

Understanding the Relationship of Chronic *Toxoplasma Gondii* Infection and Schizophrenia



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Abstract

Toxoplasma Gondii is a widely distributed neurotropic protozoan parasite that causes Toxoplasmosis in humans and animals. Approximately, 30-50% populations are infected by this parasite world-wide. After acute infection, *T. gondii* undergoes developmental switching from its highly replicating tachyzoite stage to slowly replicating dormant bradyzoite stage preferentially in brain and skeletal muscle. Therefore, brain and skeletal muscle may act as a reservoir of *T. gondii* for persistent infection. In immunocompromised individual and developing fetus, *T. gondii* bradyzoite can reactivate and thereby can cause severe neurological disease for instance schizophrenia. In this mini-review, we have linked the association of chronic *T. gondii* infection with schizophrenia. This study would help scientist to perform in-depth research on discovering the mechanism of *T. gondii* infection in schizophrenia.

Keywords: *Toxoplasma Gondii*; Brain; Schizophrenia; Immunocompromised patients; Fetus

Introduction

Toxoplasma Gondii is an important neurotropic parasite that can infect any warm-blooded animals including humans and causes Toxoplasmosis. The infection rate varies from 10-90 % world-wide depending on environmental or socioeconomic factors and geographic locations [1]. Due to its wide-spread nature and infection capacity, *T. gondii* has both medical and veterinary importance. After acute infection in human and warm-blooded livestock animals, *T. gondii* accomplishes its asexual stages life cycle through stage conversion of the parasite from fast replicating tachyzoite to slow replicating dormant bradyzoite particularly in brain and skeletal muscle [2]. This stage differentiation of the parasite brain and skeletal muscle permit them for establishing lifelong persistent infection. It has to be stressed that human can become infected primarily by ingesting undercooked or raw meats of infected livestock animals or eating contaminated foods and water [3].

European multicenter case control study showed that 30-63 % of acute infections of pregnant women are due to consumption of infected undercooked meats [4]. In immunocompetent host, primary infection is generally mild with flu-like symptoms whereas *T. gondii* infection can cause life threatening problems in immunocompromised patients for instance AIDS, transplant and cancer [5]. Furthermore, congenital toxoplasmosis can interfere brain development in fetus [6] which may provide significant

impact on severe neurological damage or even death of the fetus. Neurological disorder like schizophrenia is present in ~1% people and ninth most common cause of disability over the world. The symptoms of the disease start from late teens to early adulthood, although the psychotic episodes can persist throughout the entire life of the patient. Till now, no single contributory driving force has been discovered; therefore, it is realistic only to explain some factors that were shown to be positively associated with schizophrenia. The well-known risk factors include: genetic predisposition, neurodevelopmental instability and environmental factors, including infectious agents [7,8]. In this study, we have deciphered the relationship of *T. gondii* infection and common neurological mental disorder schizophrenia.

Impact of *T. gondii* on schizophrenia

Schizophrenia is a chronic and severe neurological disease that affect nearly one percent of adult population world-wide [9]. People with this disease suffer from hallucination (false perception), delusion (beliefs that conflict with reality), depression, apathy, poor social activities and lack of speech [10]. The exact etiological factor for Schizophrenia has not been identified yet but it is widely accepted that mostly genetic and environmental factors are prominent cause for pathogenesis of the disease [11]. Recent evidence suggests that infectious agents can act as a high-risk factor for onset of schizophrenia

[12]. Previous studies have shown that infection with virus e. g influenza, herpes simplex, rubella, polio and varicella zoster may contribute the development of schizophrenia [13].

Recently, *T. gondii*, an important neurotrophic protozoan parasite, has been identified as an important risk factor for developing symptoms of schizophrenia [14]. Thereafter a number studies has investigated the association of *T. gondii* and schizophrenia. has summarized 38 studies of *T. gondii* and schizophrenia and confirmed the increased prevalence of anti-*T. gondii* antibodies in schizophrenia patients (odd ratio 2.73, 95% CI) [15]. For instance, Hamidinejat and colleagues has shown that 57 % patients with schizophrenia carry anti-*T. gondii* IgG antibodies compared to healthy controls [16]. Another study conducted by Mortensen and colleagues suggest that early infection of *T. gondii* in newborn may develop schizophrenia in later stage of life [17]. Contrary, some other studies has failed to reveal the association of *T. gondii* infection with schizophrenia [18-20]. Although the role of *T. gondii* infection in developing schizophrenia is controversial in very few studies, the above majority studies nevertheless, confirm the positive correlation of *T. gondii* infection and development of psychiatric disease like schizophrenia. However, the mechanism of how the parasite develops the disease is remaining unknown.

Conclusion

Despite having few controversial findings, it can be said that there is strong correlation of *T. gondii* infection and schizophrenia. The correlation between *T. gondii* and schizophrenia might be explained in the way as *T. gondii* develop bradyzoite tissue cysts and persist in brain of infected patient's life time, thereby the parasite may somehow modulate neurological parameter which may directly or indirectly involved in that process. Further research is needed to find out the exact factors that determine the relationship of *T. gondii* infection in schizophrenic patients.

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