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Avian Bornavirus

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Proventricular dilatation disease (PDD), formerly known as "macaw wasting disease," is one of the most important psittacine diseases. PDD has been observed in many psittacine species and large parrots seem to be most frequently affected. Therefore, the disease is a problem for aviculturists, bird breeders and conservation programs for endangered species. PDD is caused by a non-suppurative inflammation of nerves of the gastrointestinal tract, as well as the central and peripheral nervous system (Gregory et al., 1996). The clinical signs can be nonspecific like fluffing, sleepiness and weight loss. The gastrointestinal signs include anorexia, regurgitation, diarrhea and passage of undigested seeds in faeces. Seizures, tremor and ataxias can occur as neurological signs alone or in combination with the other clinical signs. The disease always leads sooner or later to death (Berhane et al., 2001; Lierz, 2005; Lublin et al., 2006). At this point no treatment is known for PDD. Alleviation of clinical signs by using anti-inflammatory drugs renders no measurable success (Lublin et al., 2006).

Avian bornavirus was discovered in 2008 and has been proved to be the aetiologic agent of proventricular dilatation disease, a threatening disease for psittacine and other bird orders worldwide (Honkavuori et al., 2008; Piepenbring et al., 2012). So far, eight genotypes are known in psittacine birds. Due to a reorganization of the taxonomy of the family Bornaviridae, avian bornavirus is renamed for parrots into the species Psittaciform 1 bornavirus with the virus parrot bornavirus (PaBV) (Kuhn et al., 2015). Psittaciform 1 bornavirus includes ABV 1, 2, 3, 4, 7 - ABV 5 and 6 remain unassigned as available sequences and the absence of isolates from those genotypes does not allow classification so far. Only recently, a distinct ABV has been detected in captive psittacine birds in Brazil, named parrot bornavirus 8 (PaBV-8) forming a separate branch within Psittaciform 1 bornavirus species (Philadelpho et al., 2014).Avian Bornaviruses were also detected in other bird orders like finches, canaries, waterfowl and Galliformes (Weissenböck et al., 2009; Delnatte et al., 2011; Payne et al., 2011; Guo et al., 2012; Rubbenstroth et al., 2013, 2014; Bourque et al., 2015). Even though the Henle-Kochs Postulates were fulfilled (Piepenbring et al., 2012), many questions are still unanswered. In flocks several birds are carriers without clinical disease, others demonstrate severe clinical signs and birds with close contacts are not getting infected (Heffels-Redmann et al., 2011, 2012). In routine diagnosis a PCR for the detection of ABV-RNA (Honkavuori et al., 2008) and a serology (Herzog et al., 2010) for the detection of ABV-specific antibodies are performed demonstrating sometimes contrary results creating problems when explaining this to bird owners. Infectious trials were able to demonstrate that different ABV- genotypes causes different clinical outcomes which might be able to explain some of the contrary test results. In particular, ABV-2 caused a variety of clinical courses in experimentally infected cockatiels and viral distribution in organs and success of re-isolation differed depending on the time point of death. In contrast, ABV-4 causes a more constant clinical picture (Piepenbring et al., 2016). The oronasal route of infection is regularly assumed in the literature. In the latest infection trials, oral and nasal transmissions of PaBV, as well as application on mucosal lesions, are not leading to a valid infection with the virus. Vertical transmission is still suggested as an infection route (Lierz, et al., 2011). This has influence on flock management in ABV- affected collections. The factors responsible to cause a clinical disease from infection are presently unknown, even apart from viral factors, host factors seem to play an important role. So far, it is very likely that ABV triggers an immune disease.

For more detailed information please see:

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