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Research Article

Differential Reproductive Timing And The Frequency Of ADHD.

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Abstract

Twin studies indicate that ADHD is a strongly genetic disorder. The prevalence of genetic diseases is generally considered to be set and unchangeable. Some studies claim ADHD has increased in prevalence. Environmental factors such as changes in diagnostic criteria, increased awareness, and improved detection have been proposed to explain this increase. A demographic model is presented, showing that the earlier mean age of reproduction among individuals with ADHD versus those without ADHD, can result long term in a significant increase in the prevalence of ADHD.

INTRODUCTION

Is ADHD increasing in frequency? Polanczyk et al, (2014)[1] identified 135 original studies and concluded that variability in ADHD prevalence estimates was mostly explained by methodological characteristics of the studies and in the past three decades, there has been no evidence to suggest an increase in the number of children in the community who meet criteria for ADHD, when standardized diagnostic procedures were followed. Li et al (2022)[2] reviewed a total of 37,609 individuals aged 4 to 17 years across multiple racial groups. Among them 10.90% were reported to have ever been diagnosed with ADHD. All subgroups evaluated showed no significant change in prevalence from 2017 to 2022. Martin et al (2025)[3] examined forty studies across 17 countries, with one study spanning 42 countries. No significant rise in ADHD prevalence was found.

In contrast, several studies have suggested that the prevalence of ADHD has increased.Yu et al, (2018)[4] reported on the National Health Interview Survey, a nationwide, population-based, cross-sectional survey conducted annually from 1997 to 2016. The data were derived from 186,457 children and adolescents aged 4 to17 years. The estimated prevalence of diagnosed attention-deficit/hyperactivity disorder in US children and adolescents increased from 6.1% in 1997-1998 to 10.2% in 2015-2016.

Another National Survey by Abdennour et al (2022)[5] was based on parent-report data from National Survey of Children's Health (NSCH). The prevalence of ADHD in U.S. children increased from 7.8% in 2003 to 11.0% in 2011.

The CDC reported that in 2022, an estimated 11.4 % of U.S. children (ages 3–17) had ever been diagnosed with ADHD, representing about 1 million more than diagnosed in 2016.[6,7] Vesser et al (2014)[8] stated that an estimated two million more U.S. children aged 4 – 17 years had been diagnosed with ADHD in 2011 compared to 2003. More than two-thirds of them were taking medication indicating, an increasing burden of ADHD in the U.S. They stated that efforts understand this increase are warranted. The current hypothesis provides a mechanism for a true increase in ADHD prevalence.

ADHD is strongly genetic

One of the most powerful methods of determining if a disorder is partly or largely genetic, is twin studies. These studies ask what the concordance rate is in identical versus fraternal twins. Identical twins have 100% of their genes in common, while fraternal twins have 50% in common. Twin studies of ADHD show MZ concordance rates of 50-82% and DZ rates of 0 – 40%.[9-11] This indicates that ADHD is strongly genetic. Many genome-wide association studies (GWA) studies of ADHD have been reported in attempts to identify what genes

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were involved. Most have reported that none of the SNPs studied had reached the level of significance recommended for GWS studies.

In contrast to these negative studies, a genome-wide association meta-analysis of 20,183 individuals diagnosed with ADHD and 35,191 controls by Demontis et al (2019)[12] identified variants surpassing genome-wide significance in 12 independent loci. They also found support for a genetic overlap with educational attainment.

Sánchez-Mora C. et al. (2015)[13] identified FBXO33 as a novel susceptibility gene for ADHD. Hawi Z. et al (2018)[14] found an association between ADHD and the Tenascin R (TNR) gene ($p = 3.15E^{-08}$) which is part of a gene family for extracellular matrix glycoproteins that play a role in neural cell adhesion and neurite outgrowth. In addition, they found suggestive evidence of associations with ADHD was observed for an additional 111 genes at ($<9.91E^{-05}$). In a GWS of adult ADHD subjects, Lesch et al (2008)[15] identified novel risk genes and revealed a remarkable overlap with findings from recent GWA scans in substance use disorders.

An extensive genetic study by Grotzinger et al (2025)[16] of over a million subjects with 14 different psychiatric disorders showed considerable genetic overlap between different disorders. They identified 5 major groupings (F1 to F5) with each representing a specific subset of disorders. F3 included two disorders, ADHD and autism spectrum disorder, which showed a genetic correlation of 0.426.

Risk of Transmission of ADHD

A relevant question is – what percent of children of a parent with ADHD develop ADHD? Research studies using a structured diagnostic assessment show that approximately 40 % of children with a parent diagnosed with ADHD meet full DSM diagnostic criteria for the disorder.

