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Short Communication

A Note on Toxoplasmosis and it's Modern Treatments

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DESCRIPTION

One third of the world's population is seropositive for toxoplasmosis, Toxoplasma gondii is among the parasites with the greatest degree of success. While many gaps have been filled in the diagnosis field, in epidemiology, or in the understanding of parasite-cell interplay, few advancements have been made in the treatment of toxoplasmosis. The detrimental effects of primary infection during pregnancy, as well as the reactivation of the disease in immunocompromised patients, are well known and have been known for decades [1]. With an estimated 1.2 million Disability-Adjusted Life Years (DALY) for more than 190.000 annual cases, Congenital Toxoplasmosis (CT) continues to have a significant negative impact on global health. If children who have the infection are left untreated, long-term neurological sequelae and visual impairment may result [2].

Whereas HIV-associated Toxoplasma reactivation has eagerly decreased in high income countries, after the introduction of highly active antiretroviral therapy, it is still very prevalent in low income countries.

There aren't many choices for chemotherapy for toxoplasmosis. Anti-Toxoplasma medications primarily target the folate pathway, which is essential in DNA synthesis and contains the enzymes Dihydrofolate Reductase (DHFR) and Dihydropteroate Synthetase (DHPS). Both of the two main medications used to treat acute toxoplasmosis, Pyrimethamine (PYR) and Trimethoprime (TMP), act on the parasite DHFR but are unable to distinguish it from the human host's enzyme. They must be used in combination regimens with sulfonamides, which suppress DHPS, as they are insufficiently potent when taken alone. Therefore, existing treatment regimens have myelotoxic side effects that necessitate therapy termination or, more frequently, cause noncompliance (not to mention more severe side effects that can be life-threatening) [3].

This is a significant disadvantage because patients with immunodeficiencies and congenitally infected newborns typically require lengthy courses of treatment. Most importantly, no treatment in use today can remove *T. gondii* cysts from an infected host where they are dormant and remain so as long as the immune system is robust enough to prevent their reactivation as tachyzoites [4].

In comparison to poor compliance and the range of adverse effects, drug resistance in *T. gondii* is seen as a minor problem. It has been reported that the long-term PYR-based treatment for Congenital Toxoplasmosis (CT) has failed, potentially as a result of the emergence of a drug-resistant *T. gondii* strain. And from congenitally infected neonates sulfadiazine-resistant *T. gondii* strain is discovered. This finding demonstrates that the development of drug resistance in *T. gondii* is conceivable, while being constrained by the slow parasite multiplication and the disease's transmission methods, both of which preclude the large spread of a resistant strain [5].

CONCLUSION

Therefore, it is crucial to find new, effective candidates that can act on both tachyzoites and cysts and are safe for both pregnant women and newborns to use. In order to lower the amount of cysts, the optimum medication should be bioavailable, concentrate in the placenta while also diffusing into the foetal compartment, pass the blood-brain barrier, and diffuse into the Central Nervous System (CNS) as well as the eye compartment. Current *T. gondii* treatment would be revolutionised by the creation of well-tolerated, non-toxic medications that would stop reactivation, reduce treatment time, or perhaps eliminate chronic toxoplasmosis. The price of such a new drug should also be reasonable so that it can be used in developing nations.

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