Autism Course

With Michael Ash BSc (Hons) DO, ND, Dip ION

Thursday 12th March 2009
9am to 4pm

VENUE

Bramhope Methodist Church Hall
Brearey Lane
Bramhope
Leeds LS16 9AA
2 miles from Leeds Bradford airport

Cost **£350** inclusive of VAT

To book please call Nutri-Link: **08704 054 002**

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summary extracted and edited from:

Altered gene expression and function of peripheral blood natural killer cells in children with autism. Brain Behaviour and Immunity Pages 124-133. Amanda M. Enstrom, Lisa Lit, Charity E. Onore, Jeff P. Gregg, Robin L. Hansen, Isaac N. Pessah, Irva Hertz-Picciotto, Judy A. Van de Water, Frank R. Sharp, Paul Ashwood

Michael will explore the evolving science in the relationship to immune disturbances and how the choice of foods may contribute or mitigate this. He will also look at the role of microbial agents and other therapeutic interventions relating to biomedical and biochemical interventions.

Case histories and practical strategies will also be discussed.

This is aimed at practising health care professionals seeking to expand their knowledge in this area of health disruption in an open and intellectually stimulating day.

Autism spectrum disorders (ASD)[1] are complex neurodevelopmental disorders which are typically diagnosed within the first three years of life. ASD are characterised by significant impairments in social interaction and communicative skills, as well as restricted and stereotyped behaviours and interests.[2] ASD includes both Asperger's syndrome and autism disorder, as well as pervasive developmental disorder not otherwise specified (PDD-NOS) (Specific diagnosis is determined by the nature and severity of delays or deficits in communication, social interactions and the presence or absence of restricted and stereotyped behaviours/interests. Males are four times more likely to be diagnosed with ASD than females.[3] Over the past decade, intense interest has focused on ASD as the prevalence appears to be increasing. Recent estimates, including the recent CDC study, place overall prevalence of ASD at 1 per 150 children.[4]

Despite expanding research in ASD, its aetiologies remain poorly understood and the relative contribution from genetic, epigenetic, and environmental susceptibility factors remains widely debated.[5] Twin studies indicate a strong heritability for ASD risk[6] and whole genome scans have revealed potential ASD candidate genes on nearly every chromosome.[7,8] Several studies have demonstrated ASD associations with immune related genes, including: complement C4 null allele.[9,10] In addition, systemic abnormalities of the immune system have been one of the most common and long-standing reported findings in ASD.[11,12] Extensive neuroimmune interactions, beginning as early as embryogenesis, offer one possible explanation for the involvement of the immune response in the development of ASD and the ongoing immune alterations demonstrated in affected individuals.

Immunological findings in ASD have been reported systemically and at the cellular level, including familial associations with autoimmune and/or immune disorders such as atopy and asthma.[13,14] Notably, altered production of proinflammatory signaling proteins, such as cytokines, have been identified in the plasma, peripheral immune cells, brain, and CSF of individuals with ASD.[15] There is a growing literature that demonstrates the increased presence of autoantibodies, especially to CNS proteins, in children with ASD and some mothers of children with ASD.[16] In susceptible individuals, immune dysregulation may predispose to the generation of aberrant or inappropriate immune responses such as autoimmunity and/or adverse neuroimmune interactions which during critical developmental windows may ultimately lead to changes in neurodevelopment.

Abbreviations used: ASD, autism spectrum disorder; PDD, pervasive developmental disorder.

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