

Review Article

Attention Deficit Hyperactivity Disorder and its Evolution

Abstract

The organic process standing of Attentiveness deficit/hyperactivity disorder (ADHD) is principal to the estimation of whether the fashionable community has generated it, either mundanely or culturally, and is doubtless helpful in the apprehension of biological sciences' cornerstone and foundation. The high widespread of the syndrome (5–10%) and its intrinsic coalition with the 7-repeat citrons of DRD4, which is entirely elite in advancement, augment the chance that the syndrome will increase the procreative robustness of the individual and the cluster. Nevertheless, preceding recommendations of organic process roles for syndrome haven't reckoned for its restrain to a considerable minority. As a result of one among the pointer options of the syndrome is its divergences, and plenty of edges of diverging community area unit well conceded (as in exemption), we tend to analyze the influence of bands' activity diversification is shown on their health. Diversity happens to several extents, and just for intangibility, we elect unforeseeable (or changeable), way over that may be a well-orthodox attribute of the syndrome.

Counterfeit of the dynamic Food cluster charge puts before you the uncertain behavior by the outnumbered optimizing results shown from the cluster. The trait of such cluster inspection chore area unit peril danger-taking, within which prices area unit borne principally by a discrete individual; and statics-sharing, within which edges ensure the complete cluster due to previous knowledge of evolved selflessness.

We conclude that even singly impairing combos of genes, like syndrome, will carry particular edges for the community, which may be elite at that stage, instead of simply genetic fortuity with outcomes limited to the individual. The communal edges presented by diversity occur within and outdoors. The 'normal' varies, and these could also be definite. This read has the extra advantage of clarifying the usual sexuality and age distribution, asperity dispersal, and no uniformity of syndrome.

1. Introduction

Attention deficit hyperactivity disorder (ADHD) is a trait through scientific features, similar to distraction, inattention, and involuntary action (American Medical Specialist Association 2000). Several tries had been made to explain MBD and feature junction rectifiers for a giant set of experimental results: molecular, neuroscience, structural, interactive, and social clusters. Of those categories, possibly the most outstanding in accounting for the disparate findings in neuropsychology; however, there are numerous theories and high-quality outcomes (1,2,3). Even though a few trust that MBD may be proven to have the handiest 1 or 2 causes, it's miles an increasing number of possibilities that MBD is a whole lot extra heterogeneous than that.

Apropos to any or all of these categories of simplification and the need to fully apprehend them is an organic process that involves minimal brain damage. This is frequently the polar opposite of the principle question of whether fashionable society causes it through toxins (4,5,6) or a dire social abode (7,8) or whether the prevalence is rising. It's plausible that the increase in recent decades is attributable to bui better recollection (9) or diagnostic errors (10,11). It is predo meant to note that although reports on the slightc processes of minute brain injury cardinally address whether minimal brain damage is wholly a contemporary detriment, they ordinarily do not support one conjecture over another. Psychological science axiom includes brain mechanisms that resolve the nature of the activity (12), while this article criticizes the past and present effects.

Although there are substantial contributions, several studies of twins, families, and adoptions have revealed that even minor brain injury has a high heritability (13,14). In terms of statistics, the ADD | ADHD | hyperkinetic syndrome, minimal brain dysfunction, minimal brain damage | MBD | syndrome represents a watershed moment in the typical allocation frenzies, spontaneity, and basic cognitive processes in the community, because the leading source is in the supplemented implication of varied genes (15). Many genes and alleles are correlated with minimal brain damage, with odds ratios, Organized into three or further studies, at varying intervals. 161.44 (all noteworthy) for variant DRD4, DRD5, monoamine neurotransmitter conveyer (DAT), dopamine beta-hydroxylase, SNAP25, 5HT conveyer (5HTT), and 1B 5HT receptor (16). Nonetheless, genome-wide linkage studies have unconcealed divergent undisclosed companies (17), and also the chunk portion of minimal brain damage thanks to these, supposedly non-neurotransmitter, genes aren't commonly accepted. The presumption of

the diagnosis is virtually a symptom score combined with a subjective appraisal of the disorder (18); These two measures show good reconciliation (19).

The commitment to base analysis on personal data retrieved from folks and academics establishes goodish deadlocks once scrutiny diagnostic rates from comprehensive heterogeneous customs are accomplished. It is, thus subsequently, reckoned to search out goodish modification in estimates of minimal brain damage prevalence—from four to nineteen victimization sundry diagnostic scenarios in several countries (20,21). Per contra, once estimation strategies square allowance are adroitly standardized, prevalence is 6–10% pole to pole (21,23).

In the paper above, we gravitate toward the high-strung/wild side (or magnitude) of minimal brain damage. The distraught side of minimal brain damage is tied in with depressed I.Q. Moreover, additional organic process dilemma than authority (24,25,26). Impersonally specific women seldom retain simply high-strung /off-hand issues (27); as a consequence, this leans as legitimate in medical specialty teams also (28,29). Thus women's square measure is mostly precluded. To honesty, we tend to additionally suspend contributive ponderable including unusual genetic factors, although this square measure central in a few cases (30,31).

