar las historias clínicas se indicaba que en las aves jóvenes había mortalidad variable desde el primer día de edad y se continuaba por una semana. La morbilidad era del 1 al 10 %, había deshidratación,debilidad,ema-- ciación, palidez,diarrea blanquecina,tristeza,incoordinación,anorexia y taponamiento de la cloaca.Las lesiones eran similares a las producidas en la Infección del Saco Vitelino.

Por otro lado,en aves cuya edad variaba desde una hasta treinta y tres semanas de edad,mostraban mortalidad cuyos porcentajes variaban del 1.5 al 5%, morbilidad del 1 al 10%,poliuria,diarrea de color verdosa,deshidratación, - erizamiento de plumas,debilidad,emaciación y palidez.

En el examen postmortem las lesiones más notables estaban localizadas en el hígado el cual mostraba aumento de volumen,friable y de color bronceado. -- También había esplenomegalia,peritonitis y folículos ováricos rotos congestionados y hemorrágicos, los riñones se observaron pálidos,aumentados de tamaño y con uratos.

DIAGNOSTICO BACTERIOLOGICO.-

Después de realizada la necropsia de las aves,se colectaron muestras de los órganos afectado,las cuales se trabajaron de acuerdo a las técnicas convencionales (5,8,10,12,4).

Las muestras de plumón se trabajaron de acuerdo a la técnica de Gentry (7).

Las muestras de alimento se analizaron según la técnica propuesta Food and Drug Administration (6).

CUADRO I

AISLAMIENTOS DE ARIZONA hinshawii OBTENIDO DE QUINCE LOTES DE AVES PROCEDENTES DE DIFERENTES ESTADOS DE LA REPUBLICA MEXICANA,PRESENTADOS AL DEPARTAMENTO DE PRODUCCION ANIMAL:AVES.

MUESTRA	RAZA	EDAD	PROCEDENCIA	ORGANOS
R +	PESADA	2 días	MORELOS	B, V.B. *
R	PESADA	3 días	MORELOS	B, V.B. *
R	PESADA	4 días	MORELOS	B, V.B. *
P.E. ++		6 días	EDO.DE MEXICO	H,B.S.V.,V.B.*
R	PESADA	6 días	EDO.DE MEXICO	S.V.,V.B.
R	PESADA	10 días	MORELOS	B.
P.E.		10 días	EDO.DE MEXICO	H.
R	PESADA	1 sem.	MORELOS	H.
R	PESADA	8 sem.	MORELOS	H.B.V.B.
R	PESADA	15 sem.	MORELOS	V.B.
R	PESADA	18 sem.	VERACRUZ	HB,CP.
R	PESADA	27 sem.	EDO.DE MEXICO	H.
R	PESADA	31 sem.	YUCATAN	CLOACA
P +++	PESADA	33 sem.	EDO.DE MEXICO	B.
R	PESADA	2 días	GUANAJUATO	H.B.V.B.S.V.

* B.- Bazo
H.- Hígado
V.B.-Vesícula biliar
S.V.-Saco Vitelino
C.P.-Corazón-Pulmón

\+ Reproductoras
++ Pollo de engorda
+++ Ponedoras

RESULTADOS Y CONCLUSIONES

Los resultados obtenidos se anexan en los cuadros 1 y 2.

De acuerdo a los resultados obtenidos se puede deducir que las materias primas utilizadas para la elaboración de alimento para las aves,están contaminados con Arizona hinshawii en un porcentaje elevado,lo mismo que los detritus de incubadora;por lo tanto consideramos que se deben realizar estudios bacteriológicos periódicos de las materias primas y aves,así como realizar una buena desinfección en las plantas incubadoras,mientras tanto debemos estar alertas ante la presencia de una nueva enfermedad la Arizonosis.

CUADRO II

AISLAMIENTOS DE ARIZONA hinshawii OBTENIDOS DE SEIS MUESTRAS DE HARINA DE HUESO Y UNA MUESTRA DE DETRITUS DE INCUBACION.

HARINA DE CARNE	EDO. DE MEXICO	CLOACA
HARINA DE CARNE	EDO. DE MEXICO	CLOACA
HARINA DE CARNE	EDO. DE MEXICO	CLOACA
HARINA DE CARNE	EDO. DE MEXICO	CLOACA
HARINA DE CARNE	EDO. DE MEXICO	CLOACA
HARINA DE HUESO	EDO. DE MEXICO	CLOACA
HARINA DE CARNE	EDO. DE MEXICO	CLOACA
PLUMON	GUANAJUATO	CLOACA

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"A ELEVEN YEAR STUDY OF SALMONELLA SEROTYPES IN AN INTEGRATED TURKEY OPERATION"

R. A. Bagley, D.V.M., M.S., and M. J. Ramsay, D.V.M.
Moroni Feed Company
Moroni, Utah 84646

Forty six salmonella serotypes were isolated during a eleven year monitoring program in an integrated turkey operation.

To determine the serological relationship of the 46 serotypes the Kuffmann-White Schema was used. It was found that 15 groups were involved. Groups B, C and E represent 82.6% of all the serotypes.

The turkey operation was divided into 3 areas. Area 1 was the breeder-hatchery operation. Area 2 was the poults, growing birds and processing. Area 3 was the complete feed and ingredients. Area 3 was often the source of new serotypes entering the operation. An average of 71% of the serotypes recovered annually came from the feed, while from Area 1 it was 49% and Area 2 it was 51%.

UN ESTUDIO DE 11 AÑOS DE LOS SEROTIPOS DE SALMONELLA EN UNA OPERACIÓN INTEGRADO DE LOS PAVOS

Se aislaron 46 serotipos de la Salmonella durante un programa monitorial de 11 años en una operación integrada de los pavos.

Para determinar la relación serológica de los 45 serotipos, se usaron el esquema de Kauffman-White. Se encontró que había 15 grupos. Los grupos B,C y E representaron 82.6% de todos los serotipos.

La operación de los pavos se dividieron en 3 áreas: La primera área: La operación de criadora-reproductor; la segunda área: Los pavitos y los pavos crecientes y el proceso; la tercera área: El alimento completo y su contenido. La tercera área fue muchas veces el origen de serotipos nuevos entrando en la operación. Un promedio de 71% de los serotipos recubiertos por año venían del alimento, mientras los de la primera area representaron 49% y los de la segunda área representaron 51%.

"A ELEVEN YEAR STUDY OF SALMONELLA SEROTYPES IN AN INTERGRATED TURKEY OPERATION"

R. A. Bagley, D.V.M., M.S., and M. J. Ramsay, D.V.M.
Moroni Feed Company
Moroni, Utah 84646

In the Report of the U. S. Advisory Committee on Salmonella, January 1978, the preface (3) states that the problem of Salmonella contamination of the meat and poultry supplies has existed probably for as long as man has been using animals for food. The U. S. Department of Agriculture, the Food and Drug Administration, and the Food Industry have attempted to attack this problem many times in various ways. The complexity of the problem is underscored by the fact that, dispite the best efforts of all these groups, salmonella infection in meat and poultry food supply is occuring.

The literature has many references (1,2,3,4,5,6,7) that indicates the complexity of the salmonella problems. In the attempt to reduce or eliminate salmonella from a turkey operation, a monitoring program was instituted over 12 years ago, for the purpose of evaluating the various medication and sanitation programs. This paper will discuss the classification of the salmonella isolates recovered during this period of time.

During the eleven years of monitoring from 1968 to 1978 a total of 46 serotypes were isolated. Using the Kuffmann-White Schema (4), 15 serogroups were identified. Eighty-three percent of all the serotypes were classified in groups B, C and E.

Classification of the 46 serotypes by Kuffmann-White Schema

Groups	Isolates	Percent
B	12	26.09
C	13	28.26
D	1	2.17
E	13	28.26
G	2	4.35
K	2	4.35
L	1	2.17
O	1	2.17
R	1	2.18
Total	46	100.00%

The source of these serotypes are from three areas. Area 1 - breeder-hatchery. Area 2 - birds in the coop, range and processing plant. Area 3 - complete feed and ingredients.

Number of serotypes isolated by year

Year	Total Number	Area 1 #	Area 1 %	Area 2 #	Area 2 %	Area 3 #	Area 3 %
1968	30	21	70%	11	35%	22	73%
1969	32	19	59%	25	78%	24	72%
1970	24	13	54%	13	54%	20	83%
1971	18	11	61%	8	44%	16	89%
1972	24	10	42%	7	31%	21	88%
1973	13	7	54%	10	77%	6	46%
1974	11	6	55%	6	55%	5	45%
1975	17	1	6%	5	29%	15	88%
1976	18	5	29%	8	47%	16	89%
1977	28	19	69%	15	54%	12	42%
1978	26	11	42%	15	58%	18	69%
			49%		51%		71%

The introduction of a serotype into the operation frequently came by the feed route. In monitoring breeder flocks non-pelleted feed was often the source of a new serotype appearing in the environment and intestines of a laying hen.

