Analysis of Schizophrenia Link to T. gondii Parasite

Sz schizophrenia.com/sznews/archives/004867.html

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E. Fuller Torrey, John J. Bartko, Zhao-Rong Lun, and Robert H. Yolke took on the task of analyzing data from a multitude of research sources from around the world, studying the connection between infection with a one-celled parasitic organism called *Toxoplasma gondii* and schizophrenia.

People can get the organism by inhaling or ingesting the oocysts (dormant form) which are shed by infected cats into litter boxes, gardens and sandboxes, or through ingesting (eating) the undercooked meat of sheep, goats, or other animals that had become infected with it from exposure to cats. It had been observed that some individuals who develop adult-onset toxoplasmosis (infection with *T. gondii*) exhibit delusions and hallucinations.

Torrey, et al. say that the data came from studies carried out over 5 decades in 17 countries employing several different methods of antibody measurement. The data show that the prevalence of antibodies to *T. gondii* in individuals with schizophrenia is significantly higher - over two times as common - than in individuals without schizophrenia.

The data raises many questions, and some very interesting points are made in the paper.

T. gondii tends to reside in brain neurons, especially the ones called, "glia", which are sometimes referred to has the "helper cells" - heavily involved in forming the scaffold for brain structure, guiding the growth of other brain cells. Glia may be centrally involved in schizophrenia. Next, the authors point out that some antipsychotic drugs used to treat schizophrenia have been shown to inhibit the growth of *T. gondii* in cell culture. Also discussed is the relationship of winter and spring births to the development of schizophrenia and that toxoplasmosis, like many infectious diseases, also occurs more commonly in the winter and spring months.

Still, the authors point out reasons that all this research is still not definitive as to cause and effect, not the least of which is the fact that the majority of individuals with schizophrenia do not have measurable antibodies to *T. gondii*. E.F. Torrey and colleagues also raise the point that some individuals who are exposed to *T. gondii* do not develop schizophrenia.

Questions raised for future research includes investigation of timing -- When might exposure to *T. gondii* be critical in causing the harm that leads to the development of schizophrenia? A discussion of the article in Schizophrenia Forum points out that:

> Authors also cite publications that implicate maternal T. gondii infection in the etiology of schizophrenia. This raises the question whether the presence of antibodies in adults reflects prenatal infection, or whether postnatal infection with T. gondii is also a risk factor.

> What are the underlying mechanisms by which exposure to T. gondii leads to schizophrenia vulnerability, and how do these interact with other risk factors, including genetic factors?

> Possible reasons include differences in genetic susceptibility, organism strain differences, route of infection (eg, ingestion of oocysts from infected cats versus tissue cysts from meat), and timing of the infection (eg, in utero, early postnatal, childhood, adulthood). Each of these factors is known to lead to different disease outcomes for other infectious agents. The study of these factors will be important in further defining the relationship between *T. gondii* and schizophrenia.

Read the full article: Antibodies to *Toxoplasma gondii* in Patients With Schizophrenia: A Meta-Analysis (PDF file - requires Adobe reader)

Read the article discussion: Forum Discussion: Antibodies to *Toxoplasma gondii* in Patients with Schizophrenia: A Meta-Analysis

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Posted by Jeanie Wolfson at April 18, 2007 06:30 AM

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Comments

I would like to join a study group for this somehow. I belive I may have been infected and I know possible how.

When I was 5 years old my mother had a friend that had a kid the same age as me. We used to play together.

He was involved with beasieality and his cat.

I'm wondering if I could have picked this parasite up from playing with him.

He is now 30 years old the same age as me. He has developed symtoms similar to schizophrenia while I've developed it full blown.

Why I developed it full blow I dont know? Maybe I was more geneticaly suseptable?

But I remember he had some major screws loose when we were kids. I wonder if it was the parasite. Wonder is his mom was infected prenatal thats why he was screwed up. I used to joke he was gonna be the next Jeffry Dohmer.

My mom still talks to his mom and from what I hear he's not doing to well.

This article raises alot of questions for me.

Posted by: Josh at April 18, 2007 09:13 AM

Interesting summary.Informative. Since infection is in immunocompromised individuals, good health, nutrition, lifestyle important to all. Authors raise the best question, why might some babies react to infection this way. Brings focus back to the individual and what is going wrong in their dna, immune system, or brain. But nothing yet is "definitive" about this.

Posted by: Fran at April 18, 2007 04:04 PM

Fran,I am currently studying psychology and had come upon your dilemma. Since I have been raised around cats and my daughter too, I was just wondering how long it had taken for your doctor or family to notice this change in you? You have the right to have many questions on this subject and right now we are studying schizophrenia and I would like to pass any information along to you if found. I would highly like to keep in touch with you if this is possible.

Posted by: Debby Knight at January 1, 2008 07:19 AM