As for diagnosis, the single-factor allele provides further unbiased data on progression. The seven-repeat taxonomic category (dopamine receptor -D47R) of the monoamine neurotransmitter receptor dopamine receptor D4 emerges to correlate, albeit decrepit ably, with sensation searching (32), moreover rather more strongly with minimal brain infliction (33,34,35), Tourette (36) also conceivably dreariness (37). The pervasiveness of dopamine receptor D47R ranges starting with zero towards one hour within the date in very separate geographic areas (38). To illustrate, the Mandarin were more likely to attribute a minimal pervasiveness of brain damage in the West (22,39) and exceptionally the truancy of dopamine receptor D4-7R (40). dopamine receptor D4-7R demonstrates goodish atomic proof about getting withheld latest decisive choice, next to looks by the structurally trendy mortals (41,42,43). As a result of minimal brain damage, the indication produces a widely contemplated problem as non-adaptive (44). The peculiar coalition with dopamine receptor-D4-7R increases the likelihood that minimal brain damage is also elected because of new progression. Society tends to like the proof as to the

reason for Attention deficit hyperactive disorder (hi-strung/automatic) that is examined in barely a fraction of folks.

2. Constituents affecting the resoluteness of ADHD

We tend to provide a generic framework that defines the biological process stresses that are important to a minor brain malfunction. Many unitary ideas have been advanced previously to understand the occurrence of minimum brain malfunction.

Factors that are likely to influence the generality of hyper activeness-emotional behavior in the general community. Components of questionable significance are shown with parenthesis.

There square measure many factors that square measure seemingly to own a job in crucial the generality of AD/HD-linked genes. We tend to state vital peril to the discretely, solely, and concisely, as they're considerably treated within the literature composition. Folks with minimal brain dysfunction have elevated danger of different medicine disarray (45) and instructional and occupational toil (46), additionally, as some two hundredths accrued utilization of exigency and patient medical services (47). trendy community leans to visualize unbridled folks as aberrant (48). Sure, This is something that cultures have done in the past, possibly a lot (49).

Variability also includes the desire for novelty. The discovery of a link between novelty seeking and DRD4-7R, as well as precise information on the gene's regional distribution, has led to two significant possibilities. First, DRD4-7R may impose a level of novelty-seeking that is beneficial in some societies, such as feminine-influenced farming (for male brawling and intercourse-associated contests) and an impediment in others, such as hunter-gatherer and urbanized societies—instead of being such trouble in growingly affluent economies per se. This explanation adequately explains numerous features of the geospatial data, but it fails to explain why the alleles are significantly more important in the five Central And South American ethnic communities than in all 33 ethnic groups studied on other continents. Novelty-seeking may drive migration in migrated/migrating communities or, more likely, provide adaptive value. Because transitory cultures typically include a small number of the antecedents, genetic variability is reduced, assessing adaptation becomes even more challenging. Exploratory methodologies that allow for 'from before the extraction of natural resources,' on the other hand, would benefit groups that have undergone recurrent evolutionary (or emigrational) divergence. This could

explain why DRD4 -7R has a high prevalence in South America, where the most inventive newcomers would be rewarded for filling unexpected or unfilled ecological niches. On the other hand, associations with specific alleles can be too coarse granulated to reflect the accurate transformative current regulated by phenotypes. Myriad variants that create the same trait will not face selection pressure.

Activity variance is a big part of minor brain impairment, so mysterious. It's mainly one of ADHD's profusely different traits(50,51). However, It's not always clear how much of the changeability is quite desultory and how much of it is merely behavior that we haven't yet ascertained to foresee (can not be predicted vaguely) (53). Verifiable uncertainty is one factor that contributes to the appearance of impulsivity(54). A decrement inefficient memory retentiveness, as an alternative, might have similar effects—at least in some dogmas. Another facet of fluctuation is the propensity for taking chances, which can be beneficial in some situations (55,56)and will be discussed later.

There are a variety of adjusted individual traits connected with a syndrome that aren't linked to behavioral discrepancies. It has been suggested to have a more significant potential to elicit parental attention (57). Furthermore, being 'difficult' will help infants survive during a drought (54). Nevertheless, betwixt such predicament, maternal devotion to children with the syndrome is significantly lower than that to children without syndrome (58), which may have negative consequences for emotional and behavioral development, acting directly or indirectly thru reduced maternal warmth (59,60).

Do the children continue to reproduce after they have reached adulthood? The solution to the current issue is unclear. There's corroboration that people with the syndrome are more likely to have unsafe coitus (61), and girls prefer sensation-seeking men (62). But even so, the syndrome is common and associated (in our gift society) with several psychiatric conditions, the majority of which decrease copy number (63,64).

Other ideas in the broad category of specific adjustments are even less convincing. Improved creativity, for instance, has been indicated, but formal assessments of creativity in children with ADHD are no higher in contrast to the standard. Teenagers are typically unable to constrain their variability to situations where it is advantageous. Increased territorial exploration could enhance

foraging, recognition of threats, and (at least in theory) learning —but, in contrast, hyperkinesis is uncommon and highly detrimental, especially when widespread. Although hostility is beneficial, it is more presumably linked to oppositionality synchronizing with ADHD in contrast to ADHD itself. Although surveillance, retaliation—preparedness, ardor, and versatility have been indicated, these characteristics are not exclusive to ADHD. Finally, none of the preceding benefits appear to be present in the inattentive subtype of ADHD, which appears to be neither malleable for the discreet sole nor the community.

Disorders can also manifest themselves in the absence of adaptability. Given the great familiarity of ADHD and the degree of coherence of related alleles, *de novo* mutation is restricted to occur on occasion, but is incapable of being a substantial facet(74). DRD4-7R, which is linked to ADHD, also exhibits multiple 'fingerprints' of positive selection. Simple genetic drift is unlikely given the global prevalence of ADHD and its behavioral implications. Preferable mating amongst persons with ADHD could theoretically develop the maintenance of genetic dissimilarity once they have formed, but no evidence has been instituted to support this notion(76). In conclusion, high-effect mutations, drift, and preferred copulation are expected to undergo minimal consequences. Nonetheless, because the inheritability of composite phenotypes (such as ADHD) is significantly larger than the heritability of individual components, the potential of unselected or fast-evolving individual factors cannot be ruled out.

