Schizophrenia is serious, multifactorial and chronic disease. The crucial tasks to introduce effective preventive, diagnostic and treatment measures is to identify etiology factors, their interaction and etiology factors-genetic predispositon interactions. TG (*Toxoplasma gondii*) is the most promising infectious candidate with „schizophrenogenic" potential. Toxoplasmosis is a lifelong parasitosis considered as a risk factor to schizophrenia. No consistent clinical pattern has been detected in *Toxoplasma*-infected schizophrenia patients as yet. We assessed symptom profile, cognitive performance and treatment response of *Toxoplasma*-infected and *Toxoplasma*-free schizophrenia patients to determine whether co-occurrence of schizophrenia and *Toxoplasma* infection modifies clinical presentation and the course of schizophrenia. We screened for anti-*Toxoplasma* antibodies in 251 patients with schizophrenia spectrum disorder consecutively admitted to Prague Psychiatric Centre between 2000 and 2010. Fifty-seven patients were infected (22.7%). Infected patients spent more days in hospital during their last admission compared to uninfected ones (p=0,003; mean difference 32.9 days). Schizophrenia started approximately one year earlier in infected men and about 3 years later in infected women. This corresponds to sex related toxoplasmosis incidence curves in the Czech Republic. Infected and uninfected patients differed in severity of symptoms measured with the Positive and Negative Symptom Scale (PANSS), p=0,032. The post hoc tests showed that all infected patients scored higher in positive subscale of PANSS and infected men scored higher also in Total PANSS score, and negative, reality distortion, disorganisation and cognitive scores. The concentration of IgG anti-*Toxoplasma* antibodies decreases with the duration of of infection (Kodym et al., 2007). Higher PANSS scores of positive, negative and disorganised psychopathology were associated with the low titres of anti-*Toxoplasma* antibodies. It indicates that severity of schizophrenic symptoms in our sample relates to the duration of the *Toxoplasma* infection. Our findings suggest that toxoplasmosis may lead to a more severe positive schizophrenic psychopathology and perhaps less favourable course of schizophrenia. Toxoplasmosis-associated schizophrenia may represent a distinct subtype of pathogenetic process. We believe that the long-term effect of toxoplasmosis in schizophrenia needs a more careful investigation because it may result in poorer outcomes, such as higher risk for relapse, incomplete treatment response, insufficient adherence to treatment or poor psychosocial functioning. The toxoplasmosis-associated changes in the course of psychotic illness may provide a rationale for the inclusion of anti-*Toxoplasma* antibodies screening programmes and more assertive preventive, educational and diagnostic measures. Highly effective interventions to prevent TG transmission include the use of gloves while having interactions with soil, adequate cooking of meat before consumption except using microwave owens, washing kitchen knives after cutting meat, fruits and vegetables, and frequent hand washing. Other interventions include reduction of feral cat populations, protection of livestock feed from contamination by cat faeces and mandatory vaccination of cats for toxoplasmosis.