Molecular mechanisms underlying neurodevelopmental disorders, ADHD and autism.

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Abstract

Neurodevelopmental disorders such as attention deficit hyperactivity disorder and autism represent a significant economic burden, which justify vigorous research to uncover its genetics and developmental clinics for a diagnostic workup. The urgency of addressing attention deficit hyperactivity disorder comorbidities is seen in the chilling fact that attention deficit hyperactivity disorder (ADHD), mood disorders, substance use disorders and obesity each increase the risk for mortality. However, data about comorbidity is mainly descriptive, with mechanistic studies limited to genetic epidemiological studies that document shared genetic risk factors among these conditions. Autism and intellectual disability affects 1.5 to 2% of the population in Western countries with many individuals displaying social-emotional agnosia and having difficulty in forming attachments and relationships. Underlying mechanisms include: (i) dysfunctions of neuronal miRNAs; (ii) deletions in the chromosome 21, subtelomeric deletions, duplications and a maternally inherited duplication of the chromosomal region 15q11-q13; (iii) microdeletions in on the long (q) arm of the chromosome in a region designated q21.1 increases the risk of delayed development, intellectual disability, physical abnormalities, and neurological and psychiatric problems associated with autism, schizophrenia, and epilepsy and weak muscle tone (hypotonia); (iv) interstitial duplications encompassing 16p13.11.