The most common contemporary theory of ADHD's evolutionary position is that it is a side-effect of genes that typically aid but cause individual impairment when they occur in bad combinations or significant numbers. Under this viewpoint, ADHD results from malleable constraint for something other than itself, i.e., a 'maladaptive spandrel.' The attribute chosen is frequently assumed to be either a collection of specific traits or diversity in general.

The most common contemporary theory of ADHD's developmental position is a side-effect of genes that typically aid but cause individual impairment when they occur in wrong configurations or significant numbers. According to this viewpoint, ADHD is the outcome of adaptable stress for something other than itself, i.e., a 'maladaptive spandrel.' The distinctive feature chosen is frequently assumed to be either a collection of different traits or a variety, which is chosen arbitrarily.

3. Discussion

We've conferred a sophisticated read of the organic process standing of ADHD–HI based on examining the literature. We've included two new components to this paradigm at various points: I, the merit of uncertain conduct in dynamic situations and (ii) the advantage of constraining such unforeseeable behavior to a lower community.

It is undeniable that there is a task category ('group exploration tasks') in which a minority's uncertain conduct enhances results for the cluster. The activities have the attributes of (a) risk-taking, since the value is held up mainly by a sole person, and (b) the exchange of information, as the edges expand with cluster volume. Such tasks haven't been the concern of a lot of research. We indicate that clustered investigation tasks reflect real-world pursuits that were important in human development due to inductive learning being so important to humans.

Our synthesis takes into account, and thus requires, several seemingly unrelated aspects of hyperkinetic syndrome–HI; (i) It's primarily heritable; (ii) it's incredibly diverse and multigenic; (iii) ADHD impulsive behavior is halved by adulthood (65) when the value of losing a private is at its highest; (iv) the gravity of hyperkinetic syndrome is typically narrowed due to the necessity for personage with it to interact in ventures that their peers can comprehend (v) it genetically essentially creates a mode that can even be attained on the basis (vi) it's mundane mainly among the sexes with not so much parental investment (66,67); (vii) it's restricted to a lower community but ordinary enough to exist in multiple villages; and (viii) its gravity has roughly Gaussian dissemination within the demography (68). Taken simultaneously, (i)–(viii) form a fancy style for the hyperkinetic syndrome (69), implying that it is a related adaptation.

A gaggle variation within the Baldwin result was used (70,71). According to the Baldwin conclusion, associating organisms' economic discovery of solutions to significant evolutionary tasks would raise individual fitness and, as a result, the likelihood of subsequent generations being genetically susceptible to seek out a comparable solution will grow. In our simulations, the teams grow over extensive periods to become genetically predisposed to conducting atmospheric expeditions in the most efficient manner possible.

Learning and developmental interactions do not appear to be simple (71,72); introducing cooperative subgroups at intervals within a species adds several new challenges (73,74,75). As an

example, once agamous creatures realize (through entirely organic process change) an unlikely and troublesome answer to a survival drawback, those people will undoubtedly pass it on to subsequent generations. This unique profit is lost when you reproduce everything simultaneously, but it can be returned mainly by adding additional experimenting and learning (76). Similarly, an individual with ADHD–cumulative HI's exposure to danger risks erasing all uninheritable knowledge gained during his lifetime—but once he's classed with more trustworthy people, his blunders will be visible to others, and data will not be lost.

Game speculations are broadly employed in organic process representations (77,78), and It gives us an insight into the hyperkinetic syndrome's genesis. Many such age groups of diverse mentees (roundabout commensurate to our uncertain people) and social novices (like our sure discrete persons but limiting the ability for sole learning) will contend over a long organic process time, with the overall equilibrium betwixt them set on by the rate of atmosphere amendment (79,80).

6.1 Testable prognosis

We expect that

- a. When an ADHD–HI individual is replaced for a handling member of the group, the assembled small groups perform more effectively on computerized group foraging activities. The discrepancy may be obvious when a youngster with ADHD–HI joins a batch or quits taking his very consistently prescribed deep-rooted prescription.
- b. ADHD children's younger siblings (or step-siblings) will be marginally more liberal and progressive and less prone to accidents than their older siblings (or step-siblings),
- c. Individuals who live solitary lifestyles, especially those raised alone, or who indulge in aloneness foraging, will have less inter-personal variance on genetic or behavioral parameters.

6.2 Restrictions

The countless common critique at our concept is that it's just another developmental "just-so" scenario. Albeit we have shown that an unpredictable lower social batch can assist the more considerable lot with a specific set of activities (mentioned aforesaid), we can not showing that these activities have frequently betided enough to have influenced evolution. Several of the metrics for an adaptation research question (81-87), such as (i) weighing replacement mechanisms; (ii) trying to install a linkage with genetic uncovering; (iii) taking account for separate facets of ADHD-HI that cannot be explained by cognitive process or other hypotheses; and (iv) making testable predictions have all been met.

4. Conclusion

Previous explanations for the adaptive development of syndrome have failed to account for the disorder's complexity and the syndrome's widespread restriction to a small percentage of the population. To address these concerns, we must examine the balance of benefits and downsides of the syndrome for each individual and the neighborhood wherein he lives. Aside from the well-studied impacts on individual morbidity and replication, which deserve further quantification, we've identified two social benefits of ADHD-HI: first, the accumulation of activity potentialities, and second, the confinement of concurrent social and physical danger to a community.

