HEDG

HEALTH, ECONOMETRICS AND DATA GROUP

THE UNIVERSITY of York

WP 24/06

The Impact of Genetic Risk for ADHD on Parental Breakdown

Alessio Gaggero; Joan Gil and Dolores Jiménez-Rubio

June 2024

http://www.york.ac.uk/economics/postgrad/herc/hedg/wps/

The Impact of Genetic Risk for ADHD on Parental Breakdown

Alessio Gaggero [a] Joan Gil [b] Dolores Jiménez-Rubio [c]

[a] Dept. of Quantitative Methods for Economics and Business, University of Granada, Spain
[b] Dept. of Economics, University of Barcelona, Spain
[c] Dept. of Applied Economics, University of Granada, Spain

June 2024

Abstract

Objectives: The literature shows a positive association between children with Attention Deficit and Hyperactivity Disorder (ADHD) and parental divorce or marital conflicts. This is a significant health policy issue because the behavioural disturbances of these children, which may persist for years, tend to increase parental stress, impacting their marital status, which in turn may negatively impact children's wellbeing. The objective of this paper is to estimate the causal impact of genetic risk for ADHD on parental divorce or separation.

Methods: We draw upon longitudinal data from the English Longitudinal Study of Ageing (ELSA), a panel of individuals aged 50+ and their partners in England. This dataset includes life history information on crucial events impacting people's health and quality of life, as well as genetic information which enables the use of polygenic scores (PGS) for various traits, including ADHD. Consistently with Mendel's laws, our identification strategy mimics a randomised evaluation and can be informative of causal effects.

Results and Conclusions: Based on different specifications and controlling for a wide set of observables, population stratification and time fixed effects, we report a positive significant influence of having a child with a high genetic risk for ADHD disorder on parental divorce. The estimated effects are particularly strong for individuals with a high genetic risk of developing ADHD, and for adolescents, but do not differ significantly by gender. We suggest that the family's economic and psychological burden arising from raising children with ADHD could be a potential channel for our findings. Our results remain robust across a number of alternative specifications.

Key words: ADHD, Neurodevelopmental Disorders, Parental Divorce, Polygenic Scores
JEL: I10, I12

Acknowledgements: The authors would like to thank the participants of the Alfasigma Monday Health Economics Seminar (University of Cantabria, April 2024) for the comments given to a previous version of this paper.

1. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a highly prevalent complex neurodevelopmental disorder, affecting approximately 5-7% of children globally (Vos & Hartman, 2022), with a slightly higher prevalence in the US, where approximately 9-10% of children are diagnosed with this disease (Bitsko RH. et al., 2022). Its manifestations, including pervasive inattention, hyperactivity and impulsivity not appropriate to the child's age, not only challenge the individuals diagnosed but also significantly impact their families, particularly the parents. While ADHD's effects on educational attainment and social interactions have been well documented, its broader implications for family dynamics, especially as concerns parental marital outcomes and psychological wellbeing, have garnered less attention.

Existing research underscores the profound influence of children's health and behaviour on parental life satisfaction (Balbo & Bolano, 2024; Peasgood et al., 2021), mental health (Chen et al., 2023; Leitch et al., 2019; Schermerhorn et al., 2012; Theule et al., 2013) and labour outcomes (Adhvaryu et al., 2022; Eriksen et al., 2021; Fletcher, 2014; Kvist et al., 2013; Lynch et al., 2023). Moreover, several studies have observed that in families of children with disabilities and health conditions, mothers are subject to a greater economic burden but both parents seem to experience equal levels of psychological distress (Adhvaryu et al., 2022; Balbo & Bolano, 2024; Eriksen et al., 2021; Theule et al., 2013). These challenges are believed to stem not only from the direct care demands imposed but also from the broader societal and economic implications of managing ADHD within the family unit.

Research exploring the relationship between children diagnosed with ADHD and the resultant effects on parental outcomes face several challenges. First, the methodology used to diagnose ADHD is inherently subjective and ADHD varies widely across US states and according to physician characteristics and to those of the patient, such as age or gender (Fulton et al., 2009; Furzer et al., 2022), a situation which can lead to misclassification and/or bias. Furthermore, the diagnosis of ADHD typically results in a binary categorisation—either a child is diagnosed or not—oversimplifying the condition's complex spectrum of symptom severity. Crucially, establishing causality is hindered due to reverse causality issues and the presence of confounding factors. The former refers to the difficulty in establishing whether ADHD in children directly leads to negative outcomes for parents, or conversely, whether certain parental conditions might

aggravate or even precipitate ADHD symptoms in children. For example, factors such as parental stress or marital conflict might affect both the behaviour of children and the reporting of symptoms, potentially leading to an ADHD diagnosis (Heckel et al., 2009; Burt et al., 2003). Moreover, a number of confounding variables—such as socioeconomic status, parental mental health and genetic predispositions—can simultaneously affect the probability of an ADHD diagnosis in children and various aspects of parental well-being.