Total number of years serotypes were isolated by sources

Salmonella Serotypes	KWS	Area 1	Area 2	Area 3
agona	B	1	2	1
albany	C - 3	2	0	1
alachua	O	1	3	3
anatum	E - 1	10	10	9
barielly	C - 1	0	0	2
binza	E - 2	2	1	5
blockley	C - 2	1	2	1
bredeney	B	4	2	6
california	B	1	0	1
cerro	K	0	1	2
chester	B	1	2	1
derby	B	4	6	8
drypool	E - 2	1	0	1
eimsbuettel	C - 4	5	6	10
enteritidis	D - 1	1	1	1
give	E - 1	2	1	2
grumpensis	G - 2	0	0	1
heidelberg	B	11	11	7
infantis	C - 1	5	4	5
johannesburg	R	0	2	1
kentucky	C - 3	0	3	7
lexington	E - 1	0	0	1
livingstone	C - 1	0	1	3
meleagridis	E - 1	2	0	4
minnesota	L	4	4	6
montevideo	C - 1	6	4	9
muenchen	C - 2	4	5	3
muenster	E - 1	1	5	6
newington	E - 2	1	1	1
newport	C - 2	3	1	2
oranienburg	C - 1	2	2	5
orion	E - 1	1	2	3
reading	B	2	5	7
san diego	B	9	7	5
st. paul	B	8	8	2
schwarzengrund	B	4	4	6
seftenberg	E - 4	5	1	5
sieburg	K	0	1	6
simsbury	E - 4	1	2	3
stanley	B	1	0	0
taksony	E - 4	0	0	2
tennessee	C - 1	5	2	6
thomasville	E - 3	0	1	0
thompson	C - 1	4	4	5
tyrimurium	B	5	2	3
worthington	G - 2	3	1	4

Summary

1. Forty six salmonella serotypes were isolated during the 11 year monitoring program.

2. The serotypes were constantly changing.

3. Some serotypes were capable of persisting for many years such as, S. heidelberg and S. anatum, in all three areas.

4. Non-pelleted feed was a good source for introducing new serotypes.

5. Salmonellosis represents a public health concern and problem, however, in this operation it did not manifest itself as a serious disease problem.

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Competitive exclusion of paratyphoid salmonellae and *E. coli* by natural protective microflora.

G.H. Snoeyenbos, Olga M. Weinack and C.F. Smyser

Department of Veterinary and Animal Sciences
University of Massachusetts
Amherst, Massachusetts 01003

The major findings during the past few years of the several groups working with competitive exclusion of salmonellae in the chicken by naturally occurring intestinal microflora will be reviewed. More recent data indicates that existing infections are abbreviated by the protective microflora, that stress can interfere with protection, and that at least some strains of *Escherichia coli* are also inhibited. The limited work done with turkeys indicates the presence of a comparable protective mechanism.

Results of field trials will be used to consider possible practical field application of competitive exclusion to assist in the control of salmonellae.

Exclusión competitiva de Salmonellae paratifoidea y *E. coli* por la microflora natural protectora

Una revisión de los trabajos realizados en pollos sobre exclusión competitiva de Salmonellae por la microflora intestinal natural sera presentada, haciendo referencia a los hallazgos más importantes ocurridos durante los últimos años. Los resultados más recientes indican que los efectos de infecciones existentes en pollos son disminuidos por la microflora protectora, que el stress puede interferir con la proteción, y que por lo menos algunas cepas de *Escherichia coli* son inhibidas. Los pocos trabajos realizados en pavos muestran mecanismos de proteción comparables a los hallados en pollos.

Resultados obtenidos en experimentos de campo serán usados para considerar el possible uso practico de exclusión competitiva en el control de salmonellae en condiciones de campo.

COMPETITIVE EXCLUSION OF PARATYPHOID SALMONELLAE AND E. COLI BY NATURAL PROTECTIVE MICROFLORA.

G.H. Snoeyenbos, Olga M. Weinack and C. F. Smyser
Department of Veterinary and Animal Sciences
University of Massachusetts
Amherst, Massachusetts 01003

Nurmi and Rantala (1) in 1973 were the first to report that intestinal microflora from adult chickens could be transferred to chicks with resulting enhanced resistance to subsequent infection by paratyphoid salmonellae. This paper is intended to review some of the findings of the several research groups who have investigated this phenomenon of competitive exclusion (CE) in the intervening years. Although not a part of this presentation, it is worth noting that direct and indirect information indicates the existence of a comparable protective mechanism in a range of avian and mammalian species.

Neither the mechanism of protection nor the microflora responsible for protection has been identified. Developing evidence supports the hypothesis that the protective flora attaches to epithelial cells of the crop and ceca, the critical sites of salmonella colonization in the chicken, and prevents attachment and colonization by salmonella. Although salmonella inhibitory metabolites of the protective flora may play a role, it seems likely that attachment is of the first order of importance. There is reason to expect that CE is the result of action by a complex of bacteria. One of the reasons for this appraisal has been the finding that microflora from different flock sources have variable degrees of protective activity; some have substantially no activity. This is a practical point of considerable importance. Published and unpublished work has demonstrated that colonization of the gut by avian strains of Lactobacilli does not result in significant CE of salmonellae. It is perhaps premature to conclude that Lactobacilli do not play a role in CE as interaction among several bacteria may prove necessary for protection; variable CE activity found on subsequent trials with the *Streptococcus faecalis* strain reported as partially protective by Soerjadi *et al*. (4) suggests that such interaction may occur.

For about the past two years we have used for reference test purposes, as well as for field trials, a suspension of fresh feces from a group of about 50 SPF chickens. A selected protective microflora was transferred to these birds following anaerobic broth passage. Protection by the microflora from these donor birds is equal to that of the originals and the birds are free of pathogens as indicated by monthly tests by SPAFAS standards.

Table 1 includes abbreviated data to show the usual rapid spread and long duration of salmonella excretion following introduction of infected seeders, the degree of control that is expected by CE, and the abbreviation of infection resulting from CE activity. Apparently the protective microflora dislodges salmonella colonies in the gut and may be considered to have therapeutic activity.

Table 1. Isolation of Salmonella enteritidis (Na) from cloacal swabs and litter samples from control and treated chicks following exposure by seeders.

Age (days)	Not treated		Treated 2 days before adding seeders		Treated 3 days after adding seeders	
	Seeders	Contacts	Seeders	Contacts	Seeders	Contacts
7	3/3	47/50	3/3	5/50	3/3	49/50
21	3/3	48/50	2/3	1/50	2/2	38/50
42	1/3	25/50	1/3	0/50	0/2	3/50
63	0/3	20/50	0/3	0/50	0/2	2/50
84	0/3	18/50	0/3	1/50	0/2	0/50
Cum.	3/3	50/50	3/3	7/50	3/3	50/50

Abbreviated data, Snoeyenbos *et al*. (2).

For about the past year we have used an individual challenge test system in parallel with a seeder bird test system to allow more precise measurement of CE in laboratory trials. A nalidixic acid resistant strain of salmonella is used for challenge to allow enumeration of salmonella by

direct plating of fecal suspensions. Table 2 shows data indicating that exposure ranging from $5x10^3$ to $5x10^8$ cells infected all unprotected chicks and resulted in comparable levels of salmonella excretion. The numbers of birds infected and the excretion rate of CE protected chicks was dose-related and showed measurable protection even at a challenge dose as high as $5x10^8$. Use of the combined test systems indicated that the stress of overheating, chilling, or food and water starvation of chicks resulted in some loss of protective activity by CE (3). Several reports have documented the ability of some broad spectrum antibacterials to significantly interfere with CE.

Table 2. Influence of dose of Salmonella enteritidis (Na) on recovery from cloacal swabs and feces of treated and control chickens.

	Culture results at 6 days of age.					
Salm.	Not treated			Treated		
dose	Cl. swabs	Feces	Count	Cl. swabs	Feces	Count
$5x10^3$	10/10	+	$6x10^6$	1/10	+	< 10
$5x10^4$	10/10	+	$8x10^6$	0/10	-	< 10
$5x10^5$	10/10	+	$1x10^7$	0/10	+	< 10
$5x10^6$	10/10	+	$2x10^7$	1/10	+	$1x10^4$
$5x10^7$	10/10	+	$4x10^7$	1/10	+	$5x10^3$
$5x10^8$	10/10	+	$3x10^7$	8/10	+	$3x10^3$

Treatment at 1 day; salmonella at 3 days.
Abbreviated data, Weinack *et al*. (5).

The economy of nature suggests that CE protective mechanism is likely to be protective against pathogens other than salmonellae. Tests using our combined systems with 4 serotypes of Escherichia coli derived from pathological processes in chickens indicate a degree of protection by CE against these strains comparable to that against salmonella. The practical significance of these findings is unknown.

Table 3. Isolation of Escherichia coli OX7 from control and treated chicks.

