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NEUROLOGICAL SEQUALAE AND TOXOPLASMA INFECTION: POSSIBLE MECHANISMS TO EXPLAIN ASSOCIATIONS WITH SCHIZOPHRENIA

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Latent infection with the common intracellular protozoan parasite Toxoplasma gondii has been shown to result in altered behaviour of its host. This behaviour manipulation has been proposed to increase predation of the intermediate host (e.g. rodents and birds) enhancing parasite transmission. Other psychologicalsequalae have also been associated with latent toxoplasmosis including human affective disorders, as human are accidental hosts for Toxoplasma. During cyst stages of the life cycle found in the brain and other tissues, there is a complex interaction between the parasite and the host. Our research is concerned with the possibility that the parasite-induced behavioural changes are mediated by neurotransmitters. Potential factors in neurotransmitter levels include the location of the cyst, the host immune response, and direct parasite products. The cyst is found in many brain regions but elevated numbers have been reported in the hippocampus, amygdala, and nucleus accumbens. The host response involves interferon gamma suppressing growth through tryptophan degradation that could decrease serotonin levels. The parasite could directly manipulate the host brain through altering dopamine levels. We have found a change in dopamine associated with infection. Indeed, the parasite itself encodes the rate-limiting enzyme in dopamine synthesis, tyrosine hydroxylase. Dopamine's role is also supported by finding haloperidol, a dopamine antagonist, blocks manipulation of rodents by Toxoplasma. Our current investigations of alterations in neurotransmitter levels during chronic infection and association with brain cysts will be presented. Our data presents an interesting interplay with the 'dopamine hypothesis' that has postulated a link between elevated dopamine and schizophrenia.

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