In this paper, we explore the causal relationship between the genetic risk for ADHD, measured through polygenic risk scores (PGS) for ADHD, on parental divorce/separation in a sample of older adults living in England. Considering the genetic risk for ADHD, as opposed to its diagnosis, potentially avoids the abovementioned problems. Firstly, considering the limitations associated with the diagnosis, including subjectivity and binarisation, the genetic predisposition may offer a more objective and impartial metric for investigating this disorder. Additionally, it may also avoid the issues of reverse causality and confounding, as it is established at conception and, consistent with the fundamental principles of Mendel's laws, essentially mimics a randomised evaluation. This is grounded in the biological mechanism governing genetic inheritance, which creates a quasi-experimental scenario resembling the genetic lottery at conception, ensuring genetic diversity among siblings.

Using data drawn from the English Longitudinal Study of Ageing (ELSA), we provide evidence of a significant link between the genetic risk for ADHD and marital stability. Specifically, we find that a one standard deviation increase in the genetic risk for ADHD increases the probability that the parents will divorce/separate before the child reaches the age of sixteen by 1.4%. Our heterogeneity analysis suggests that the effects are stronger for individuals with a higher genetic risk of developing ADHD, and as children enter adolescence, but interestingly do not differ by gender. Additionally, we find that family disruptions due to the health-related problems or behaviour of children with ADHD might be a potential channel for our findings. Our results remain robust across a number of alternative specifications.

We contribute to the literature in several important ways. First, to the best of our knowledge, we provide the first comprehensive causal analysis of the impact of the genetic propensity to ADHD on divorce/separation. Second, as opposed to the majority of previous studies, which mainly focus on the impact of ADHD in terms of education and labour market outcomes (Currie & Stabile, 2006; Fletcher, 2014; Rajah et al., 2023), our analysis extends beyond these individual effects and explores the wider implications

of ADHD within the family context, a crucial aspect which has been overlooked in the economics literature. Hence, our study provides a more nuanced understanding of the disorder's societal and economic ramifications. Third, in addition to explicitly accounting for endogeneity issues, the use of PGS enables a more objective and accurate measure of the disorder's complexities than ADHD diagnosis. Fourth, compared to previous studies our rich data also allows for a better understanding of the timing of divorce relative to children's age. In fact, a number of previous studies in which preadolescent data were considered, and which found no association between ADHD and parental breakdown, failed to account for the probability of divorce over a longer time span (Wymbs et al., 2008). Furthermore, our study highlights the potential for integrating genetic insights into interventions and policies aimed at supporting families affected by ADHD, ultimately fostering resilience and reducing the burdens associated with the disorder.

The remainder of the paper is structured as follows: Section 2 reviews the relevant literature on the impact of ADHD on individuals and families, Section 3 describes the data source and presents descriptive statistics, Section 4 outlines the methodology employed, Section 5 discusses the findings and their implications, and Section 6 concludes with a summary of the results and considerations for future research.

2. Related Literature

Several studies have documented a positive association between children with ADHD and parental divorce or marital problems, although the particular underlying mechanisms remain unclear (Schermerhorn et al., 2012).[1] On the one hand, Brown & Pacini (1989) found a positive association between being a parent of children with ADHD and being separated or legally divorced. Wymbs et al. (2008) showed that parents of children with ADHD were more likely to be divorced than those of neurotypical children. These studies show that both parent and child characteristics appear to interact, increasing marital problems and hence marital dissolution within families whose children are diagnosed with ADHD. Similarly, Kvist et al. (2013) using Danish registry-based data of children born between 1990-1997 revealed that parents of a first-born child with ADHD had a 75% higher probability of experiencing marital dissolution within ten years of childbirth and 7-13% lower participation in the labour market. The effects on both

[1] For a systematic review of this issue see the study of Anchesi et al. (2023).

outcomes increase over the course of a ten-year period.[2] According to the authors, a plausible explanatory mechanism for the marital breakdown and occupational limitations of parents of children with ADHD could be the greater need of time to adequately manage the needs of these children. On the other hand, some earlier studies (e.g. Barkley et al., 1991; Schachar & Wachsmuth, 1991) found no divorce-related differences between ADHD parents and parents of neurotypical children. However, most of these studies are based on data for pre-adolescents and use a single assessment point, thus limiting the scope of the analysis to a short period of time (Wymbs et al., 2008).