	Culture results by days of age								Cumulative Positive	
	7				28					
	Exposure				Exposure					
Treat.	Dir.	Cont.	F.	Ct.	Dir.	Cont.	F.	Ct.	Dir.	Cont.
None	2/2	3/10	+	10^6	1/2	0/10	+	10^5	2/2	8/10
"	7/10		+	10^6	3/10		-	10^3	10/10	
PH	2/2	0/10	+	10^3	0/2	0/10	-	<10	2/2	0/10
"	0/10		-	<10	1/10		-	<10	1/10	

Treated at 1 day; E. coli at 3 days. (E. coli OX7 strain Na resistant)
Abbreviated data.

Field trials to assess activity of CE against salmonellae are hampered by apparent high transferability of the protective flora. Available data must, unfortunately, be judged on a basis of expectation of very rapid spread of salmonella, detectable by cloacal swab culture, among young chicks. We have consistently secured transmission regardless of serotype or strain to a majority if not all chicks reared in a dependably clean environment if "seeders" are added to the group during the first few days of life. The data on Table 4 demonstrates the low numbers of cloacal swab positive birds from naturally infected chicks which were treated in drinking water at time of starting chicks. The data on Table 5 is a summary of salmonella isolations from litter and cloacal swabs from a trial using naturally infected chicks treated individually at the hatchery or by drinking water at the time they were placed in the pens. Each pen was populated by either 55 male or 68 female chicks. There were 32 replicate pens for each treatment. The incidence of infection in birds treated by either method was strikingly lower throughout the trial than would be expected in untreated birds in a clean environment. These results indicate that protection is improved if chicks are individually treated in the hatchery rather than later in the drinking water when placed in the house.

Table 4. Isolation of salmonellae from cloacal swabs and litter of naturally infected chickens treated in drinking water.

(Birds: 13,740) Growing House (Pens: 4)			(Birds: 7,969) Laying House (Pens: 20)		
Age/days	Litter	Cloacal	Age/days	Litter	Cloacal
4	17/20	Not done	174	12/100	8/400
13	60/62	11/199	188	20/100	1/400
25	55/61	2/200	201	13/100	0/400
41	35/40	9/200	214	0/100	0/400
53	31/40	4/200	242	0/100	Not done
68	35/40	4/200	280	0/100	" "
82	36/40	2/200	307	0/100	" "
86	30/40	5/200	327	1/100	" "
110	19/40	2/200	362	0/100	" "
124	23/40	2/200	405	0/100	" "
138	14/40	0/200	439	0/100	" "
			494	0/100	" "

Isolations included: Salmonella senftenberg, S. infantis, S. heidelberg, S. agona, S. montevideo and S. enteritidis.
Snoeyenbos et al. (3).

Table 5. Salmonella isolations from litter and cloacal swabs from naturally infected chickens.

Treatment		Culture results by weeks 1	3	5	7	TOTAL
Individual	Litter					
	Samples	43/128[A]	84/128	58/128	19/128	204/512
	Pens	14/32[B]	26/32	24/32	11/32	27/32
	Cloacal					
	Samples	2/192	0/320	10/320	1/384	13/1216
	Pens	1/32	0/32	5/32	1/32	6/32
Water	Litter					
	Samples	128/128	128/128	53/128	37/128	346/512
	Pens	32/32	32/32	25/32	20/32	32/32
	Cloacal					
	Samples	37/192	17/320	67/320	7/384	128/1216
	Pens	25/32	15/32	26/32	7/32	31/32

Isolations included: Salmonella infantis, S. agona, S. blockley and S. typhimurium.
[A]Samples positive/samples tested. [B]Pens positive/pens tested.

Although much work is yet to be done to allow understanding of the complex protective mechanism, it appears that early colonization of the gut of the chick and poult with a protective microflora should prove to be a useful adjunct to some of the more usual methods used to control and prevent salmonellosis and perhaps other infections.

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PODEMOS ERRADICAR LA TIFOIDEA AVIARIA EN MEXICO?

José Antonio Quintana López.- Departamento de Producción Animal: Aves, F.M.V.Z.Universidad Nacional Autónoma de México.

La tifoideaa aviaria en México produce pérdidas económicas por más de 800 millones de pesos, debidas a mortalidad del pollo de engorda y de reproductoras, así como reducción en la producción de huevo y en la incubabilidad. En un año se dejaron de producir 100 toneladas de carne debido al déficit de 600 000 reproductoras, responsables de la producción de 8 millones de pollitos mensuales, siendo los productores no integrados los que han sufrido más. Esto ha sido el resultado de un aumento en los brotes de tifoidea a partir de 1978, mismos que hicieron crisis en 1979. Durante 1979 hubo una fuga de divisas por 93 millones de pesos por concepto de huevo fértil, pollito de engorda y reproductoras; ésto no se había visto en México desde 1961 en que el país se convirtió en autosuficiente.
La principal forma de transmisión de la tifoidea es la transovárica, sin embargo algunos veterinarios piensan que las harinas de orígen animal contaminadas, las ratas, el agua y el personal pueden tener importancia en su transmisión.
Algunas recomendaciones básicas que deben ayudarnos en la erradicación de la tifoidea son:

1) Las granjas de reproductoras deben estar aisladas de otras aves por 1 o 2 km.
2) Las granjas de reproductoras deberán tener parvadas de una sola edad.
3) Todo el personal deberá bañarse y cambiarse de ropa antes de entrar en la granja. El equipo, los edificios y alrededores deberán ser lavados y desinfectados antes de recibir a los pollitos.
4) Debe implementarse un programa de erradicación de ratas y pájaros.
5) Los pollitos deben provenir de parvadas certificadas libres de la enfermedad.
6) El agua debe ser tratada con cloro. El alimento debe ser peletizado y estar libre de harinas de orígen animal.
7) Cuando la parvada alcance el 5% de producción el 100% de las aves deberán ser sangradas para realizar la prueba de aglutinación en placa con sangre completa. Las aves no deberán haber recibido ninguna droga capaz de enmascarar la reacción en las 4 semanas previas a la prueba.
8) Las aves positivas deberán ser sangradas para realizar una prueba de aglutinación en tubo con su suero.
9) Las aves positivas a la prueba de aglutinación en tubo deberán ser alimentadas y deberá mandarse 5 a 10 de ellas a un laboratorio de diagnóstico para su examen bacteriológico.
10) El huevo deberá incubarse sólo después de que se haya descartado la presencia de Salmonella *pullorum-gallinarum* por examen bacteriológico.

IS IT POSSIBLE TO ERRADICATE AVIAN TYPHOID FROM MEXICO?

Avian Typhoid in México causes economic losses of up to 800 million pesos due to mortality in broilers and breeders, reduced egg production and incubability. One hundred thousand tons of meat/year were not produced because a deficit of 600 000 breeders that should have produced 8 million baby chicks per month. Unintegrated producers are suffering the most. This is the result of an increase in Avian Typhoid outbreaks,starting in 1978 and reaching its peak in 1979. During 1979 ninety three million pesos were spent abroad in fertile eggs, baby broiler chicks, when it became self sufficient.
The chief means of transmission is transovarian, although some veterinarians have indications of contaminaed animal by-product meals, and suspect that birds, rats,caretakers and water may play a role.
Some basic rules helpfull in erradicating *Salmonella pullorum-gallinarum* are:
1) Breeder farms should be separated from other poultry by 1 or 2 kilometers.
2) Breeder farms should have single age flocks.
3) All personnel should change clothing and bath before entering the premisses; equipment, building and their surroundings should be thouroughly washed and desinfected before the chicks arrive.
4) An erradication program of rats and birds should be implemented.
5) Chicks should come from disease-free-certified breeder flocks.
6) Water should be treated with chlorine; feed should be pelletized and free of animal-by products.
7) When the flock reaches 5% production one hundred percent of the birds should be tested for typhoid by whole blood plate agglutination, taking care that no drugs capable of inhibiting the reaction had been administered to the birds during the previous four weeks.
8) Positive reactors should be tested using the tube agglutination test.
9) Positive reactors to the tube agglutination test should be dispossed off and 5 to 10 sent to a diagnostical laboratory for bacteriological confirmation.
10) Eggs should be incubated only after *Salmonella pullorum-gallinarum* have been ruled out.

APPLICATION OF MICROTITER TEST ON SALMONELLA GALLINARIUM VACCINATED FLOCKS

FIELD REPORT

MONTE N. FRAZIER, D.V.M.
DIRECTOR OF POULTRY HEALTH
ARBOR ACRES FARM, INC.
GLASTONBURY, CONNECTICUT 06033

Salmonella gallinarium infection (Fowl Typhoid) is still a major disease problem in many areas of the world. Chemotherapy has been of little value for either prevention or treatment. The use of a rough mutant strain referred to as 9R, has proven effective as a biological method of control. When this vaccine is used there are some reactions to the whole blood or serum plate agglutination test. This makes it difficult to identify infected flocks in any type of a program for control of the disease. The use of the micro agglutination test using a Salmonella group D antigen can differentiate between vaccinated non-infected flocks and infected flocks. This antigen is now commercially available and the test is an official test under the National Poultry Improvement Plan(NPIP). This approach could be a very valuable tool in using the 9R vaccine to reduce the incidence of the disease in control program using test and slaughter.

