**Sarcocystis calchasi** - a New Threat for Pigeons and Psittacine Birds

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*Sarcocystis calchasi* is the causative agent of Pigeon Protozoal Encephalitis (PPE) a severe neurologic disease with high mortality rates in domestic pigeons. From 2006 to 2008, *S. calchasi* was recognized for the first time in Northern Germany in several pigeon flocks with neurologic signs (e.g. head tilt, ataxia, torticollis, paresis and paralysis) and losses of up to 25 (Olias et al., 2009b). Infections with PMV-1 and Salmonella spp. which represent the major differential diagnosis had been excluded. Sarcocysts were detected in the skeletal muscle of the affected pigeons and a granulomatous encephalitis was present (Olias et al., 2009a). Subsequently, European Accipiter hawks (Northern goshawks (*A. gentilis*)) and sparrow-hawks (*A. nisus*) were identified as definitive hosts of *S. calchasi* (Olias et al., 2011). Dogs (*Canis lupus familiaris*), ferrets (*Mustela puterius furo*), rats (*Rattus norvegicus domesticus*), mice (*Mus musculus domesticus*) and Gyr-Saker hybrid falcons (*Falco rusticolus x Falco cherrug*) did not shed *S. calchasi* sporocysts after experimental infection (Olias et al., 2010a).

Since its description in 2009, *S. calchasi* has been reported also in pigeons with virtually identical PPE in the United States indicating a worldwide distribution of the parasite (Wünschmann et al., 2011; Olias et al., 2014). Recently, several psittacine species have been demonstrated as intermediate hosts of *S. calchasi* in natural and experimental infections (Rimoldi et al., 2013; Olias et al., 2014). The psittacine birds developed a disease that closely resembled PPE in pigeons. Despite the exclusion of chickens as intermediate hosts, *S. calchasi* might be capable of using a broad spectrum of intermediate hosts since psittacines and pigeons are only distantly related.

In the intermediate hosts (pigeons and psittacine birds) a biphasic disease occurred after experimental infection with *S. calchasi* sporocysts (Olias et al., 2010b; Olias et al., 2014). An acute phase with apathy and polyuria began at day 10 post infection (dpi) which was either followed by a complete recovery or death. This phase correlated with the first schizogenic phase of *S. calchasi* infection with asexual replication located mainly in liver and spleen. Central nervous signs like ataxia, head tilt and paresis developed around dpi 50. During this phase, mature sarcocysts were observed in the skeletal muscle and a granulomatous, necrotizing encephalitis was present. In pigeons, the development of clinical signs was dose-dependent. Pigeons with medium infectious doses (103 to 104 sporocysts) demonstrated both clinical phases. Pigeons with low infectious doses (102 sporocysts) developed only a chronic phase with neurologic signs and pigeons infected with high numbers of sporocysts (8 x 104 to 3 x 106) died during the acute phase (Olias et al., 2010b). In contrast, cockatiels (*Nymphicus hollandicus*) developed similar clinical signs of varying severity which was independent of the infectious dose (Olias et al., 2014). This difference between pigeons and psittacine birds was suggested to be the consequence of an evolutionary younger parasite-host relationship in psittacine birds (Maier et al., 2013).

Despite severe encephalitis during the chronic phase of PPE, parasite stages have rarely been identified in the CNS of some experimentally infected animals so far. These schizonts and sarcocysts were not associated with the inflammatory lesions (Olias et al., 2013). In naturally infected pigeons in the USA, however, intraleisional schizonts were detected in some diseased pigeons (Olias et al., 2014). Since re-infection and strain differences cannot be ruled out in those natural infections, the direct role of *S. calchasi* in the development of CNS lesions remains unclear. Other Sarcocystis spp. causing encephalitis in avian species, including the extensively described *S. falcatula*, are usually associated with the schizogenic phase of the parasite’s life cycle. In chickens (Mutalib et al., 1995) a cockatiel (Jacobson et al., 1984), a Northern gannet (Spalding et al., 2002), raptors (Olson et al., 2007; Wünschmann et al., 2009, 2010) and psittacine birds (Villar et al., 2008) numerous schizonts
were identified within the cerebral lesions. Few cases have been reported of Sarcocystis–related encephalitis without associated parasite stages in the neuronal tissue as was seen in S. calchasi infections (Munday et al., 1984; Foreyt et al., 1995). However, evidence on the pathogenesis of the encephalitis in absence of parasite stages is still lacking. It has been demonstrated for S. calchasi that the encephalitis appears already very early after infection and several weeks before the onset of neurologic signs (Maier et al., 2014). A transient or low-grade neuroinvasion of schizonts or a trigger located outside the CNS (either schizonts or immature sarcocysts) have been suggested to induce the encephalitis. An immune-mediated pathomechanism seems very likely since the amount of sarcocysts in the skeletal muscle did not correlate with the severity of the encephalitis (Maier et al., 2014). It was demonstrated, that S. calchasi is capable of modulating the cellular immune response as it is known from its closely related sister taxon S. neurona (Witonsky et al., 2008). During the acute, schizogonic phase Th1-related cytokines interleukin (IL)-12, IL-18 and IFN-γ were down-modulated and the second, neurologic phase was shown to be characterized by an extensive Th1-biased T-cell driven immune response suggesting a T-cell mediated delayed-type hypersensitivity reaction (Olias et al., 2013). However, a direct relation between the activated immune response and the development of the cerebral lesions has not been proven yet.

References