Uchida et al, (2021)[17] pooled data from four studies of offspring whose biological parent had clinically diagnosed ADHD and found a combined prevalence of ADHD in children of ~38 % (95 % CI 8–68 %). When an EMR (Electronic Medical Record)-based study that likely underestimated diagnoses was removed, the prevalence increased to ~52 % (95 % CI 43–61 %). In a second study by Uchida et al (2022)[18] of a large at-risk sample, 36% of at-risk children had full or subsyndromal ADHD. Minde et al (1990)[19] observed that ~43 % of children of parents with ADHD met DSM criteria for ADHD, while Joseph et al. (2020)[20] found ~40 % met criteria.

Educational Achievement (EA) is Also Strongly Genetic

Since EA showed an even greater range of years at first childbearing for those scoring low verses high on EA (see below) the genetics of EA is also of interest. Summarizing multiple studies[21-24] showed MZ correlations were consistently ~0.75–0.90, while DZ correlations were ~0.40–0.60, implying a heritability of ~55–70%

There have been several Genome Wide Association Studies (GWAS) studies of EA. They involve large numbers of subjects since large data bases with EA data are available. Rietveld et al (2013)[25] performed GWAS on 101,069 individuals for which data on educational achievement was available plus an additional replication study involving 25,490 subjects. They identified 3 genome-wide significant SNPs for EA. A polygenic score explained ~2% of variance in EA. A second study by Okbay, A. et al. (2016)[26] involved a meta-analysis of 293,723 subjects with data on EA and a replication sample of 111,349 UK Biobank samples. They identified 74 genome-wide significant loci for EA (years of schooling). A third study by Lee et al (2018)[27] involved 1,131,881 subjects (69 cohorts + UK Biobank + 23andMe). They identified 1,271 approximately independent genome-wide significant SNPs. Polygenic scores explained ~11–13% of variance in EA in European-ancestry samples. The SNP results implicated genes involved in brain-development processes and neuron-to-neuron communication. A fourth study, also by Okbay, A. et al. (2022)[28] had a combined N of 3,037,499 from expanded 23andMe and UK Biobank samples. They identified 3,952 approximately uncorrelated genome-wide significant SNPs for EA. A polygenic score explained ~12–16% of variance in EA. These studies show that EA has a significant genetic component that involved many genes.

In the UK, tests are given at the end of compulsory schooling. These are called GCSE. Krapohl et al (2014)[29] examined the scores that were given to 13,303 twins at age 16. They found that the English and mathematics scores alone had a heritability of 62%. Although specific intelligence test scores accounted for more of the heritability of GCSE than any other single domain, the other domains collectively accounted for about as much GCSE heritability as intelligence. These other domain scores included traits such as such as self-efficacy, personality, well-being, and behavior problems, and had a heritability of 35 to 58%. Together with intelligence, these domains accounted for 75% of the heritability of GCSE. Even though these tests were given at the end of compulsory schooling and thus are not quite equivalent to EA, which covers a longer period, they do illustrate the very large role of genetics in education achievement.

Age of First Childbirth: ADHD

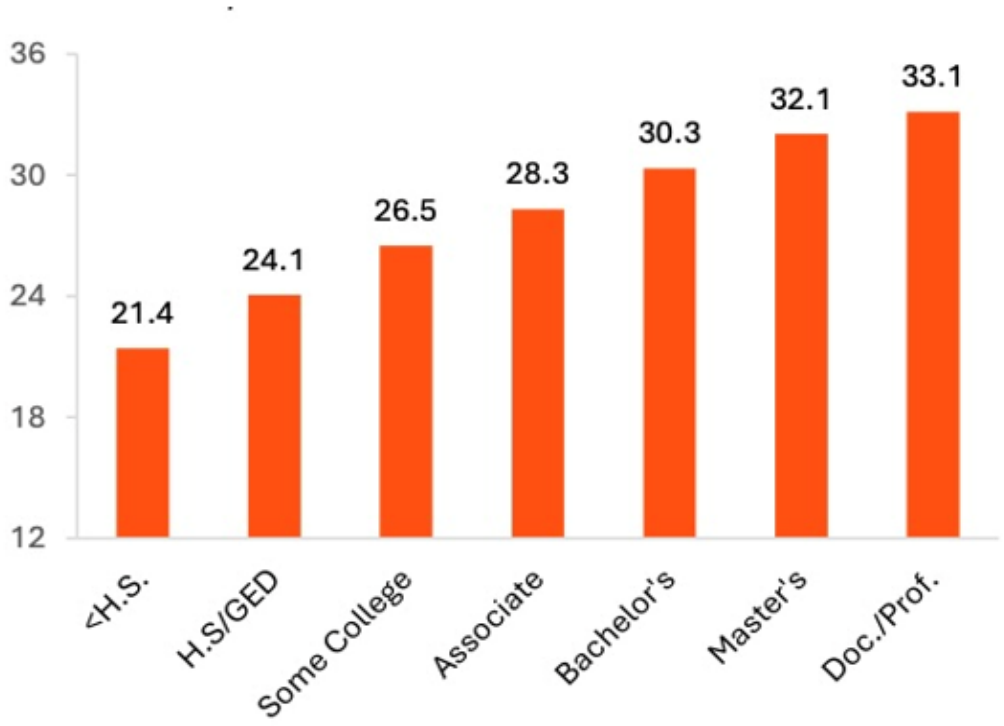
Across large Scandinavian registry studies Ostergaard et al (2017) [30] found that subjects with ADHD consistently had an earlier age of entry into parenthood, mainly driven by a much higher fraction of teenage first births. Females had a higher frequency of beginning childbirth compared to controls at ages 12 to 16, 17 to 19 and 20 to 24, while controls were more likely to begin having children than ADHD subjects in the 25 to 29, 30 to 34 and 35+ age groups. Males were more likely to

