Attention deficit hyperactivity disorder (ADHD) in phenotypically similar neurogenetic conditions: Turner syndrome and the RASopathies.

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Abstract

BACKGROUND:
ADHD (attention deficit hyperactivity disorder) is a common neurodevelopmental disorder. There has been extensive clinical and basic research in the field of ADHD over the past 20 years, but the mechanisms underlying ADHD risk are multifactorial, complex and heterogeneous and, as yet, are poorly defined. In this review, we argue that one approach to address this challenge is to study well-defined disorders to provide insights into potential biological pathways that may be involved in idiopathic ADHD.

MAIN BODY:
To address this premise, we selected two neurogenetic conditions that are associated with significantly increased ADHD risk: Turner syndrome and the RASopathies (of which Noonan syndrome and neurofibromatosis type 1 are the best-defined with regard to ADHD-related phenotypes). These syndromes were chosen for two main reasons: first, because intellectual functioning is relatively preserved, and second because they are strikingly phenotypically similar but are etiologically distinct. We review the cognitive, behavioral, neural and cellular phenotypes associated with these conditions and examine their relevance as a model for idiopathic ADHD.

CONCLUSION:
We conclude by discussing current and future opportunities in the clinical and basic research of these conditions, which, in turn, may shed light on the biological pathways underlying idiopathic ADHD.