LA APLICACIÓN DE LA PRUEBA DEL MICROTITULACIÓN EN LAS PARVADAS VACUNADAS CON SALMONELLA GALLINARIUM

La infección de Salmonella Gallinarium (el Tifoideo de las Aves) es todavía una enfermedad principal y problemática en muchas partes del mundo. La terapía química ha tenido poco valor en o la prevención o el tratamiento. El uso de la clase mutante tosca que se llama 9R ha probado eficaz como un método de control biológico. Cuando se usa esta vacuna hay algunas reacciones de la sangre total o la prueba de la aglutinación de la estructura del sero. Por eso, es difícil identificar las parvadas infectadas en cualquier programa para el control de la enfermedad. Se puede diferenciar entre las parvadas infectadas y las que no son infectadas por el uso de una prueba de la micra-aglutinación usando un antígeno del grupo D de Salmonella. Este antígeno ya es disponsible comercialmente y la prueba es una prueba oficial bajo El Plan de Mejoramiento Nacional de Las Aves de Corral. Este método pueda ser un instrumento muy importante en el uso de la vacuna 9R para reducir la incidencia de la enfermedad en el programa de control usando la prueba y la mantanza.

APPLICATION OF THE MICROTITER TEST ON SALMONELLA GALLINARUM VACCINATED FLOCKS FIELD REPORT

M. N. Frazier, D.V.M.
Arbor Acres Farm, Inc.
Glastonbury, Connecticut 06033

Salmonella gallinarum infection (Fowl typhoid) is a major poultry disease problem in many areas of the world. Chemotherapy has been of limited value for prevention or treatment. Attempts at immunization with inactivated agents have been disapointing, using either autogenous or stock cultures. However, Smith in 1956 (1) reported successful immunization using a smooth (9S) or rough (9R) attenuated culture. The rough culture did not produce agglutinins, mortality in 1 day old chicks nor have any effect on egg production. The smooth culture however, produced agglutinins, killed 1 day old chicks and caused depression in egg production. Subjequent reports of satisfactory results of the 9R cultures as an immunizing agent were by Harbourne in 1957 (2), Gordon et al. (3) and Gordon and Luke (4) in 1959. In addition, Harbourne in 1963 (5) and Smith in 1969 (6) reported the successful use of a freeze dried 9R vaccine. The author is aware of the use of the vaccine in Argentina, Columbia, Ecuador, England, Greece, Italy, Jordan, Mexico, Lebanon, North Ireland, South Africa and Venezuela.

The stained antigen rapid whole blood agglutination (RWB) test, the rapid serum agglutination (RS) test and the standard tube agglutination (TA) test have all been used to detect infection with S pullorum and S gallinarum. In 1971 Williams and Whittemore (7) reported the use of the microagglutination (MA) test for detecting infection. The MA test has been adopted as an official test by the National Poultry Improvement Plan (NPIP).

Materials and Methods

A single house of 5000 broiler breeders on a farm of 16 houses with a total of 80,000 breeders was found to be infected with S gallinarum. The entire farm was vaccinated with the 9R vaccine when the diagnosis was confirmed. In spite of the use of the vaccine abnormally high mortality continued in the infected flock and egg transmission became evident in the progeny. The infected flock was disposed of and a program of vaccination every 8 wks. was undertaken on the remainder of the farm. When these flocks were tested by the RWB test they were found to have varying numbers of reactors, see Table 1.

Table 1

Results of whole blood test

Number of times vaccinated with 9R	Time since last vaccination	0%+
0	--------	0
1	9 weeks	0
1	2 weeks	1.6
1	8 weeks	1.6
1	12 weeks	1.6
1	6 weeks	2.7
2	6 weeks	3.6
4	2 weeks	7.7
4	6 weeks	8.1
4	2 weeks	11.3
4	2 weeks	28.3
0	-------	30.7

These results make it difficult to determine if such flocks are infected with the virulent organism or are merely reacting as a response to the vaccine. In spite of Smiths' observation on the lack of agglutinins in 9R vaccinated birds it has been the authors' experience that reactions usually do occur, the number decreasing over time.

Trial 1

One hundred blood samples from birds that were positive to the RWB test (2 plus or greater reactions are considered positive in all trials) were taken to the laboratory and the serum separated. In addition 25 random samples were obtained from a known infected flock that was not vaccinated to be used along with 25 known negative samples as controls. The RS test was performed on sera undiluted and diluted at 1:5,1:10, 1:20, 1:40 and 1:80 with 0.85% NaCl.

Trial II

Because of the difficulty of working with the serum dilution in the RS test it was decided to utilize the MA test using dilutions of 1:20,1:40, 1:80 and 1:160. The remainder of the flock, approximately 75,000 birds were tested by the RWB test and serum saved from blood samples taken from any bird that was considered a reactor. These birds were then killed. A total of 2165 reactors were found (2.8%). 100 samples were taken from known reactors in an infected non-vaccinated flock and 25 samples from a known negative non-vaccinated flock.

Results

The results of Trial I are shown in Table 2

Table 2

Dilution	Vaccinated chicken		Random sample infected flock non-vaccinated		Non-infected Non-vaccinated	
	+	—	+	—	+	—
None	100	0	8	17	0	25
1:5	65	35	8	17	0	25
1:10	10	90	8	17	0	25
1:20	2	98	8	17	0	25
1:40	1	99	7	18	0	25
1:80	0	100	6	19	0	25

The results of Trial II are shown in Table 3

Table 3

Dilution	Vaccinated birds		Non-vaccinated reactors from infected flock		Non-vaccinated Non infected birds	
	+	—	+	—	+	—
1:20	161	2004	100	0	0	25
1:40	32	2133	100	0	0	25
1:80	5	2160	100	0	0	25
1:160	0	2165	98	2	0	25

Summary and discussion

In interpreting the results it was very striking that the vaccinated reactors did not react at the 1:160 dilution and the infected reactors did.

In the NPIP the 1:20 dilution is used to determine reactors. It is obvious that this dilution could not be used to determine reactors in vaccinated flocks. The RWB test has very limited use in vaccinated flocks. The standard tube agglutination test using dilutions would probably be of value but the MA test has many advantages, particularly for a laboratory not already set up for tube testing.

It was unfortunate in this trial that we did not have some of the reactors at the different dilutions for culture. However, since these trials all mortality on this farm has been cultured (heart blood, liver and spleen) and S. gallinarum has not been recovered.

The use of any vaccine that produces agglutinins is going to confound a program using the agglutination test as a tool for long-term control. The author believes that the MA test can detect significant reactors, using dilution, more accurately than the RWB test. This is highly important for selecting birds for microbiological examination to detect true infection. With 10 to 20% reactors to the RWB test selection of individuals is, at best, difficult. With the highly virulent form of S. gallinarum infection prevalent in many areas of the world, and with the disease epidemic in many of these areas, control of the disease is going to be extremely difficult without some method of reducing the incidence of the disease. Medication to prevent or reduce the severity of the disease has been very disappointing; delayed onset or temporary reduction of mortality is the usual result.

It will take a comprehensive long-term project to control this disease when the disease is highly endemic or epidemic.

1. All grandparents (GP) must be kept free of the disease by isolation and sanitation. Vaccination of GP flocks with 9R is totally unacceptable if there is any hope to control the disease. Medication of the GP flock and their progeny for the first 2 weeks should be limited to give the disease, if present, a chance to express itself so as to identify infected flocks.

2. Broiler parents or commercial egg parents and commercial egg producers can be vaccinated but it should be a planned program. The 9R vaccine will not contain the disease if vaccination is done in the face of an outbreak, in fact, it may add fuel to the fire. Once clinical disease occurs in a flock, it should not be used for breeding. This only serves to disseminate the disease. Infected commercial egg producers should be put under a limited quarantine to help control infection.

3. A single 100% blood test at sexual maturity is not sufficient in a highly endemic or epidemic area. Repeat blood testing and cultures of mortality of progeny and of adult mortality should be part of the program.

4. Good sanitary procedures in combinationwith the 9R vaccine can work to reduce the incidence, vaccination alone will not. A good educational program is also essential. Producers must be made aware of the limitations of vaccination; that it will not work in face of an outbreak; that it will not prevent egg transmission in an infected flock.

5. The production and sale of the vaccine should be controlled, standardization is essential.

6. The microtest can help to differentiate vaccinated non-infected flocks from infected flocks, however, more work needs to be done to establish parameters. The antigen is commercially available.

I wish to thank Dr. J. E. Williams
Southeast Poultry Research Lab.
U.S.D.A., Science & Education Administration
934 College Station Road
Athens, Georgia 30601

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PREPARACION DE ANTIGENO PULLORUM DE ALTA SENSIBILIDAD Y ESPECIFICIDAD

Mateo H. Delamer
San Sebastián S.A.
Belgrano 347, Buenos Aires, Argentina

RESUMEN

So logró disminuir las aglutinaciones inespecíficas con antígeno pullorum standard en aves no infectadas ("non-pullorum-reactors") mediate el reemplazo de la formalina como inactivante ya que se observó que producía un deterioro de las células con tendencia a la autoaglutinación. Asimismo, se reemplazaron las dos cepas standard y las tres cepas variantes de Salmonella Pullorum por una cepa de Salmonella Gallinarum carente del factor "O" 1 pero con alto contenido del factor "O" 9 y cantidades suficientes de los factores "O" 12_2 y 12_3.

