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12ᵗʰ International Conference on

Pediatric Pathology & Laboratory Medicine

March 15-16, 2017   London, UK

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Prenatal screening of maternal immune antigen biomarkers linked to microglial regulation of brain development may predict autism risk

Recent discoveries of the connections between the maternal immune system (IS) and prenatal brain development suggest that routine prenatal screening for chronic disorders associated with IS dysfunction may be useful in identifying women at heightened risk for giving birth to a child with autism. Epidemiological studies have shown that the incidence of IS disorders, including systemic lupus erythematosus (SLE), rheumatoid arthritis (RA) and chronic obesity in combination with insulin-resistant diabetes has increased significantly over the past several decades and that pregnant women with these conditions are at increased risk for having a child with autism. For this reason, physiological parameters associated with these prenatal conditions that can be detected before onset or at early stages of disease may serve as biomarkers for increased autism risk. A physiological relationship between maternal IS dysfunction and impaired embryonic/fetal brain development may be defined by critical neurodevelopmental functions of brain microglia that are responsive to both neural and immunological stimuli. Impaired regulation of the developmentally versus immunologically defined functions of brain microglia may represent a primary cause of the neurological impairments characteristic of ASD. This critical cause/effect of relationship provides the rationale for autism risk factor assessment using biomarkers associated with chronic immune conditions that impair the neurodevelopmental functions of microglia as a consequence of their inappropriate immunological activation. Moreover, the connection between abnormal IS function and impaired neural development suggests preventive approaches that can be used to decrease the overall risk for ASD in children born to mothers with these conditions.

Biography

Sarah Adelaide Crawford completed her Doctoral degree in Physicians and Surgeons Department of Biochemistry and Biophysics from Columbia University College. She completed her Master's degree in Biochemistry from the Princeton University. Her Post-doctoral research was carried out at Memorial Sloan Kettering Cancer Center in New York. She is a Professor of Genetics at Southern Connecticut State University and Director of Cancer Biology Research Laboratory. Currently, she is working on the causes and prevention of autism. She has developed a new model to explain the causes of autism and its recent dramatic increase. Applications of this model can be used in preventive approaches to screen for autism risk factors to reduce the occurrence of this disorder.

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J Clin Exp Pathol
ISSN: 2161-0681 JCEP, an open access journal

Pediatric Pathology & Laboratory Medicine 2017
March 15-16, 2017

Volume 7, Issue 1 (Suppl)

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