The issue of identifying one, or possibly many, essential deficiencies in syndrome is exemplified by evolution's desire for variants. Even among the confined subtype ADHD-HI, biological process time appears to have resulted in an extended range of variability. This isn't to say that the search for explanations is futile: a foundation for high blood pressure factors has meticulously stripped away various tributary genes and interactions like a jaybird. Future additions to this field should concentrate on measuring the weights of the components rather than determining which of the numerous genes or neuro-psychological explanations aptly represent this syndrome. In most scientific disciplines and psychological medicine, equally advanced effort networks are still being investigated.

References

1. Sergeant J. The cognitive-energetic model: an empirical approach to attention-deficit hyperactivity disorder. *Neurosci Biobehav Rev.* 2000;24:7–12. [Doi:10.1016/S0149-7634\(99\)00060-3](https://doi.org/10.1016/S0149-7634(99)00060-3) [PubMed] [Google Scholar]
2. Sonuga-Barke E.J. The dual pathway model of AD/HD: an elaboration of neurodevelopmental characteristics. *Neurosci Biobehav Rev.* 2003a;27:593–604. [Doi:10.1016/j.neubiorev.2003.08.005](https://doi.org/10.1016/j.neubiorev.2003.08.005) [PubMed] [Google Scholar]
3. Sagvolden T, Johansen E.B, Aase H, Russell V.A. A dynamic developmental theory of Attention-Deficit/Hyperactivity Disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. *Behav Brain Sci.* 2005;28(3):397–419. [PubMed] [Google Scholar] [Ref list]
4. Holene E, Nafstad I, Skaare J.U, Sagvolden T. Behavioural hyperactivity in rats following postnatal exposure to sub-toxic doses of polychlorinated biphenyl congeners 153 and 126. *Behav Brain Res.* 1998;94:213–224. [doi:10.1016/S0166-4328\(97\)00181-2](https://doi.org/10.1016/S0166-4328(97)00181-2) [PubMed] [Google Scholar]
5. Berger D.F, Lombardo J.P, Jeffers P.M, Hunt A.E, Bush B, Casey A, Quimby F. Hyperactivity and impulsiveness in rats fed diets supplemented with either Aroclor 1248 or PCB-contaminated St. Lawrence river fish. *Behav Brain Res.* 2001;126:1–11. [doi:10.1016/S0166-4328\(01\)00244-3](https://doi.org/10.1016/S0166-4328(01)00244-3) [PubMed] [Google Scholar]
6. Schettler T. Toxic threats to neurologic development of children. *Environ Health Perspect.* 2001;109:813–816. [PMC free article] [PubMed] [Google Scholar]
7. Joseph J. Not in their genes: a critical view of the genetics of attention-deficit hyperactivity disorder. *Dev Rev.* 2000;20:539–567. [doi:10.1006/drev.2000.0511](https://doi.org/10.1006/drev.2000.0511) [Google Scholar]
8. Timimi S, Taylor E. ADHD is best understood as a cultural construct. *Br J Psychiatry.* 2004;184:8–9. [doi:10.1192/bjp.184.1.8](https://doi.org/10.1192/bjp.184.1.8) [PubMed] [Google Scholar]
9. Prendergast M, Taylor E, Rapoport J.L, Bartko J, Donnelly M, Zametkin A, Ahearn M.B, Dunn G, Wieselberg H.M. The diagnosis of childhood hyperactivity. A U.S.–U.K. cross-national study of DSM-III and ICD-9. *J Child Psychol Psychiatry.* 1988;29:289–300. [PubMed] [Google Scholar]

10. Normand C. ADHD as a disorder of adaptation. *J. Am. Acad. Child Adolesc. Psychiatry*. 1998;37:797. [\[PubMed\]](#) [\[Google Scholar\]](#)
11. Timimi S, Taylor E. ADHD is best understood as a cultural construct. *Br. J. Psychiatry*. 2004;184:8–9. [doi:10.1192/bjp.184.1.8](https://doi.org/10.1192/bjp.184.1.8) [\[PubMed\]](#) [\[Google Scholar\]](#)
12. Bolhuis J.J, Macphail E.M. A critique of the neuroecology of learning and memory. *Trends Cogn. Sci.* 2001;5:426–433. [doi:10.1016/S1364-6613\(00\)01753-8](https://doi.org/10.1016/S1364-6613(00)01753-8) [\[PubMed\]](#) [\[Google Scholar\]](#)
13. Levy F, Hay D.A, McStephen M, Wood C, Waldman I. Attention-deficit hyperactivity disorder: a category or a continuum? Genetic analysis of a large-scale twin study. *J. Am. Acad. Child Adolesc. Psychiatry*. 1997;36:737–744. [doi:10.1097/00004583-199706000-00009](https://doi.org/10.1097/00004583-199706000-00009) [\[PubMed\]](#) [\[Google Scholar\]](#)
14. Swanson J.M, et al. Dopamine genes and ADHD. *Neurosci. Biobehav. Rev.* 2000;24:21–25. [doi:10.1016/S0149-7634\(99\)00062-7](https://doi.org/10.1016/S0149-7634(99)00062-7) [\[PubMed\]](#) [\[Google Scholar\]](#)
15. Gjone H, Stevenson J, Sundet J.M. Genetic influence on parent-reported attention-related problems in a Norwegian general population twin sample. *J. Am. Acad. Child Adolesc. Psychiatry*. 1996;35:588–596. [doi:10.1097/00004583-199605000-00013](https://doi.org/10.1097/00004583-199605000-00013) [\[PubMed\]](#) [\[Google Scholar\]](#)
16. Faraone S.V, Perlis R.H, Doyle A.E, Smoller J.W, Goralnick J.J, Holmgren M.A, Sklar P. Molecular genetics of attention-deficit/hyperactivity disorder. *Biol. Psychiatry*. 2005;57:1313–1323. [doi:10.1016/j.biopsych.2004.11.024](https://doi.org/10.1016/j.biopsych.2004.11.024) [\[PubMed\]](#) [\[Google Scholar\]](#)
17. Sklar P. Principles of haplotype mapping and potential applications to attention-deficit/hyperactivity disorder. *Biol. Psychiatry*. 2005;57:1357–1366. [doi:10.1016/j.biopsych.2005.01.005](https://doi.org/10.1016/j.biopsych.2005.01.005) [\[PubMed\]](#) [\[Google Scholar\]](#)
18. American Psychiatric Association. Diagnostic and statistical manual of mental disorders: DSM-IV-TR 2000. American Psychiatric Association; Washington, DC: 2000. [\[Google Scholar\]](#)
19. Leung P.W, Luk S.L, Ho T.P, Taylor E, Mak F.L, Bacon-Shone J. The diagnosis and prevalence of hyperactivity in Chinese schoolboys. *Br. J. Psychiatry*. 1996;168:486–496. [\[PubMed\]](#) [\[Google Scholar\]](#)