Un antígeno preparado con la cepa TI-184/64 (Kösters) de S. gallinarum inactivado con Merthiolate y coloreado con cristal violeta, se mostró en pruebas de campo y de laboratorio más específico y sensible que el antígeno pullorum polivante "K" standard.

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PREPARATION OF PULLORUM ANTIGEN OF HIGH SENSITIVITY AND SPECIFICITY

Nonspecific agglutinations with standard pullorum antigen were reduced in "non-pullorum reactors" by replacing formalin as an inactivant, since it was observed that same caused a deterioration of the cells with a tendency to autoagglutination. Furthermore, the two standard strains and the three variant strains of Salmonella pullorum were replaced by one strain of Salmonella gallinarum without factor "O" 1 but with a high "O" 9 content and sufficient quantities of factors "O" 12_2 and 12_3.

An antigen prepared with strain TI-184/64 (Kösters) of S. gallinarum inactivated with Merthiolate and colored with crystal violet, proved in field and laboratory tests to be more specific and sensitive than the standard polyvalent "K" pullorum antigen.

(Translation courtesy of Dr. Carlos López Cuello.)

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INTRODUCCIÓN

El antígeno pullorum polivalente "K" standard es el reactivo universalmente usado para la detección de las aves portadoras de S. gallinarum y S. pullorum. (1)

Con frecuencia aparecen en lotes no infectados aves reaccionantes, lo que ha dado origen a la denominación de "falsos reaccionantes", a "non-pullorum reactors". (2-4-5-11)

Los dos causas mas frecuentes para la aparición de estas racciones inespecíficas son el empleo de antígenos parcialmente autoaglutinados y el gran mosaico antigénico del antígeno standard. (3-6-7-12-13)

El método de preparación del antígeno pullorum polivalente "K" standard no ha tenido ninguna revisión desde su propuesta en 1947 por MacDonald (10). El control de autoaglutinación recomendado se basa únicamente en la observación microscópica de la homogeneidad del antígeno. Una prueba mas sensitiva la constituye el test de Pampana o de la acriflavina por lo que se la incluyó en los estudios de autoaglutinación que aquí se informan.

El problema del amplio mosaico antigénico del antígeno pullorum ha sido convenientemente aborado por Kösters (8-0) quien caracterizó una cepa de S. gallinarum carente del factor "O" 1, con gran componente "O" 9 y una equilibrada composición de los factores "O" 12_2 y 12_3. Esta cepa, TI-184/64 para disminuir las reacciones inespecíficas.

MATERIALES Y MÉTODOS

Cepas utilizadas

Las cepas 11, standard, y 79, variante de S. pullorum y la cepa TI-184/64 (Kösters) de S. gallinarum fueron empleadas para la elaboración de los antígenos experimentales.

Medio de cultivo y método de preparación de los antígenos

Se siguió la técnica propuesta en "Methods for Examining Poultry Biologics" del National Academy of Sciences, Washington 1971, con las siguientes variantes:

- se prepararon tres diferentes soluciones para el lavado y suspensión de las bac-

terias, una con formalina al 1% (solución K original), una con fenol al 0,5% y otra con Merthiolate al 1:10.000.

- Los antígenos suspendidos en las tres diferentes soluciones fueron coloreados con cristal violeta según el método original, o con verde de malaquita a igual concentración. Una tercera alícuota se dejó sin colorear. Luego de una semana a 4°C, los antígenos fueron observados microscopicamente y por la prueba de acriflavina.

Pruebas con suero en placa

63 gallinas reproductoras pesadas con reacciones positivas o sospechosas en la prueba rápida de sangre total con un antígeno pullorum polivalente "K" standard(+) fueron sangradas para la obtención de sueros con los que se realizaron pruebas rápidas en placa comparando al antígeno comercial con los antígenos experimentales inactivados con Merthiolate y coloreados con cristal violeta.

Examenes bacteriológicos

15 de las 28 gallinas con racciones positivas, y

15 de las 35 gallinas con reacciones sospechosas con el antígeno pullorum comercial fueron sacrificadas y examinadas bacteriológicamente mediante cultivos directos en agar McConkey y previo enriquecimiento en caldo selenito, caldo tetrationato y caldo nutritivo. Se cultivaron porciones homogeneizadas de los siguientes órganos: hígado, bazo, ovario y pancreas.

RESULTADOS

Las tres cepas usadas para la elaboración de los antígenos tuvieron un comportamiento similar. A la observación microscópica todos los antígenos mostraron un aspecto homogéneo, sin presencia de grumos ni formas filamentosas.

Con la prueba deacriflavina se puso en evidencia que la formalina provoca modificaciones en las bacterias que favorecen su autoaglutinación.

Cuadro Nro. 1: Efecto de diferentes colorantes e inactivantes sobre la autoglutinación de antígenos de S. pullorum y S. gallinarum

Inactivante / Colorante	Formalina		Fenol		Merthiolate	
	$A/1_b$	$A10_c$	A1	A10	A1	A10
Cristal violeta	++++	++	-	-	-	-
Verde de Malaquita	++++	++	-	-	-	-
---	++++	++	-	-	-	-

b = Acriflavina 1:1.000
c = " 1:10.000

El fenol no produjo ese efecto, pero en cambia decoloró notablemente tanto al cristal violeta como al verde malaquita.

Los antígenos coloreados con cristal violeta tuvieron una coloración mas intesa y de mejor contraste quelos coloredos con verde de malaquita.

Los antigenos inactivados con Merthiolate y coloreados con cristal violeta resultaron los de mejor calidad según los parámetros establecidos en este ensayo.

En el Cuadro Nro. 2 se muestran los resultados de lasreacciones rápidas con suero frente al antígeno comercial de referencia y los distintos antígenos experimentales.

Se puede observar como el antígeno variante de S. pullorum con su elevado contenido en factor "O" 12_2 ha sido el responable de la mayoría de las reacciones inespecíficas, en tanto que el antígeno con la cepa TI-184/64 (Kösters) de S. gallinarum dió la menor cantidad de estas reacciones. Sin embargo, cuando este mismo antígeno ha sido empleado en lotes naturalmente infectados con S. gallinarum, el porcentaje de reactores y la rapidez e intensidad de las aglutinaciones han sido mayores que con el antígeno pullorum polivalente "K" standard.

(+)Salsbury Laboratories - Charles City, Iowa. Acriflavina + 1:10.000.

Cuadro Nro. 2: Aglutinación rápida en placa de sueros de aves con antígeno pullorum polivalente :K: standard y 3 antígenos experimentales monovalentes inactivados con Merthiolate.

Antígenos / Sueros	Polivalente "K" standard	Monovalente cepa 79 variante	Monovalente cepa 11 standard	Monovalente cepa TI-184/64
Positivos	28	9	4	0
Sospechosos	35	40	16	8
Negativos	-	14	43	55

Los estudios bacteriológicos de las 30 aves seleccionadas fueron negativos para S. pullorum y S. gallinarum, resultado que se correlaciona con la ausencia de evidencias clínico-epizootiológicas en los planteles estudiados.

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TIFOSIS AVIARIA EN LA REPUBLICA ARGENTINA.

A.D. Colusi* con la colaboración de R.O. Romano y Dr. J.C. Manetti

*Cervino No.3953, Buenos Aires, Argentina

RESUMEN:

Luego de un brote de Tifosis en un criadero con 100.000 aves en producción - a jaula (50% Leghorn y 50% AA-Harco), un 8,38% de positivos (Harco)a la prueba de aglutinación rápida, son trasladados a tinglado individual y alojados igualmente en jaula. Luego de 50 coprocultivos negativos, los animales son - reanalizados con la misma prueba 30-6-90 y 120 días después,comprobándose que dichos positivos disminuían del 100 al 32%, no habiéndose constatado en todo ese tiempo, diferencia con su lote similar.
Transcurrido ese período, 30 animales son descargados con cepa virulenta,comprobándose que sólo el 1% muere por Tifosis,frente al lote testigo donde los insucesos son del 50%. Estos hechos son analizados estadísticamente entendiendo que la prueba de aglutinación no debe aplicarse con el mismo criterio que para la pullorosis ya que, según los resultados, muestra un valor muy relativo. Se efectúan paralelamente, consideraciones inmunológicas sobre la posible importancia de la Ig A en los mecanismos de defensa de las aves.
Utilizando la cepa rugosa 9-R de Salmonella gallinarum, fue preparada vacuna activa en suspensión y concentrada a razón de 4 x 10^8 viables/ml.
La aplicación de la misma efectuada inyectable 0.5 ml. subcutánea y administrando 0.5 ml. por vía oral simultáneamente.
Los ensayos experimentales con aves susceptibles, demostraron una notoria ventaja de esta nueva técnica contra la clásica subcutánea.
De un total de 800,000 aves vacunadas, se exponen resultados a campo en criaderos con seguimento y control. Ellos demostraron que:
a) La vacuna aplicada en recría confiere una alta protección en lotes ingresados a criaderos infectados.
b) En brotes severos-aún en postura- su aplicación interfiere sensiblemente - el curso de la enfermedad, disminuyendo los porcentajes de morbi/mortalidad.
Tras un planteo epidemiológico realista del país, se efectúan consideraciones sobre la utilidad demostrada por este inmunógeno como una respuesta de primera etapa, con el objetivo mediato de iniciar una disminución en los guarismos de prevalencia de la tifosis aviaria.

