

## **IDO1 (indoleamine 2,3-dioxygenase 1) has a role in the Psychiatric manifestations of Latent Toxoplasmosis?**

**BaharakKhabazghazvini**

University of Maryland, USA

The human IDO1 gene with 403 amino acids, located on chromosome 8p12-p11 encodes the intracellular enzyme indoleamine 2, 3-dioxygenase 1. The gene's promoter carries transcription factor sites that makes it responsive to the type I and type II interferons, IFN- $\alpha$ ,  $\beta$  and  $\gamma$ . IFN- $\gamma$  strongly induces the IDO1 transcription, resulting in an increase of Indoleamine 2,3-dioxygenase I activity. IDO1 is the first enzyme in the kynurenine pathway (KYN) where tryptophan is broken down into nicotinic acid adenine dinucleotide (NAD<sup>+</sup>) and an intermediate metabolite, N<sup>1</sup>-Formylkynurenine. Tryptophan is an essential amino acid for growth of many organisms including the proliferation of *Toxoplasma gondii*. However, tryptophan 5-hydroxylase can also use tryptophan in order to produce serotonin, an important neurotransmitter in psychopathologic disorders. *T. gondii* is an intracellular protozoan with a complex life cycle that can impact the human immune system. In the United States of America, more than 60 million individuals carry this parasite and over half of the world population is estimated to be infected, but few have any symptom manifestations. *T. gondii* causes a strong Th1 immune response where IFN- $\gamma$  is specifically secreted. The chronic form of Toxoplasmosis primarily had been assumed to be asymptomatic, however the idea has been revised toward the belief that in chronic toxoplasmosis there is a decrease in immune response as well as an increase in IFN- $\gamma$  and cytokine levels. IFN- $\gamma$  through IDO activity can lower the serotonin that in conjugation with KYN pathway metabolites participate in the pathophysiology of neural degeneration, seizure disorders, neuroimmunological disorders, schizophrenia, depression, and perhaps increases the risk of suicidality.

### **Biography**

Dr. BaharakKhabazghazvini completed her Medical Doctorate at the Tehran Azad University in Tehran, Iran in 2003. Currently she is working as a post doctorate fellow at the University of Maryland, coordinating the NIH granted study on Pharmacogenomics of Thiazolidinediones in Personalized and Genomic Medicine. Her personal interest in the metabolic aspects of psychiatric disease, lead her to pursue a research fellowship in the Mood and Anxiety Program at the University of Maryland from April 2009 until August 2010. She took charge as a study sub-investigator in the NIH sponsored study, Bright Light Therapy: Metabolic effects and prediction of antidepressant response by immediate improvement. She also coordinated the study on Past suicide attempts, schizophrenia and *Toxoplasma gondii* IgG antibodies, with collaboration from the University of Munich and Johns Hopkins University. During her fellowship she coauthored seven peer reviewed journal articles with two being published in PubMed. She coauthored a chapter on Vitamin D and suicide risk factors, in Environment, Mood Disorders and Suicide published in 2011. She was also first author of an article published in the International Journal of Disability and Human Development in the area of mediators in chronic latent neurotropic infections and behavioral abnormalities.