20. Bird H.R. Epidemiology of childhood disorders in a cross-cultural context. *J. Child Psychol. Psychiatry*. 1996;37:35–49. [\[PubMed\]](#) [\[Google Scholar\]](#)
21. Leung P.W, Luk S.L, Ho T.P, Taylor E, Mak F.L, Bacon-Shone J. The diagnosis and prevalence of hyperactivity in Chinese schoolboys. *Br. J. Psychiatry*. 1996;168:486–496. [\[PubMed\]](#) [\[Google Scholar\]](#)
22. Leung P.W, Luk S.L, Ho T.P, Taylor E, Mak F.L, Bacon-Shone J. The diagnosis and prevalence of hyperactivity in Chinese schoolboys. *Br. J. Psychiatry*. 1996;168:486–496. [\[PubMed\]](#) [\[Google Scholar\]](#)
23. Rohde L.A, Biederman J, Busnello E.A, Zimmermann H, Schmitz M, Martins S, Tramontina S. ADHD in a school sample of Brazilian adolescents: a study of prevalence, comorbid conditions, and impairments. *J. Am. Acad. Child Adolesc. Psychiatry*. 1999;38:716–722. [doi:10.1097/00004583-199906000-00018](https://doi.org/10.1097/00004583-199906000-00018) [\[PubMed\]](#) [\[Google Scholar\]](#)
24. Willcutt E.G, Pennington B.F, Chhabildas N.A, Friedman M.C, Alexander J. Psychiatric comorbidity associated with DSM-IV ADHD in a nonreferred sample of twins. *J. Am. Acad. Child Adolesc. Psychiatry*. 1999;38:1355–1362. [doi:10.1097/00004583-199911000-00009](https://doi.org/10.1097/00004583-199911000-00009) [\[PubMed\]](#) [\[Google Scholar\]](#)
25. Hinshaw S.P, Carte E.T, Sami N, Treuting J.J, Zupan B.A. Preadolescent girls with attention-deficit/hyperactivity disorder: II. Neuropsychological performance in relation to subtypes and individual classification. *J. Consult Clin. Psychol.* 2002;70:1099–1111. [doi:10.1037/0022-006X.70.5.1099](https://doi.org/10.1037/0022-006X.70.5.1099) [\[PubMed\]](#) [\[Google Scholar\]](#)
26. Pitcher T.M, Piek J.P, Hay D.A. Fine and gross motor ability in males with ADHD. *Dev. Med. Child Neurol.* 2003;45:525–535. [doi:10.1017/S0012162203000975](https://doi.org/10.1017/S0012162203000975) [\[PubMed\]](#) [\[Google Scholar\]](#)
27. Rucklidge J.J, Tannock R. Psychiatric, psychosocial, and cognitive functioning of female adolescents with ADHD. *J. Am. Acad. Child Adolesc. Psychiatry*. 2001;40:530–540. [doi:10.1097/00004583-200105000-00012](https://doi.org/10.1097/00004583-200105000-00012) [\[PubMed\]](#) [\[Google Scholar\]](#)
28. Conners C.K, Epstein J.N, Angold A, Klaric J. Continuous performance test performance in a normative epidemiological sample. *J. Abnorm. Child Psychol.* 2003;31:555–562. [doi:10.1023/A:1025457300409](https://doi.org/10.1023/A:1025457300409) [\[PubMed\]](#) [\[Google Scholar\]](#)
29. Kooij J.J, Buitelaar J.K, Van den Oord E.J, Furer J.W, Rijnders C.A, Hodiamont P.P. Internal and external validity of attention-deficit hyperactivity disorder in a population-based sample

