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Overview of Neurotropism

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Editorial

The presence of melanoma cells around nerve sheaths (perineural invasion) or within nerves indicates neurotropism (intraneural invasion). The tumour may produce neuroid structures on its own (a process known as 'neural transformation,' which is also known as neurotropism). The presence of melanoma cells around nerves in the main tumour mass (which commonly indicates "entrapment" of nerves in the developing tumour) should not be misinterpreted as neurotropism, according to pathologists. Infiltration along nerve sheaths (or, on rare occasions, within the endoneurium) has been linked to a higher rate of local recurrence (local persistence). Neurotropism is more common in desmoplastic melanoma (desmoplastic neurotropic melanoma), however it can also be found in other types of melanoma. The presence of neurotropism is linked to a higher risk of local recurrence and can be addressed in some circumstances.

Parasitic infections of the central and peripheral nervous systems are a major cause of illness and mortality in humans, particularly in low-to-middle-income nations where they are endemic. Cerebral malaria, neurocysticercosis, African and American trypanosomiasis, and toxoplasmic encephalitis are examples of diseases that show a preference for the CNS.Despite the recognised link between parasitic infections and particular neurological, cognitive, and mental diseases, the exact processes by which "neurotropic" parasites I infiltrate the CNS across the Blood-brain Barrier (BBB) and cause neurological harm have yet to be fully understood.

Neuroinflammation and brain damage are interwoven in the research of parasite brain infections. Neuroinflammation serves to protect the Central Nervous System (CNS) from a variety of threats, including invasion and infection by infectious pathogens. Pathogens can take advantage of immune responses to enhance neuroinvasion, such as when neuroinflammation accelerates the opening of physical barriers or when the pathogen uses Trojan horse techniques to infiltrate the brain by utilising activated cells. Pathogens are difficult to dislodge once inside the brain, causing CNS dysfunctions. This is true for a variety of protozoan parasites that cause chronic CNS infections, such as internal Toxoplasma and external African trypanosomes. Fungi including *Cryptococcus neoformans*, *Aspergillus fumigatus*, and *Candida albicans* have been shown to use several of these neuroinvasion pathways.

Toxoplasma infection affects one-third of the world's population. Schlüter and Barragan examine how immune cells can aid in the spread of Toxoplasma and are programmed to spread into the CNS by Toxoplasma infection, as well as how parasite-specific Tcell-mediated immune responses control but do not eliminate the infection in this Research Topic (RT). Furthermore, the function of brain resident cell groups is examined, including how neurons, astrocytes, and microglia serve as both parasite target cells and actively contribute to the immune response.

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There is now mounting evidence that chronic CNS toxoplasmosis may have an impact on neuropsychiatric illnesses or modify cognitive abilities. S. studied transcriptomes from primary human retinal pigment epithelial cells and cell lines infected with T. gondii strains in vitro, focusing on ocular toxoplasmosis. The immunological activation of *T. gondii*-infected retinal pigment epithelial cells, which constitute the blood-ocular barrier, was activated by *T. gondii* infection.

For millennia, Human African Trypanosomiasis (HAT), often known as sleeping sickness, has been recognised as a scourge in Sub-Saharan Africa. *Trypanosoma brucei*, a protozoan parasite, causes the disease, which is a major source of mortality and morbidity in both animals and humans. This parasite causes an early hemolymphatic stage, which is followed by a late encephalitic stage, in which the parasites travel into the CNS. Kennedy and Rodgers offer an overview of the clinical aspects of HAT in this paper, with a focus on the late-stage symptoms of the disease. The molecular aspects of *T. brucei*, new medication options, and diagnostic methods are explored, as well as the epidemiological implications of previously undiscovered asymptomatic patients.

Circumventricular Organs (CVOs), which are neural structures located around the third and fourth ventricles, have vessels devoid of a continuous nonfenestrated barrier, similar to the choroid plexus, which allow them to sense immune-stimulatory molecules in the blood circulation, but may also increase the risk of microbe exposure. Microbe attacks against CVOs, on the other hand, are rarely documented. *T. brucei* circulating in the bloodstream can target the CVOs and choroid plexus, allowing for brain penetration, according to M. Bentivoglio, Trypanosomes can seed into the ventricles from the choroid plexus, causing an increased infiltration of T cells and parasites in the periventricular regions. Trypanosomes can also infect CVOs such as the median eminence in the base of the third ventricle and the BBB-protected hypothalamus arcuate nuclei by crossing the boundary. Sleep/wake behaviour is influenced by activity in arcuate nucleus neurons. The findings reveal that trypanosome invasion of CVOs can contribute to the onset of HAT's initial and unique CNS abnormalities, as well as the processes involved.

Plasmodium, for example, causes profound changes and inflammation in the cerebral vasculature without accessing the brain parenchyma. Cerebral malaria pathogenesis is characterised by the sequestration of Plasmodium-infected red blood cells in brain microvessels. Pais and Penha-Goncalves discuss the importance of innate immune responses in cerebral malaria. They describe how brain endothelial cells' innate immune receptors recognise various parasite-derived molecules, mediating their activation and triggering inflammatory responses that lead to microcirculatory and coagulation disturbances, as well as altered vascular permeability, impairing BBB integrity.

Invasion of cerebrospinal tissues by metazoan parasites has evolved as well. They may infect the CNS through hematogenous spread of larval stages to small vessels, in situ deposition or embolism of eggs following abnormal adult worm migration to the CNS, attachment to the nasal neuroepithelium and penetration via the olfactory nerve pathway, or direct invasion of the neural skull and intervertebral foramina. The most prevalent helminth infection of the CNS in humans is neurocysticercosis, which is caused by blood spread of Tenia solium larval stages (cysticerci). The nature and location of cysts inside the brain parenchyma or in the cerebral ventricles and subarachnoid regions (extraparenchymal) affects the clinical presentation and immunology of neurocysticercosis. Toledo et al. address the importance of identifying sensitive and specific biomarkers that might predict the severity of an inflammatory response to medication treatment in this RT.

Although CNS fungal infections are infrequent, long-term systemic fungus

circulation can impact the CNS, increasing morbidity and mortality in infected patients. This RT includes an original research paper by M. D'Alessandre Sanches et al., who demonstrate the potential of three non-albicans Candida species to spread to the CNS in a mouse model, which is important because these species are becoming more commonly involved in CNS candidiasis infections. They show that Candida glabrata, Candida krusei, and Candida parapsilosis can spread to the CNS and cause local inflammation in both immunocompetent and immunosuppressed rats [1-5].

Parasitic infections have an effect on the peripheral nervous system (PNS), most likely through parasitic compounds (such as Entamoeba cysteine proteases) and/or immunological responses triggered by the infection. The altered activity of peripheral neurons in these illnesses may regulate immune activation via gut-brain communication. When exposed to various enteric protozoan and metazoan parasitic diseases, enteric nervous system distress plays a significant role in the pathophysiology of diarrhoea (i.e. Giardia. sp; *E. histolytica*). Echagasic megasyndromes and post-infectious problems have both been linked to this suffering.The pathophysiology of parasitosis is linked to the functions of systemic innate and adaptive immune responses in the persistence or eradication of parasitic infections in the brain. Understanding these roles will aid in the development of parasite-induced neurological illness prevention strategies. This RT focused on current developments in the pathophysiology of parasites that impact the CNS and PNS, as well as the involvement of immune responses in these infections, with the goal of revealing new therapies.

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