

New IBV Variant Designated DMV/1639

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Avian infectious bronchitis virus (IBV) causes an economically significant upper respiratory tract disease in chickens. Like most RNA viruses, IBV is genetically diverse due to a high mutation rate and then selection of these variants through host fitness and immune pressure. New IBV variants are continuously emerging, which complicates vaccination-based infectious bronchitis (IB) control. The most recent variant of IBV, DMV/1639, was originally isolated and characterized on the Delaware, Maryland, Virginia (DelMarVa) peninsula in 2011, but did not cause significant economic losses in the broiler industry until the winter of 2014/2015. In these early infections, the main presentation was nephritis and urate deposition in the kidneys (nephropathogenic disease). This led to severe flushing and dehydration, which was compounded by birds huddling in the chicken house, hypothermia from damp, cool litter/feces, and secondary bacterial infections. In total, both mortality and condemnation were significantly increased. Disease caused by DMV/1639 began to decline in broilers in 2016 and 2017 in the US, but in 2017 the layer industry in Canada was significantly affected with false layer syndrome. False layer syndrome is characterized by mature layer hens that never come into production due to the oviduct developing into a large, fluid-filled cyst from damage to the reproductive tract caused by IBV infection early in life. At the time, IBV was suspected but DMV/1639 was not implicated until later. Also, in 2017 and 2018, cases of false layer syndrome and cystic oviduct development were seen in the US where DMV/1639 type IBV was detected in pullet flocks. By 2019, detections of DMV/1639 began to increase in broilers again, particularly on the DelMarVa peninsula, but the clinical presentation had shifted from nephropathogenic to classical respiratory disease. At that time, the first detections in broilers outside of the DelMarVa peninsula were also recorded in Virginia, Georgia, and Arkansas. To date, DMV/1639 has been detected in nearly all commercial poultry producing regions in the US, including DelMarVa, Ohio, Virginia, the Carolinas, Tennessee, Kentucky, Georgia, Florida, Alabama, Mississippi, Louisiana, Texas, Arkansas, and Missouri.

As the DMV/1639 virus spread across the US, the pathogenicity changed from nephropathogenic with some respiratory involvement to strictly respiratory in nature. During this time, the S1 amino acid sequence changed as well, which may, at least partially, explain the shift in pathogenicity. Phylogenetic analysis shows that sequences from 2015 isolates that were nephropathogenic in nature cluster closely (95-96% similar) with the first isolates identified outside of the DelMarVa region (mainland VA). As the virus continued to spread, the more recent sequences from viruses isolated in late 2018-2021 are more distant from the earlier isolates, but very closely related to each other. Interestingly, a Canadian isolate from a case of false layer syndrome in 2018 seems to be an intermediate between the nephropathogenic and respiratory isolates. It should be noted that the sequence identity between the original 2015 and a current 2022 isolate is 93%. It cannot be completely concluded from this data that these changes in S1 are the sole cause of the shift in pathogenicity, and analysis of the whole genome sequence should

be conducted on these isolates to determine if any other amino acid changes occurred in portions outside of S1. But with our current understanding of IBV these data do present a compelling relationship between S1 amino acid sequence and virus phenotype.

Control of DMV/1639 has been attempted using several vaccines, with Mass and GA08 type being the most common. For the most part, using these vaccines alone or in combination will reduce or eliminate clinical signs of disease and can reduce viral load shed into the environment. They do not, however, completely prevent infection nor are they capable of completely stopping viral shed and transmission. Surveillance of broilers across the Southeastern US from 2020 to present has shown that using these vaccines will influence the DMV/1639 infection profile, namely pushing the infection window to a point later in the life cycle of the broiler. After extended use, the infection window for the DMV virus will begin to move forward again, back to the typical 3-4 weeks of age. Even with this shift in infection range, often clinical signs do not reappear, especially in warmer climates. A homologous DMV/1639 live attenuated vaccine is in development but is not currently commercially available. It will be interesting to see how continual use of non-homologous vaccines influences viral evolution of DMV, and if a homologous vaccine will influence it any differently.