- of adults. *Psychol. Med.* 2005;35:817–827. [doi:10.1017/S003329170400337X](https://doi.org/10.1017/S003329170400337X) [PubMed] [Google Scholar]
30. Gjone H, Stevenson J, Sundet J.M. Genetic influence on parent-reported attention-related problems in a Norwegian general population twin sample. *J. Am. Acad. Child Adolesc. Psychiatry.* 1996;35:588–596. [doi:10.1097/00004583-199605000-00013](https://doi.org/10.1097/00004583-199605000-00013) [PubMed] [Google Scholar]
31. Jensen P.S, Mrazek D, Knapp P.K, Steinberg L, Pfeffer C, Schowalter J, Shapiro T. Evolution and revolution in child psychiatry: ADHD as a disorder of adaptation. *J. Am. Acad. Child Adolesc. Psychiatry.* 1997;36:1672–1679. [doi:10.1097/00004583-199712000-00015](https://doi.org/10.1097/00004583-199712000-00015) [PubMed] [Google Scholar]
32. Burt S.A, McGue M, Iacono W, Comings D, MacMurray J. An examination of the association between DRD4 and DRD2 polymorphisms and personality traits. *Pers. Indiv. Differ.* 2002;33:849–859. [doi:10.1016/S0191-8869\(01\)00194-5](https://doi.org/10.1016/S0191-8869(01)00194-5) [Google Scholar]
33. Kluger A.N, Siegfried Z, Ebstein R.P. A meta-analysis of the association between DRD4 polymorphism and novelty seeking. *Mol. Psychiatry.* 2002;7:712–717. [doi:10.1038/sj.mp.4001082](https://doi.org/10.1038/sj.mp.4001082) [PubMed] [Google Scholar]
34. Grady D.L, et al. High prevalence of rare dopamine receptor D4 alleles in children diagnosed with attention-deficit hyperactivity disorder. *Mol. Psychiatry.* 2003;8:536–545. [doi:10.1038/sj.mp.4001350](https://doi.org/10.1038/sj.mp.4001350) [PubMed] [Google Scholar]
35. Rogers G, Joyce P, Mulder R, Sellman D, Miller A, Allington M, Olds R, Wells E, Kennedy M. Association of a duplicated repeat polymorphism in the 5'-untranslated region of the DRD4 gene with novelty seeking. *Am. J. Med. Genet.* 2004;126B:95–98. [doi:10.1002/ajmg.b.20133](https://doi.org/10.1002/ajmg.b.20133) [PubMed] [Google Scholar]
36. Diaz-Anzaldua A, Joober R, Riviere J.B, Dion Y, Lesperance P, Richer F, Chouinard S, Rouleau G.A. Tourette syndrome and dopaminergic genes: a family-based association study in the French Canadian founder population. *Mol. Psychiatry.* 2004;9:272–277. [PubMed] [Google Scholar]
37. Lopez L.S, Croes E.A, Sayed-Tabatabaei F.A, Claes S, Van B.C, van Duijn C.M. The dopamine D4 receptor gene 48-base-pair-repeat polymorphism and mood disorders: a meta-analysis. *Biol. Psychiatry.* 2005;57:999–1003. [doi:10.1016/j.biopsych.2005.01.030](https://doi.org/10.1016/j.biopsych.2005.01.030) [PubMed] [Google Scholar]

38. Chang F.M, Kidd J.R, Livak K.J, Pakstis A.J, Kidd K.K. The world-wide distribution of allele frequencies at the human dopamine D4 receptor locus. *Hum. Genet.* 1996;98:91–101. [doi:10.1007/s004390050166](https://doi.org/10.1007/s004390050166) [PubMed] [Google Scholar]
39. Leung P.W, Luk S.L, Ho T.P, Taylor E, Mak F.L, Bacon-Shone J. The diagnosis and prevalence of hyperactivity in Chinese schoolboys. *Br. J. Psychiatry.* 1996;168:486–496. [PubMed] [Google Scholar]
40. Chang F.M, Kidd J.R, Livak K.J, Pakstis A.J, Kidd K.K. The world-wide distribution of allele frequencies at the human dopamine D4 receptor locus. *Hum. Genet.* 1996;98:91–101. [doi:10.1007/s004390050166](https://doi.org/10.1007/s004390050166) [PubMed] [Google Scholar]
41. Ding Y.C, et al. Evidence of positive selection acting at the human dopamine receptor D4 gene locus. *Proc. Natl Acad. Sci. USA.* 2002;99:309–314. [doi:10.1073/pnas.012464099](https://doi.org/10.1073/pnas.012464099) [PMC free article] [PubMed] [Google Scholar]
42. Swanson J, Moyzis R.K, Fossella J, Fan J, Posner M. Adaptationism and molecular biology: an example based on ADHD. *Behav. Brain Sci.* 2002;25:530–531. [doi:10.1017/S0140525X02490097](https://doi.org/10.1017/S0140525X02490097) [Google Scholar]
43. Wang E, et al. The genetic architecture of selection at the human dopamine receptor D4 (DRD4) gene locus. *Am. J. Hum. Genet.* 2004;74:931–944. [doi:10.1086/420854](https://doi.org/10.1086/420854) [PMC free article] [PubMed] [Google Scholar]
44. Wang E, et al. The genetic architecture of selection at the human dopamine receptor D4 (DRD4) gene locus. *Am. J. Hum. Genet.* 2004;74:931–944. [doi:10.1086/420854](https://doi.org/10.1086/420854) [PMC free article] [PubMed] [Google Scholar]
45. Jensen P.S, et al. Findings from the NIMH multimodal treatment study of ADHD (MTA): implications and applications for primary care providers. *J. Dev. Behav. Pediatr.* 2001;22:60–73. [PubMed] [Google Scholar]
46. Mannuzza S, Klein R.G. Long-term prognosis in attention-deficit/hyperactivity disorder. *Child Adolesc. Psychiatr. Clin. N. Am.* 2000;9:711–726. [PubMed] [Google Scholar]
47. DeBar L.L, Lynch F.L, Boles M. Healthcare use by children with attention deficit/hyperactivity disorder with and without psychiatric comorbidities. *J. Behav. Health Serv. Res.* 2004;31:312–323. [doi:10.1097/00075484-200407000-00007](https://doi.org/10.1097/00075484-200407000-00007) [PubMed] [Google Scholar]

