ABSTRACT

The apicomplexan parasite *Sarcocystis neurona* causes equine protozoal myeloencephalitis (EPM), a degenerative neurological disease of horses. Due to its host range expansion, *S. neurona* is an emerging threat that requires close monitoring. In apicomplexans, protein kinases (PKs) have been implicated in a myriad of critical functions, such as host cell invasion, cell cycle progression and host immune response evasion. Here, we used various bioinformatics methods to define the kinome of *S. neurona* and phylogenetic relatedness of its PKs to other apicomplexans. We identified 97 putative PKs clustering within the various eukaryotic kinase groups. Although containing the universally-conserved PKA (AGC group), *S. neurona* kinome was devoid of PKB and PKC. Moreover, the kinome contains the six-conserved apicomplexan CDPKs (CAMK group). Several OPK atypical kinases, including ROPKs 19A, 27, 30, 33, 35 and 37 were identified. Notably, *S. neurona* is devoid of the virulence-associated ROPKs 5, 6, 18 and 38, as well as the Alpha and RIO kinases. Two out of the three *S. neurona* CK1 enzymes had high sequence similarities to *Toxoplasma gondii* TgCK1-α and TgCK1-β and the *Plasmodium* PfCK1. Further experimental studies on the *S. neurona* putative PKs identified in this study are required to validate the functional roles of the PKs and to understand their involvement in mechanisms that regulate various cellular processes and host-parasite interactions. Given the essentiality of apicomplexan PKs in the survival of apicomplexans, the current study offers a platform for future development of novel therapeutics for EPM, for instance via application of PK inhibitors to block parasite invasion and development in their host.

Keywords: *Sarcocystis neurona*, EPM, apicomplexans, phylogeny, homology modeling