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Inflammmation During Pregnancy Associates with Schizophrenia

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Role of inflammation in the incidence of schizophrenia

At first glimpse, it seems that protecting women from infection during pregnancy is beyond the area of psychiatry. However, several epidemiologic and preclinical reports suggest that infection and ensuing immune activation have a pivotal role in the etiology of several neurological disease including schizophrenia, a severe, chronic, and commonly incapacitating brain and behavior condition. Consistent with this, a Child Health and Development Study (CHDS) birth cohort has revealed that maternal contact with influenza early through mid-pregnancy is likely to increase risk of schizophrenia among offspring [1]. This cohort has also indicated that elevated IgG antibody to *Toxoplasma gondii* (*T. gondii*) is associated with elevated risk of schizophrenia [2]. This result was duplicated in a Danish study demonstrating that increased IgG antibody to *T. gondii* infection in infant correlated with elevated schizophrenia risk [3]. More recently, an early increase in gestational C-reactive protein, an established inflammatory biomarker, was linked to schizophrenia in offspring [4]. The direct association between inflammation and brain disorder tested in rats using lipopolysaccharide (LPS), a bacterial protein inducing neuro-inflammation, demonstrated a surge in microglial production of the pro-inflammatory factor TNF-α paralleled by hippocampus-related cognitive deficits, both of which were greatly reduced by TNF-α inhibitor [5].

Traditionally, it has been assumed that changes in DNA sequence are mainly responsible for the transmission of schizophrenia. The genetic risk for schizophrenia has been suggested to result from various forms of DNA sequence variation- the best recognized are those caused by single nucleotide polymorphisms (SNPs) and copy number variants (CNVs). Yet, there are no confirmed causal mutations, nor families in which schizophrenia separates in a Mendelian fashion [6]. A meta-analysis by the Psychiatric Genetics Consortium (PGC) [7], found 22 loci, which contain SNP(s) genome-wide significant associated with schizophrenia. Over 8000 SNPs independently promote the incidence of schizophrenia, and together they will explain over 50% of the genetic predisposition [7]. A more recent analysis from the PGC on a considerably larger sample of almost 37,000 cases and 113,000 controls detected over 100 loci implicating about 600 genes important for schizophrenia [8]. These genetic studies corroborate the idea that schizophrenia is an extremely polygenic disorder [9,10].

The interactions between genes with environmental risk factors such as inflammation is very highly plausible. A study involving bacterial meningitis (BM) patients and 110 healthy volunteers (as the control group) revealed a significant association between genetic variability and altered inflammatory responses [11]. One speculates that genetic polymorphisms in inflammatory regulators resulting in exaggerated inflammatory responses may contribute substantially to the incidence of schizophrenia in the offspring. The exact mechanism is still unclear however, maternal infection/inflammation may be associated with the induction of placental inflammatory cytokines that could result in elevated fetal pro-inflammatory cytokine exposure [12], and development of neonatal neurologic disorder. Therefore, there is a need in emphasizing on prevention and control of inflammatory conditions in pregnant women. Comprehensive psychiatric and psychological treatment for pregnant women, as well as their physical monitoring, would be appropriate for the health of the mother as well as for the longer-term risk of mental illness such as schizophrenia in her offspring.

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