48. Harpending H, Cochran G. In our genes. Proc. Natl Acad. Sci. USA. 2002;99:10–12. [doi:10.1073/pnas.012612799](https://doi.org/10.1073/pnas.012612799) [PMC free article] [PubMed] [Google Scholar]
49. Lakoff A. Adaptive will: the evolution of attention deficit disorder. J. Hist. Behav. Sci. 2000;36:149–169. [doi:10.1002/\(SICI\)1520-6696\(200021\)36:2<149::AID-JHBS3>3.0.CO;2-9](https://doi.org/10.1002/(SICI)1520-6696(200021)36:2<149::AID-JHBS3>3.0.CO;2-9) [PubMed] [Google Scholar]
50. Leth-Steensen C, Elbaz Z.K, Douglas V.I. Mean response times, variability, and skew in the responding of ADHD children: a response time distributional approach. Acta Psychol. (Amst) 2000;104:167–190. [doi:10.1016/S0001-6918\(00\)00019-6](https://doi.org/10.1016/S0001-6918(00)00019-6) [PubMed] [Google Scholar]
51. Sagvolden T, Johansen E.B, Aase H, Russell V.A. A dynamic developmental theory of Attention-Deficit/Hyperactivity Disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes. Behav. Brain Sci. 2005;28(3):397–419. [PubMed] [Google Scholar]
52. Saldana L, Neuringer A. Is instrumental variability abnormally high in children exhibiting ADHD and aggressive behavior? Behav. Brain Res. 1998;94:51–59. [doi:10.1016/S0166-4328\(97\)00169-1](https://doi.org/10.1016/S0166-4328(97)00169-1) [PubMed] [Google Scholar]
53. Glimcher P.W. Indeterminacy in brain and behavior. Annu. Rev. Psychol. 2005;56:25–56. [doi:10.1146/annurev.psych.55.090902.141429](https://doi.org/10.1146/annurev.psych.55.090902.141429) [PubMed] [Google Scholar]
54. Williams J, Dayan P. Dopamine, learning and impulsivity: a biological account of attention-deficit/hyperactivity disorder. J. Child Adolesc. Psychopharmacol. 2005;15:160–179. [doi:10.1089/cap.2005.15.160](https://doi.org/10.1089/cap.2005.15.160) [PubMed] [Google Scholar]
55. Hartmann T. Underwood-Miller; Lancaster, UK: 1993. Attention deficit disorder: a different perception. [Google Scholar]
56. Matejcek Z. Is ADHD adaptive or non-adaptive behavior? Neuroendocrinol. Lett. 2003;24:148–150. [PubMed] [Google Scholar]
57. Shelley-Tremblay J.F, Rosen L.A. Attention deficit hyperactivity disorder: an evolutionary perspective. J. Genet. Psychol. 1996;157:443–453. [PubMed] [Google Scholar]
58. Befera M.S, Barkley R.A. Hyperactive and normal girls and boys: mother-child interaction, parent psychiatric status and child psychopathology. J. Child Psychol. Psychiatry. 1985;26:439–452. [PubMed] [Google Scholar]

59. Blair C. Early intervention for low birth weight, preterm infants: the role of negative emotionality in the specification of effects. *Dev. Psychopathol.* 2002;14:311–332. [doi:10.1017/S0954579402002079](https://doi.org/10.1017/S0954579402002079) [PubMed] [Google Scholar]
60. Tully L.A, Arseneault L, Caspi A, Moffitt T.E, Morgan J. Does maternal warmth moderate the effects of birth weight on twins' attention-deficit/hyperactivity disorder (ADHD) symptoms and low IQ? *J. Consult Clin. Psychol.* 2004;72:218–226. [doi:10.1037/0022-006X.72.2.218](https://doi.org/10.1037/0022-006X.72.2.218) [PubMed] [Google Scholar]
61. Tims F.M, Dennis M.L, Hamilton N, Buchan J, Diamond G, Funk R, Brantley L.B. Characteristics and problems of 600 adolescent cannabis abusers in outpatient treatment. *Addiction.* 2002;97:46–57. [doi:10.1046/j.1360-0443.97.s01.7.x](https://doi.org/10.1046/j.1360-0443.97.s01.7.x) [PubMed] [Google Scholar]
62. Zaromatis K, Carlo R, Racanello D. Sex, perceptions of attractiveness, and sensation seeking and ratings of the likelihood of having sexually transmitted diseases. *Psychol. Rep.* 2004;94:633–636. [PubMed] [Google Scholar]
63. Jensen P.S, et al. Findings from the NIMH multimodal treatment study of ADHD (MTA): implications and applications for primary care providers. *J. Dev. Behav. Pediatr.* 2001;22:60–73. [PubMed] [Google Scholar]
64. Puente F.G. Family planning for psychiatric patients. *IPPF. Med. Bull.* 1977;11:2–4. [PubMed] [Google Scholar]
65. El Sayed E, Larsson J.O, Persson H.E, Santosh P.J, Rydelius P.A. Maturational lag hypothesis of attention deficit hyperactivity disorder: an update. *Acta Paediatr.* 2003;92:776–784. [doi:10.1080/08035250310002777](https://doi.org/10.1080/08035250310002777) [PubMed] [Google Scholar]
66. MacDonald K. Evolution and development. In: Campbell A, Muncer S, editors. Social development. UCL Press; London: 1998. pp. 21–49. [Google Scholar]
67. Rucklidge J.J, Tannock R. Psychiatric, psychosocial, and cognitive functioning of female adolescents with ADHD. *J. Am. Acad. Child Adolesc. Psychiatry.* 2001;40:530–540. [doi:10.1097/00004583-200105000-00012](https://doi.org/10.1097/00004583-200105000-00012) [PubMed] [Google Scholar]
68. Li C.S, Chen S.H, Lin W.H, Yang Y.Y. Attentional blink in adolescents with varying levels of impulsivity. *J. Psychiatr. Res.* 2005;39:197–205. [doi:10.1016/j.jpsychires.2004.06.003](https://doi.org/10.1016/j.jpsychires.2004.06.003) [PubMed] [Google Scholar]

