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Autism Spectrum Disorders have a Neurobiological Basis

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Autism Spectrum Disorder (ASD) is a broad term used to refer to a group of prevalent and debilitating developmental disorders, which includes Autistic Disorder (AD), Asperger's Disorder and Pervasive Developmental Disorder-Not Otherwise Specified¹. Each of these pervasive neuro-developmental conditions is characterized by impairments in verbal and non-verbal communication, social interaction, and a restricted repertoire of activities and interests combined with repetitive behaviour and stereotypes. According to latest estimates, ASDs affect 1:167 individuals in the general population² with a male to female ratio of 4:1.

Although the neurobiological basis of ASDs is unquestionable, the underlying causes of this complex and heterogeneous body of disorders await further clarification. A flurry of neuropathologic and neuroimaging studies have reported developmental anomalies in specific regions of the brain (including the cerebellum, mesial temporal structures, brainstem, basal ganglia, amygdala, frontal and parietal areas and the corpus callosum), although the findings are not fully consistent across studies (reviewed in 3). However, it is generally agreed that early brain development is impaired, with consistent MRI findings of increased brain size in young children aged 2-4 years. The main anatomical anomaly identified in autism is an enlarged head circumference (HC) which is consistent with this early excessive brain growth (reviewed in 4).

An excess of neurons in the brains of individuals with autism, and/or reduced cortical pruning has been postulated as the cause of larger HC and brain volumes.

There is current consensus that the behavioral and cognitive manifestations of ASDs are brought about as a result of altered brain connectivity as evidenced in numerous fMRI studies. This view is consistent with a recent and important finding from the Autism Genome Project Consortium (2007)⁵ implicating a region on chromosome 11p, and a specific gene called neurexine 1 which is involved in nerve cell communication. Many other genes and chromosomal regions have been implicated in autism. However, further multi-disciplinary research is needed to provide an integrative model of the underlying biological mechanisms involved in ASDs.

References

¹ American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th Ed – Test Revision). Washington DC: American Psychiatric Association.

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³ Penn HE. (2006). Neurobiological correlates of autism: A review of recent research. *Child Neuropsych*, 12:57-79.

⁴ Courchesne E (2004). Brain development in autism: *Ment Retard Dev Disabil Res Rev*, 10:106-11.

⁵ The Autism Genome Project Consortium. (2007). Mapping autism risk loci using genetic linkage and chromosomal rearrangement. *Nat Genetics*, 39: 319-28.