FOWL TYPHOID IN REPUBLIC OF ARGENTINA.

SUMMARY:

After a Typhoid outbreak in a farm with 100,000 birds in production in cages (50% Leghorn and 50% AA-Harco),8.38 % of positive reactors to the quick aglutination test (Harco) were transfered to an individual house also in cages. - After 50 negative coprocultures, the birds are again assayed by same test 30-60-90 and 120 days after, finding that said positives decreased from 100 to 32%. No difference was noted during that period with similar lot. After that time, 30 birds are challenged with a virulent strain and only 1% die of - -- Typhoid; as compared with the control group where mortality was 50%.
These facts are statistically analized finding that the aglutination according to the findings, its value is quite relative.
At the same time, immunological considerations were effected on the possible importance of Ig G and Ig A in the defense mechanisms of birds against infections caused by enterobacteria.
Using a rough 9-R strain of Salmonella gallinarum, a liquid media live vaccine was prepared and concentrated at a rate of 4 X 10^8 viable/ml.
The vaccine was apllied by injection of 0.5 ml. subcutaneously and administering another 0.5 ml. orally simultaneously.
Esperimental test with susceptible birds showed a remarkable advantage of this new technique as compared to the previously used subcutaneous. From a total of 800,000 vaccinated birds, field findings in farms subject to follow-up and -- control showed that:
a) The vaccine applied in the growing period provides high protection in lots housed in infected farms.
b) In severe outbreaks even during the laying period application of the vaccine sensibly interferes the course of the disease, decreasing the morbility and - mortality percentages. After a realistic epidemiological analysis of the country,considerations are made regarding the effectivity showed by this immunogenic agent as an answer in a first stop-towards the mediate final objective of decreasing the number of cases of avian typhoid.

Some measures of the behavior of Smith strain vaccine (9R)
Salmonella gallinarum in chickens.

E.N. Silva*, G.H. Snoeyenbos, Olga M. Weinack and C.F. Smyser

Department of Veterinary and Animal Sciences
University of Massachusetts
Amherst, Massachusetts 01003

These experiments were conducted to explore the mechanism, capabilities and limitations of immunization of chickens with the live rough strain of *S. gallinarum* (9R). The vaccine partially protected chickens against experimental exposure. Subcutaneous administration was superior to oral administration. Addition of an oil adjuvant did not improve protection. Vaccination did not protect against paratyphoid infections.

The vaccine strain produced hepatic and splenic lesions without mortality in meat-type and brown egg egg-type chicks but not in Leghorns; it was not recovered from Leghorns as long following vaccination as from the other types of chicks.

All subcutaneously vaccinated chickens developed antibodies detected by the microantiglobulin (MAG) test. Some developed antibody levels which resulted in 4+ reactions to the whole blood test and to the tube test at a serum dilution of 1:25. Antibody levels were not related to protection which is probably dependent on cellular immunity.

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*FAPESP post-doctoral fellowship. Present address: Fac. Med. Vet. Zootecnia, Univ. de Sao Paulo, Cidade Universitaria, ASO 05508 Sao Paulo, SP - Brazil.

Algunos parámetros del comportamiento de la vacuna, cepa
Smith (9R), *Salmonella gallinarum* en pollos.

Estos experimentos fueron conducidos para explorar el mecanísmo, posibilidades y limitaciones de inmunización de pollos con la cepa viva rugosa de *S. gallinarum* (9R). La vacuna protegió parcialmente a los pollos contra la infección experimental. La administración subcutanea fue superior a la administración oral. La emulsión de la vacuna en aceite adyuvante no incrementó proteción. La vacunación no protegió contra infecciones paratifoideas.

La cepa de la vacuna produjo lesiones hepáticas y esplénicas sin mortalidad en pollos bebé de carne y en pollos bebé de lineas ponedoras de huevos rosados, sinembargo pollos bebé Leghorns no mostraron las mismas lesiones. La recuperación de la cepa, post-vacunación, fue posible durante un periodo más corto en pollos Leghorns que en las ostras lineas de pollos.

Todos los pollos vacunados subcutaneamente desarrollaron anticuerpos que fueron detectados por el test de microantiglobulina (MAG). Algunos pollos desarrollaron niveles de anticuerpos que resultaron en reacciones 4+ en el test the sangre entera y en el test de tubo con suero diluido a 1:25. Los niveles de anticuerpos no estaban relacionados con la protección, la qual es probablemente dependiente de la inmunidad celular.

SOME MEASUREMENTS OF THE BEHAVIOR OF SMITH STRAIN VACCINE (9R) SALMONELLA GALLINARUM IN CHICKENS.

E.N. Silva*, G.H. Snoeyenbos, Olga M. Weinack and C.F. Smyser
Department of Veterinary and Animal Sciences
University of Massachusetts
Amherst, MA 01003

Fowl typhoid has dramatically increased in parts of Latin America as well as other areas of the world. In areas where the disease is common, it has become one of the most important diseases of chickens. Although once common in some areas of the United States, it has been reduced by sanitary measures to the point of rare occurrence.

Evidence indicates that killed vaccines are of little value in control. A live avirulent rough strain (9R) of S. gallinarum (6) has been used extensively to protect poultry against fowl typhoid (1,4). This vaccine (9R) has been used subcutaneously either from broth culture (2,3,6) or reconstituted from a freeze-dried state (5,7). It stimulates better immunity when used at 8 weeks of age than at 4 weeks (2) and has not been shown to affect egg production (6).

The experiments reported here were designed to explore the mechanism, capabilities and limitations of immunization of chickens with the rough strain of S. gallinarum-9R.

Table 1 shows that subcutaneous administration of 9R produced more consistent systemic infection than if administered orally. The infectivity of 9R seems to be determined by the genetic susceptibility and age of the host. White Leghorns had fewer colony-forming units (CFU) of 9R in the spleen, lower fecal excretion, and a shorter period of systemic infection than a brown egg egg-producing strain following day-old subcutaneous vaccination. Fecal excretion of 9R was observed for at least 5 weeks following day-old vaccination. Excretion time was reduced to 24 hours in meat-type birds orally inoculated at 8 weeks of age. No fecal excretion was found in 10-week-old White Leghorns or one-year-old meat-type birds inoculated subcutaneously.

Table 1.

Fate of 9R strain in meat-type chicks inoculated either subcutaneously or orally with 10^7 CFU of S. gallinarum.

Time PI[A]	Blood[B]		Liver[C]		Intestine[D]	
	Subcut.	Oral	Subcut.	Oral	Subcut.	Oral
3 h	$<10^1$	0	10^2	0	+	+
24 h	10^1	$<10^1$	10^3	10^1	+	+
7 d	10^2	10^3	10^4	10^4	+	+
21 d	$<10^1$	0	10^4	0	+	-
35 d	10^1	0	10^4	10^1	+	-

[A]h = hour; d = day
[B]Collected by heart puncture. Results in number of C F U/ml.
[C]Number of CFU per organ.
[D]Positive in one or more segments of the intestinal tract.

No mortality was attributed to 9R vaccination in several trials using more than 400 chicks. However, meat-type and brown egg egg-producing strains consistently showed gross lesions characterized by grayish white foci in the liver and spleens for two to three weeks after day-old vaccination. Similar lesions did not occur in White Leghorn chicks similarly vaccinated.

Several trials were conducted to evaluate the protective effect of 9R among different breeds measured by mortality and liver culture of survivors. The results are presented in Table 2. The vaccine was injected into the lower neck region or by oral deposition into the esophagus with a pipette. The chicks were challenged with a pathogenic S. gallinarum strain. Birds which died, as well as survivors,

*FAPESP Post-doctoral fellowship. Present address: Fac. Med. Vet. Zootecnia, Univ. de Sao Paulo, Cidade Universitaria ASO, 05508 Sao Paulo - SP, Brazil.

were necropsied and cultured for salmonella.

A single dose of 9R injected at one-day of age gave good protection as measured by both mortality and systemic infection. The challenge strain was not sufficiently pathogenic to kill significant numbers of birds among the Leghorn test groups but protection was indicated by a decrease in systemic infection. Oral vaccination produced moderate protection.

Neither oral nor subcutaneous vaccination with 9R was protective against subsequent challenge by S. typhimurium or S. infantis as measured by fecal excretion.

Table 2.

Protector effect of 9R vaccination, measured by mortality and liver culture of survivors, among birds challenged with a pathogenic S. gallinarum strain.