69. Barkley R.A. The executive functions and self-regulation: an evolutionary neuropsychological perspective. *Neuropsychol Rev.* 2001b;11:1–29. [doi:10.1023/A:1009085417776](https://doi.org/10.1023/A:1009085417776) [PubMed] [Google Scholar]
70. Baldwin J.M. A new factor in evolution. *Am. Nat.* 1896;30:441–451. [doi:10.1086/276408](https://doi.org/10.1086/276408) [Google Scholar]
71. Hinton G.E. How learning can guide evolution. *Complex Syst.* 1987;1:495–502. [Google Scholar]
72. Hinton G.E. How learning can guide evolution. *Complex Syst.* 1987;1:495–502. [Google Scholar]
73. Aoki K. Theoretical and empirical aspects of gene-culture coevolution. *Theor. Popul. Biol.* 2001;59:253–261. [doi:10.1006/tpbi.2001.1518](https://doi.org/10.1006/tpbi.2001.1518) [PubMed] [Google Scholar]
74. Fehr E, Fischbacher U. The nature of human altruism. *Nature.* 2003;425:785–791. [doi:10.1038/nature02043](https://doi.org/10.1038/nature02043) [PubMed] [Google Scholar]
75. Nowak M.A, Sigmund K. Evolutionary dynamics of biological games. *Science.* 2004;303:793–799. [doi:10.1126/science.1093411](https://doi.org/10.1126/science.1093411) [PubMed] [Google Scholar]
76. Smith J.M. When learning guides evolution. *Nature.* 1987;329:761–762. [doi:10.1038/329761a0](https://doi.org/10.1038/329761a0) [PubMed] [Google Scholar]
77. Colman A.M, Wilson J.C. Antisocial personality disorder: an evolutionary game theory analysis. *Legal Criminol. Psychol.* 1997;2:23–34. [Google Scholar]
78. Dall S, Houston A.I, McNamara J.M. The behavioural ecology of personality: consistent individual differences from an adaptive perspective. *Ecol. Lett.* 2004;7:734–739. [doi:10.1111/j.1461-0248.2004.00618.x](https://doi.org/10.1111/j.1461-0248.2004.00618.x) [Google Scholar]
79. Rogers A.R. Does biology constrain culture? *Am. Anthropol.* 1988;90:819–831. [doi:10.1525/aa.1988.90.4.02a00030](https://doi.org/10.1525/aa.1988.90.4.02a00030) [Google Scholar]
80. Wakano J.Y, Aoki K, Feldman M.W. Evolution of social learning: a mathematical analysis. *Theor. Popul. Biol.* 2004;66:249–258. [doi:10.1016/j.tpb.2004.06.005](https://doi.org/10.1016/j.tpb.2004.06.005) [PubMed] [Google Scholar]
81. Andrews P.W, Gangestad S.W, Matthews D. Adaptationism—how to carry out an exaptationist program. *Behav. Brain Sci.* 2002;25:489–504. [doi:10.1017/S0140525X02000092](https://doi.org/10.1017/S0140525X02000092) [PubMed] [Google Scholar]

82. Deshmukh, Tejaswini, Ashish Varma, Sachin Damke, and Revat Meshram. "Predictive Efficacy of Pediatric Logistic Organ Dysfunction-2 Score in Pediatric Intensive Care Unit of Rural Hospital." *INDIAN JOURNAL OF CRITICAL CARE MEDICINE* 24, no. 8 (August 2020): 701–4. <https://doi.org/10.5005/jp-journals-10071-23528>.
83. Jameel, Patel Zeeshan, Sham Lohiya, Amol Dongre, Sachin Damke, and Bhavana B. Lakhkar. "Concurrent Diabetic Ketoacidosis and Pancreatitis in Paediatric Acute Lymphoblastic Leukemia Receiving L-Asparaginase." *BMC PEDIATRICS* 20, no. 1 (May 18, 2020). <https://doi.org/10.1186/s12887-020-02136-3>.
84. Khandare, Kiran, and Pradnya Ghormode. "Prolapsed Rectal Submucosal Hematoma in Pediatric Case." *PAN AFRICAN MEDICAL JOURNAL* 37 (October 13, 2020). <https://doi.org/10.11604/pamj.2020.37.154.26149>.
85. Taksande, Amar. "Myocardial Dysfunction in SARS-CoV-2 Infection in Infants under 1 Year of Age." *WORLD JOURNAL OF PEDIATRICS* 16, no. 5 (October 2020): 539. <https://doi.org/10.1007/s12519-020-00384-y>.
86. Yadav, Prachi, Sahil Dhaka, Richa Chaudhary, Sachin Damke, and Sham Lohiya. "A Rare Case Report of Guillain-Barre Syndrome Presenting as Unilateral Facial Palsy with Isolated Acute Bulbar Palsy." *JOURNAL OF PEDIATRIC NEUROSCIENCES* 15, no. 2 (June 2020): 157–59. https://doi.org/10.4103/jpn.JPN_129_19.
87. Anjankar, Shailendra D. "Urethral Protrusion of the Distal End of Shunt." *JOURNAL OF PEDIATRIC NEUROSCIENCES* 13, no. 3 (September 2018): 371–72. https://doi.org/10.4103/jpn.JPN_54_18.