begin having children in the 12 to 16 and 17 to 19 age group while the controls were more likely begin having children in the 25 to 29, 30 to 34 and 35+ age groups. Both females and males with ADHD were more likely than controls to have multiple children. In another Swedish study by Skoglung et al (2019)[31] teenage deliveries were 6 times more common among women and girls with ADHD than among women and girls without ADHD (15.3% vs 2.8%: odds ratio 6.23. Two other studies [32,33] also showed a significantly increased risk of teen age pregnancy for ADHD women than controls.

It is also relevant to examine the age of first childbearing in individuals ranking low versus high on EA (Educational Achievement). There is a strong association of ADHD with poor educational achievement.[34-40]

In a study of EA by Westrick-Payne & Wendy (2023)[41] in the U.S. (see **Figure 1**) the mean age at first birth was 21.4 years for those who did not complete high school. There was a progressive increase of mean age at first childbirth across increasing EA to 33.1 years for those with a doctor's degree. Thus, there was an 11.7-year difference between age of first birth for those students who did not complete high school versus those who attained a Ph.D. degree.

Figure 1. Mother's age at first birth vs Educational Achievement. National Center for Marriage and Family Research (NCFMR), Bowling Green State University. Authors Krista K. Westrick-Payne & Wendy D. Manning.



Source: NCFMR analysis of National Center for Health Statistics, National Vital Statistics System, natality date file, 2023

Other studies also relate poor educational achievement with earlier age of childbearing. Livingston, G. (2015)[42] of the Pew research Center reported that among women ages 40 to 50, the median age at which those with a master's degree or more first became mothers was 30. In comparison, the median age at first birth for women with a high school diploma or less was just 24. Women with a college degree are more than twice as likely as those who never attended college to have no children. Bhrolcháin, M. et al (2012)[43] reported that women leaving education at ≤16 years of age had first births ~6–8 years earlier than women completing university. Those with low education had their first child at 21-23 years of age, while those with higher education had their first child at 29 to 31 years. Martin, et al (2021)[44] reported the following ages for the beginning of childbirth: less than high school ~22–23 years; high school ~24–25 years; bachelor's degree or higher ~30–31 years. Berrington A (2004)45 found that each additional year of education significantly delayed age at first birth. Women with tertiary education had first births ~7 years later than women with compulsory education only.

While low educational achievement and ADHD are not the same, as mentioned above, there is a strong association of ADHD with poor educational achievement.34-40 For example, In a population-based birth cohort, 22.9% of individuals with

childhood ADHD failed to complete high school, compared with 10.0% of matched controls.[37] In addition, children with ADHD demonstrate significantly lower grade point averages and lower standardized achievement scores in reading and mathematics relative to peers without ADHD, along with increased rates of grade retention and reduced post-secondary attainment.[33] There is also a high correlation between ADHD and Specific Learning Disablities.[46-48]

Collectively, these findings indicate that ADHD is substantially overrepresented among individuals with both low educational attainment (non-completion of high school) and persistent low academic performance. Figure 1 shows the dramatic relationship between age at first birth and EA.

In summary, different reports indicate a significant difference in age of first birth for those with versus without ADHD and a difference of up to 12 years between the age of imitation of childbirth for subjects with low versus high EA.

