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TOXOPLASMA GONDII-ALTERED HOST BEHAVIOUR: CLUES AS TO MECHANISMS OF ACTION AND IMPLICATIONS FOR ITS ROLE IN SOME CASES OF HUMAN SCHIZOPHRENIA

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Introduction: Recognition of the role of infectious agents in a range of both acute and chronic diseases is increasing. One key example is the potential epidemiological and neuropathological association between some cases of schizophrenia with the protozoan Toxoplasma gondii. T. gondii establishes persistent infection within the CNS and can alter host behaviour. Altered dopamine levels have been reported for both T. gondii infection and schizophrenia. Several medications used to treat schizophrenia demonstrate anti-T. gondii properties, and haloperidol, a dopamine antagonist, can prevent the development of T. gondii-altered behaviour in rodents. Furthermore, T. gondii may actually be a source of dopamine, as it encodes a copy of the mammalian enzyme tyrosine hydroxylase, which represents the rate-limiting step in dopamine synthesis.

Aims: Using the epidemiologically and clinically applicable rat-T. gondii model, and incorporating a battery of classical and novel non-invasive behavioural and physiological assays, we aim to further elucidate the impact of T. gondii on behaviour and the mechanisms involved.

Results: T. gondii increases the rats' propensity for predation risk through enhanced activity, visibility and manipulation of their perception of predation risk, turning innate aversion into a 'suicidal' feline attraction. There is little indication that T. gondii alters the rats generalized anxiety, nor potential to enhanced predation by non-definitive mammalian host species. Preliminary associative analyses into the relationship between individual behavioural alterations and neurotransmitter and brain cysts localisation profiles will be presented.

Conclusions: Our results provide further evidence for a role of T. gondii in the aetiology of some cases of schizophrenia.

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