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The neurobiology of autism: Theoretical applications

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ABSTRACT

Autism spectrum disorders (ASD) are complex neurological disorders characterized by heterogeneity in skills and impairments. A variety of models have been developed to describe the disorders and a wide range of brain processes have been implicated. This review attempts to integrate some of the consistent neurological findings in the research with three of the dominant models of core deficits of ASDs: the weak central coherence model, the theory of mind model, and the mirror neuron system model. A review of the literature suggests that the cerebellum and the frontal lobes may be implicated in all three of the models, while the temporal lobe is associated with the theory of mind model and the mirror neuron model. In particular, the theory of mind model and the mirror neuron system model both implicate the inferior frontal gyrus and the superior temporal sulcus. This review indicates that each model appears to be heavily substantiated by neurological research, suggesting that each may capture important aspects of ASDs.

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Autism spectrum disorders (ASDs) are complex neurological disorders that are marked by social impairment, repetitive and stereotyped behavior, and difficulties in communication. They are characterized by heterogeneity in skills and impairments in each of these areas, leading to a wide range in severity of symptoms. Thus, it is not surprising that there are a variety of models that have been developed to describe the disorders, a number of genetic factors that appear to contribute to its etiology, and a wide range of brain areas implicated.

This review attempts to integrate consistent neurological findings in the research with some of the dominant models of core deficits of ASDs in order to provide clinicians with a better understanding of the neurological underpinnings of the phenotypic characteristics of clients with ASDs. First, there will be a brief overview of the etiology and developmental course of ASDs, followed by a brief description of three of the major models of the core deficits of ASDs: the weak central coherence model, the theory of mind model, and the mirror neuron system model. Next, regions of the brain that are strongly supported by the research as being involved in ASDs (i.e., the cerebellum, frontal lobe, and temporal lobe) will be reviewed, followed by a brief description of some of the other brain regions that require more empirical substantiation. Next, these findings will be integrated with the three models of ASDs outlined. Finally, challenges in neurological research on ASDs, and future directions for research will be outlined.

1. Etiology of autism

The etiology of ASDs is complex and has been associated with genes, neurological pathways, neurotransmitters, and environmental influences. Although etiologies have not been firmly established in the majority of cases, it is of interest to review possible causal factors to understand the incidence and prevalence of the spectrum of outcomes.

1.1. Genetic and environmental factors

Reviews of research in both quantitative and molecular genetics indicate that autism is polygenic, with as many as 20 chromosomes involved (Gillberg & Coleman, 1992); in particular chromosomes 1, 2, 4, 7, 13, 15, and 16 have been implicated (Chudley, 2004). Rutter (2002) estimates that approximately 70% of the variance in autism is due to genetic factors. Environmental factors have also been implicated in some cases, particularly prenatal influences of alcohol, thalidomide, and intrauterine exposure to viruses. Converging research suggests that the physiological correlates of autism develop prenatally, before 30 weeks gestation (Kern, 2003).

1.2. Development of neural pathways

Gillberg (1999) proposes that there are two neural pathways that can be affected during particular critical windows, and may underlie the development of autistic symptomatology. The first pathway develops prenatally, between the 4th and 8th week of pregnancy, and involves connections between the brain-stem and the cerebellum. According to Gillberg, this pathway may be implicated in deficits of intermodal attention shifting, clumsiness, and difficulties with imitation. The second pathway is described as the temporo-frontal pathway, which connects during the middle of the second trimester, and around two years of age in a minority of cases. This pathway is purported to be implicated in the social and communication deficits seen in autism. Gillberg proposes that dysfunction in the cerebellum may lead to disruption in temporo-frontal development, or that temporo-frontal dysfunction may occur independently.

Bauman and Kemper (2005) have been conducting research since 1985 that highlights the link between cerebellar dysfunction and autism. They suggest that cerebellar disruption occurs prenatally, and that neural changes underlying autism continue after birth. Research reviewed by Carper and Courchesne (2000) lends some support for the developmental link between dysfunction in the cerebellum and deficits in frontal lobe functioning. These models indicate that the cerebellum may be of particular importance in the development of autism, that the potential neurological markers for autism appear to develop quite early prenatally, and that there may be other post-natal critical periods. These dysfunctions in development may be caused by dysregulation of cell migration, synaptic pruning, insufficient apoptosis, and problems with myelination (for reviews see DiCicco-Bloom et al., 2006; Gillberg, 1999; Penn, 2006).

A number of brain structures have been associated with autistic symptomatology, including the cerebellum, the frontal lobes, and the temporal lobes. Although there is growing convergence regarding the structures involved, the literature is lacking a theoretical orientation that describes, what deficits are central to autistic symptomatology, and which areas of the brain can be linked to these deficits. The following section will review three models of central deficits in autism, namely the weak central coherence model, the theory of mind model, and the mirror neuron system theory. These models were selected