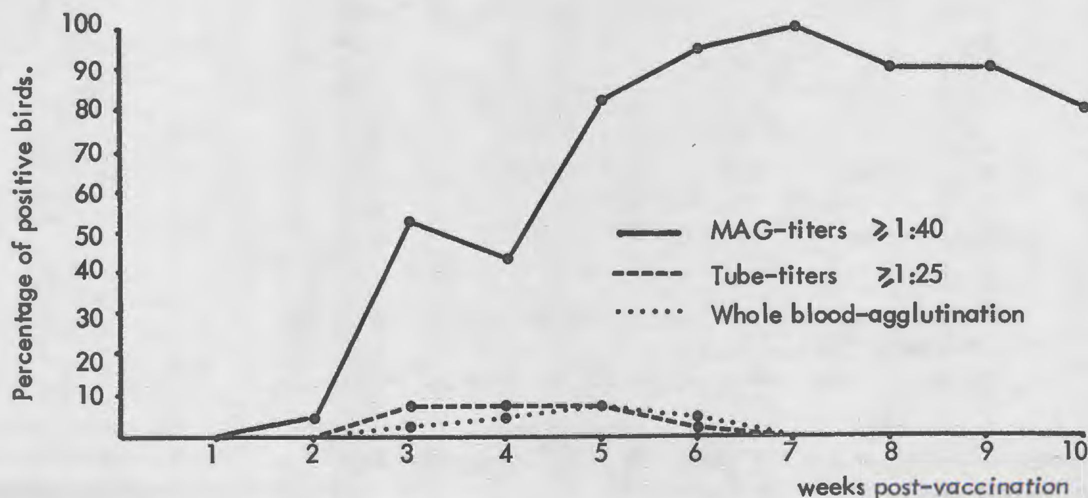
Trial	Chicken	Vaccination		Mortality		Surv. liver cult.	
		Age	Route	Vacci.	Control	Vacci.	Control
6	Meat-type	8 weeks	Subcut.	1/20[A]	5/20	0/19[B]	3/15
9	Meat-type	1 yr.	Subcut.	15/42	41/44	8/27	0/3
7	Brown-egg	day-old	Subcut.	12/24	18/18	4/12	-
7	W. Leghorn	day-old	Subcut.	0/22	1/18	3/22	4/17
5	W. Leghorn	10 wks.	Subcut.	0/20	1/20	1/20	11/19
5	W. Leghorn	10 wks.	Oral	0/20	1/20	3/20	11/19
6	Meat-type	8 wks.	Oral	4/20	5/20	0/16	3/15

[A]Number dead/number challenged [B]Number positive/number cultured.

The microantiglobulin (MAG) test was much more sensitive for the detection of antibodies from vaccinated birds than was the rapid whole blood agglutination (WB) or by pullorum-typhoid standard tube agglutination (T) tests as shown in Fig. 1. Antibodies were initially detected by the MAG test two weeks post-vaccination and the percentage of positive birds increased for more than 10 weeks after a single subcutaneous dose. Some of the vaccinated birds gave 4+ reactions to the WB test and T test at a serum dilution of 1:25 between two and seven weeks post-vaccination. This pattern of antibody response was seen in 10 week-old White Leghorns and in 8 week-old meat-type birds.

Figure 1.

Whole blood agglutination, the standard tube agglutination, and microantiglobulin test results from White Leghorns vaccinated subcutaneously with 9R strain at 10 weeks of age.



To compare 9R and oil emulsion vaccines, four groups of one-year old chickens were used. These birds were subcutaneously vaccinated with 9R vaccine, 9R oil emulsion vaccine (9ROEV), inactivated oil emulsion vaccine (IOEV) or were not vaccinated. The IOEV was prepared from a smooth pathogenic strain of S. gallinarum and was inactivated with B-propriolactone. Both 9R OEV and IOEV were aqueous suspensions homogenized in an oil emulsion of Arlacel-80 and Marcol oil.

Approximately half of the birds vaccinated with these oil adjuvanted preparations showed marked subcutaneous swelling of the head and neck for approximately three weeks post-vaccination.

The vaccines partially protected chickens against experimental exposure (Table 3). The 9R vaccine protected better than the IOEV. Addition of oil adjuvant did not improve 9R protection. Mortality in 9R and 9R OEV groups occurred primarily within two weeks post-challenge, but continued longer in the IOEV group. Classical lesions of systemic fowl typhoid were found in dead birds.

Neither vaccine nor challenge strains were isolated from more than 1,000 eggs cultured after challenge. Among 45 survivors of the groups vaccinated with 9R or 9R OEV, the challenge strain was isolated from the ovary of 8 and 9R from the ovary of one bird 7 weeks post-challenge.

All vaccinated birds developed antibodies detected by the MAG test (Fig. 2). The IOEV produced the highest and most uniform antibody response. The 9R OEV produced the higher and more persistent antibody response than 9R vaccine. Antibody levels were not related to protection which is probably dependent on cellular immunity.

Even with vaccines, the main goal must be the production of S. gallinarum-free flocks by good sanitary measures.

Table 3.

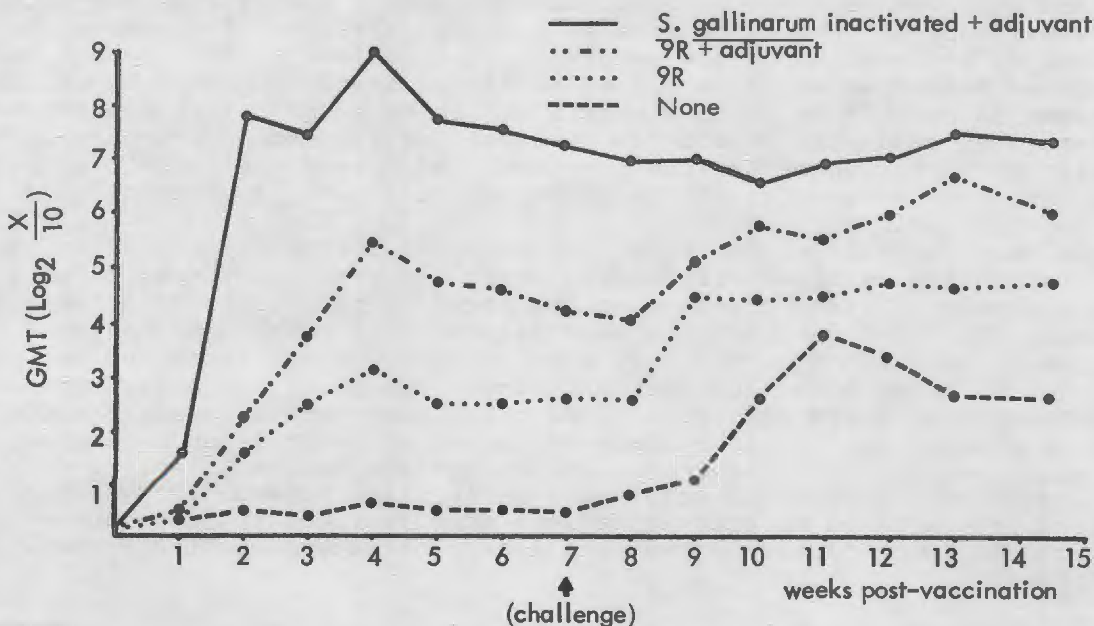
Mortality and egg culture results after challenge of one-year-old chickens vaccinated with 9R and oil emulsion vaccines.

Vaccine	Mortality (weeks post-challenge) 1	2	3	4	5	6	7	Total mortality	Egg cult. results
9R	10	4	0	1	0	0	0	15/42 (36)[A]	0/338[B]
9R OEV	11	12	0	0	0	1	0	24/44 (55)	0/237
S. gallinarum IOEV	5	8	2	2	2	0	1	20/46 (43)	0/379
Control	20	10	5	5	1	0	0	41/44 (93)	0/120

[A]Number dead/number challenged; and ( ) percentage.
[B]Number positive/number cultured.

Figure 2

MAG GMT of one-year-old chickens vaccinated with 9R and oil emulsion vaccines and challenged with a pathogenic S. gallinarum strain.



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ROTAVIRUS INFECTIONS OF TURKEYS AND CHICKENS

J. B. McFerran, Veterinary Research Laboratories
Stormont, Belfast, BT4 3SD, N. Ireland

Rotaviruses have recently been isolated from turkeys and chickens. Field and experimental evidence suggests that these viruses are an important cause of diarrhoea, poor or abnormal appetite and increased mortality, particularly in turkey poults. Experimental infections have shown that the virus grows in the villous epithelial cells of the small intestine.

Diagnosis is based on detection of the virus in faeces by direct electron microscopy, as these viruses are difficult to isolate and grow in cell cultures, sometimes requiring trypsin treatment for successful propagation. So far we have made 6 rotavirus isolates from turkeys and 2 from chickens. Most of these viruses were isolated in chick embryo liver cell cultures from the faeces of birds aged under one week. On the basis of serum neutralisation tests, 7 of the 8 isolates were grouped into 3 serotypes; the remaining isolate was an intermediate strain. Therefore it is unlikely that a vaccine incorporating only one serotype will protect birds from infection with different serotypes. Polyacrylamide gel electrophoresis of the viral RNA revealed differences between each of the 3 serotypes. Application of this technique to faecal virus may provide a rapid and relatively simple method of detecting new serotypes without having to isolate the virus in cell cultures.