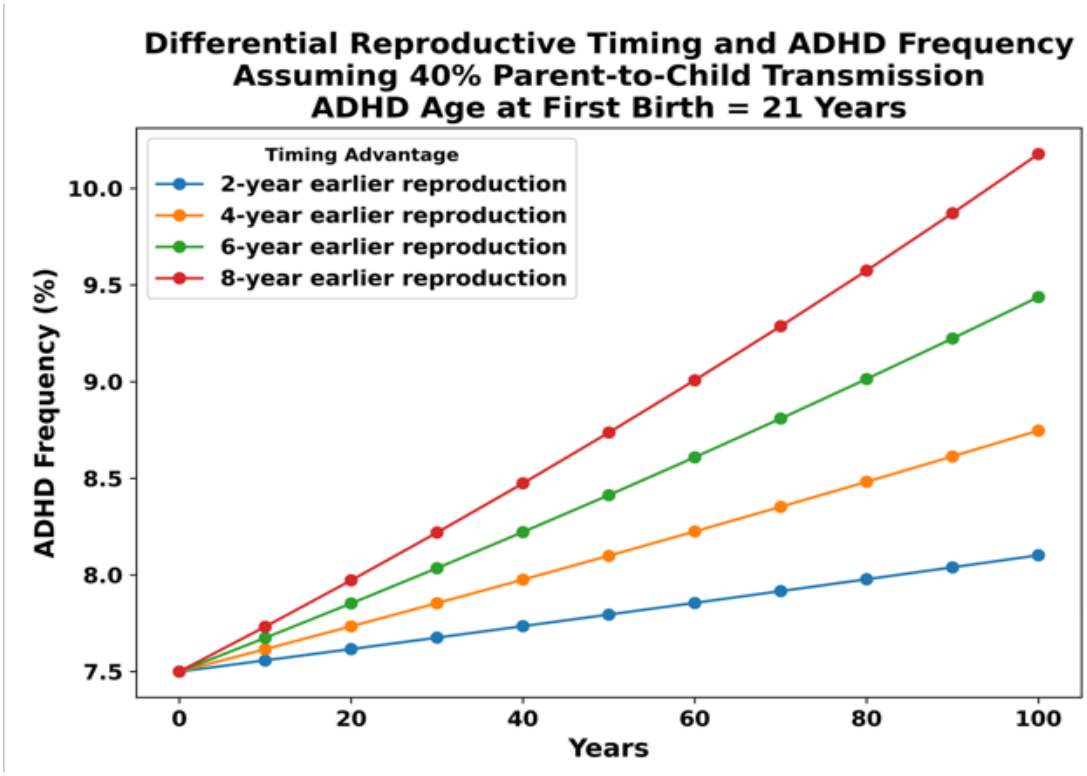
METHODS

AI in the form of ChatGPT was utilized to show in graphic form the effect of a difference of 2, 4, 6 and 8 years in the initiation of childbirth for ADHD subjects versus controls, on the prevalence of ADHD in over a period of 100 years, in 10-year increments, assuming a 40% transmission rate, and assuming the age of first childbirth for those with ADHD was 21 years (**Figure 2**). This model made several simplifying assumptions such as - for a single individual reproduction represented a doubling of the prevalence of ADHD, the difference in age of reproduction for those with ADHD versus controls remained constant over the subsequent generations, and no effort was made to include the effect of multiple children in the ADHD group or no children in the control group. The study was not meant to be highly precise. Its purpose is to illustrate a general principle relating reproductive timing to prevalence of ADHD over time.

RESULTS

The results when the age of first childbirth for those with ADHD was 21 years, and the age of the controls was 2, 4, 6, and 8 years later, and the rate of transmission was 40%, is shown in graphic form in Figure 2. A baseline frequency of 7.5% was used for all 4 groups. The results, when those ADHD subjects began having children at 24 years of age, were very similar. When ADHD individuals reproduce 2 years earlier than those of comparable age without ADHD, the frequency increases only modestly over a century (from 7.5% to ~8.1%). As reviewed above, greater reproductive spans between ADHD versus non-ADHD subjects are common and include 4 to 8-year differentials. A 4-year reproductive differential increases ADHD frequency to ~8.75% over 100 years, a 6-year differential to ~9.45%, and an 8-year differential to ~10.2%.

Figure 2. Projected change in the population frequency of ADHD over 100 years assuming four different reproductive timing differenrials of 2, 4, 6 and 8 years, for individuals with ADHD vs those without ADHD, with a transmission frequency of 40%, and 21 years as the age of onset of childbearing for ADHD subjects.



DISCUSSION

These findings illustrate how differential reproductive timing can act as a persistent evolutionary force shaping neurodevelopmental trait frequencies. Because reproduction operates multiplicatively across generations, even small reductions in mean age at reproduction for ADHD subjects versus controls, can produce long-term shifts in population frequency without requiring changes in environmental exposure or diagnostic practices.

Use of AI

The author used an AI-based language model as described in Methods to present the results in graphic form. All study concepts, scientific content, model design, interpretation, writing and conclusions were developed by the author.

Conflict of Interest and Funding

There were no conflicts of interest of any type. The study was done without funding.

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