Immunofluorescence tests showed that avian and mammalian rotaviruses share a common group antigen. Rotavirus antibody in avian sera can therefore be detected and quantified by indirect immunofluorescence tests using as antigen cell cultures infected with any of the available cell culture-adapted mammalian rotaviruses. Similarly growth of non-cytopathic avian rotavirus in cell cultures can be detected by immunofluorescent staining using antiserum to mammalian rotavirus.

INFECCIONES POR ROTAVIRUS EN PAVOS Y GALLINAS

Estos Rotavirus se han aislado recientemente de pavos y gallinas. Experimentos de campo y laboratorio indican que causan diarreas,baja de apetito o apetito anormal y aumento de mortandad principalmente en pavipollos. Estos virus se replican en las células epiteliales de las vellosidades intestinales.
El diagnóstico se hace por microscopía electrónica a partir de las heces,ya que es bastante difícil su cultivo en tejidos celulares; frecuentemente se requiere el tratamiento de las muestras con tripsina. Se han hecho 6 aislamientos de Rotavirus en pavos y 2 en gallinas, siendo logrados en células - hepáticas de embrión de pollo a partir de las heces de pollitos menores de una semana de edad. Las pruebas de virus neutralización indicaron que 7 de 8 aislamientos pertenecen a tres serotipos; el octavo aislamiento correspondió a una cepa intermedia. Por lo tanto, se dificulta la protección de aves utilizando un solo serotipo.
Observaciones hechas del RNA viral por medio de electroforesis en gel de poliacrilamida han revelado diferencias entre los tres serotipos. Si se aplica esta prueba al virus presente en las heces, será posible detectar nuevos serotipos sin tener que recurrir al aislamiento en cultivo de tejidos.
Las pruebas de inmunofluorescencia han indicado que los rotavirus del hom-- bre y los animales poseen un antígeno común. Por ello, los anticuerpos contra Rotavirus en suero de gallinas pueden ser detectados y cuantificados por medio de pruebas indirectas de inmunofluorescencia, utilizando como antígeno a cultivos celulares infectados con cualquiera de los Rotavirus de mamíferos, adaptados a cultivos celulares. De la misma manera, se pueden detectar Rotavirus aviares no citopatogénicos en cultivo de tejidos, por medio - de tinciones inmunofluorescentes, utilizando antisueros contra Rotavirus de mamíferos.

Traducción: Cortesía del Dr. Armando Antillón Rionda.

TURKEY AND CHICKEN ROTAVIRUSES

M.S. McNulty, M.V.B., Ph.D., M.R.C.V.S. and
J.B. McFerran, B.Sc., Ph.D., M.R.C.V.S.
Veterinary Research Laboratories, Stormont,
Belfast, BT4 3SD, Northern Ireland

Rotaviruses have been isolated from turkeys and chickens only very recently (1,2). Rotavirus infection in these species is associated with signs of enteric disturbance e.g. diarrhoea, poor or abnormal appetite and increased mortality (2). At necropsy, the intestinal and caecal contents are often abnormally fluid and gassy.

As these viruses are difficult to isolate in cell cultures, direct electron microscopic examination of faeces is the best method for diagnosis. Better results are obtained by examination of the pooled contents of the caeca and large intestine than by examination of small intestinal contents. To facilitate recognition of the virus, faecal material should be partially purified by fluorocarbon extraction and concentrated by ultracentrifugation (3).

To date we have made 6 cell culture adpated isolates of rotavirus from turkeys and 2 from chickens (2). Three of these required trypsin treatment for isolation and serial passage, the remainder grew without trypsin. However, even the trypsin independent strains grew to higher titre when trypsin treated. Most of our isolates were made in chick embryo liver cells from the faeces of birds under 1 week of age. Not all strains are cytopathic and it is best to monitor virus growth by immunofluorescence. As avian and mammalian rotaviruses share a common group antigen (1,2), antisera to mammalian rotaviruses may be used for this purpose if antiserum to avian rotavirus is not available. Similarly avian sera may be examined for antibody to rotavirus by indirect immunofluorescence using cell cultures infected with mammalian rotaviruses as antigen (1).

By serum neutralisation tests, 7 of our 8 avian rotaviruses were grouped into 3 distinct serotypes. One was an intermediate strain. Avian rotaviruses possess a double-stranded RNA genome consisting of 11 segments, which can be separated by polyacrylamide gel electrophoresis (4). Individual RNA segments from different isolates frequently show different electrophoretic mobilities, thereby allowing isolates to be classified electrophoretically as well as serologically. Interestingly, all the avian isoaltes which were serologically distinct were also electrophoretically distinct and 5 isolates which were serologically identical were also electrophoretically identical (2).

Experimental infection of chickens and turkeys with these viruses has shown that viral replication occurs in the villous epithelial cells of the small intestine. Clinically the effects of infection were mild. Abnormal amounts of gas, particularly in the caecal tubes, were seen for several days following infection, but faeces were soft or fluid or for only about 1 day.

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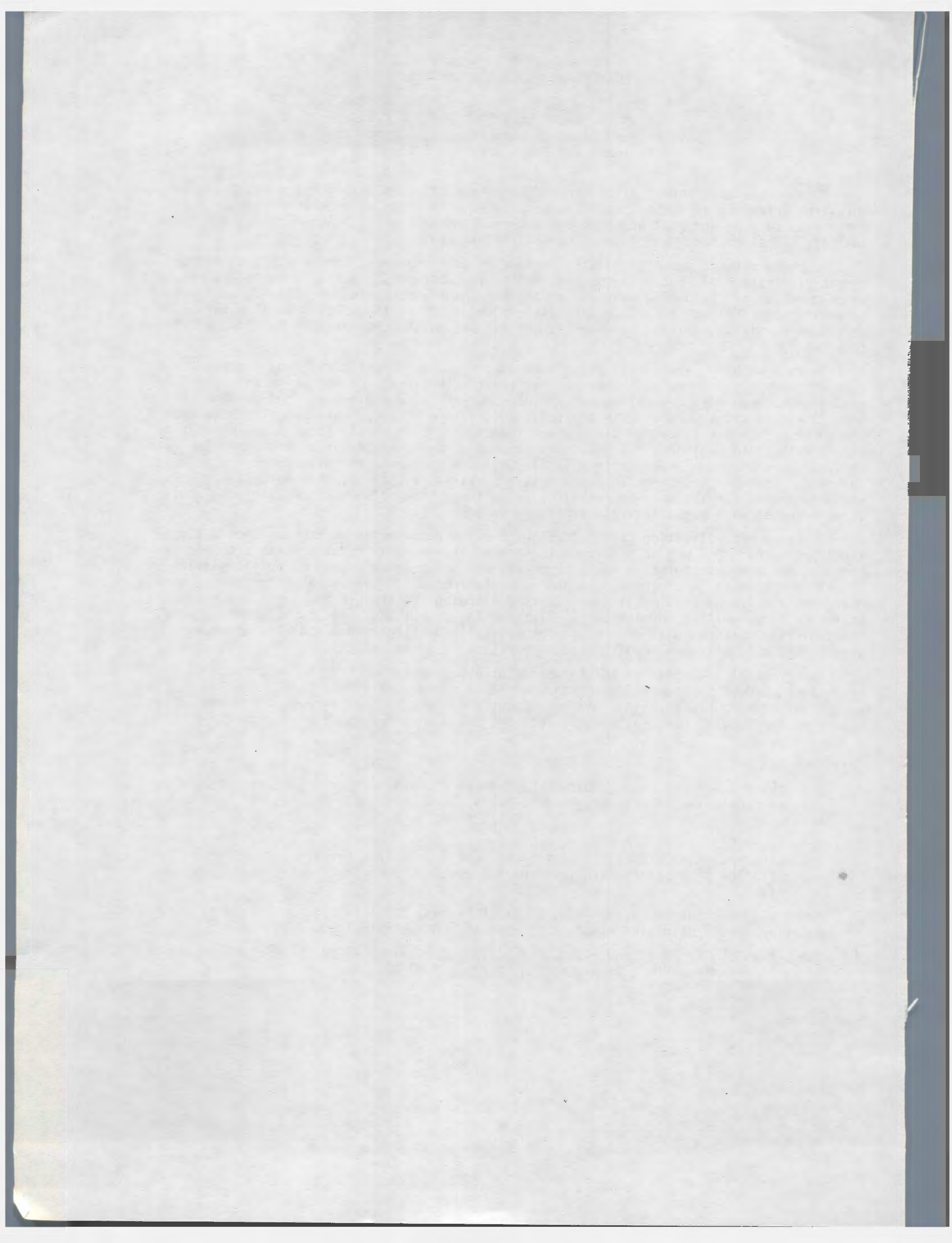

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