In contrast to previous correlational studies, Schermerhorn et al. (2012) took a step forward in their analysis of marital stability by assuming that its association with children with ADHD might be due to shared genetic, family or environmental confounds, in addition to a reverse causality problem. Specifically, based on data from the Australian National Twin Register, they found that having children with ADHD (measured by symptoms or clinical diagnosis) raised parents' risks of marital conflict (divorce/separation), after accounting for common genetic and environment confounds.[3] The authors suggest higher levels of stress for parents with ADHD children as a potential channel for the results observed. Moreover, this is one of the first studies exploiting information on the timing of divorce finding that the mean age of ADHD children when their parents divorced was 10.99 years. However, these estimates may suffer from residual confounding from individual unobserved or twin-specific differences, such as innate ability or health endowments.

3. Data and Methods

Data

To investigate the relationship between parental divorce and children with ADHD, we draw upon longitudinal data from the *English Longitudinal Study of Ageing* (ELSA). This is a representative panel of people aged 50+ and their partners living in private households in England composed by ten waves of data spanning from wave 1 (years 2000-2001) to wave 10 (years 2021-2022) and covering a wide set of demographics, health and related behaviours, wellbeing, social participation, education,

[2] The richness of the dataset enabled us to control for observables (e.g., parental SES or mental health) measured before or at the time of the presence of ADHD symptoms.

[3] Results remained after excluding families in which separation/divorce preceded or co-occurred with ADHD onset, avoiding potential reverse causality issues.

work and pensions, income and assets, housing and cognitive functioning. ELSA is an unbalanced panel dataset comprising 108,005 individual-year observations corresponding to 19,854 individuals or respondents.

In waves 2, 4, 6, 8 and 9, the eligible respondents received a follow-up visit from a nurse to collect physical examination and performance data, to obtain blood samples for biochemical tests and to perform DNA extraction for genetic studies. Relevant to our investigation, ELSA provides detailed genetic data, consisting of a wide range of genetic variants. Specifically, ELSA participants of European ancestry were genotyped in 2013/14, using the Illumina HumanOmni2.5 Bead-Chips (HumanOmni2.5-4v1, HumanOmni2.5-8v1.3), which measures roughly 2.5 million markers that capture the genomic variation down to 2.5% minor allele frequency.[4] Principal components analysis was performed to investigate population structure, and ten principal components were retained to account for the genetic relatedness matrix (Price et al., 2006). A total of 7,183 units and 1,372,240 genetic variants remained after performing quality control (NatCen Social Research, 2022).

Interestingly, the "Life History Interview" in wave 3 compiled retrospective data on important events earlier in the lives of respondents that may have influenced their health status. This dataset included information on children, partners, childhood health (before and after age 16) and parental relationships when aged under 16. To minimise the possibility of recall bias, ELSA uses the "Life history calendar ("Lifegrid") designed to help individuals remember past events more accurately.[5]

Outcomes and Main Explanatory Variables

The outcome variable in this study is measured from the responses to the question "*Did your parents permanently separate or divorce before you were 16?*" contained in

[4] A full quality control protocol is described in https://www.elsa-project.ac.uk/genetics. Briefly, individuals with suspected non-European ancestry and heterozygosity scores >3 standard deviations from the mean were removed. Furthermore, initial quality control measures were conducted to test for duplicates and missingness of more than 2% of the genotype data. Single Nucleotide Polymorphisms (SNPs) with a call rate of <98%, a minor allele frequency of <1% and Hardy-Weinberg Equilibrium p values of $<10^{-4}$ were excluded. Non-autosomal markers were also removed, as well as regions known to contain clusters of highly correlated SNPs, as these may bias the analyses. The principal components analysis was employed to identify individuals who deviated from European ancestry (i.e., ethnic outliers). This set of analyses demonstrated the presence of an ancestral admixture in 65 individuals, who were excluded from the analysis; individuals who self-reported to be of non-European ethnicity were also removed.

[5] For a review of the interviewing technique used by the Life History Interview to aid recall and/or improve response consistency, see Ward et al. (2009).

the Life History Interview.[6] An indicator variable was derived equal to 1 if the respondent's parents divorced/separated before age 16 and 0 otherwise.

Our main explanatory variable is the polygenic score (PGS) for ADHD (NatCen Social Research, 2022).[7] Increasingly employed to predict disease risk, the PGS captures the genetic propensity to a given phenotype or trait.[8] The literature shows that the PGS for ADHD is reliable, robust and strongly associated with the diagnosis and traits (Ronald et al., 2021).

The use of PGS as our main explanatory variable presents several important advantages. First, consistent with the fundamental principles of Mendel's laws of inheritance, it mimics a randomised control trial and can therefore be informative of causal effects, as their parameter estimates are not generally confounded by behavioural or environmental exposures (Davey Smith & Ebrahim, 2005). Second, the PGS is not subject to typical reporting biases when diagnosis or symptoms are measured in questionnaire data (Fulton et al., 2009; Furzer et al., 2022). Moreover, it is more likely to accurately reflect the complex spectrum of symptom severity inherent to the ADHD condition than a binary 0-1 indicator.

The PGS is calculated as a weighted average across the total number of SNPs (Single Nucleotide Polymorphism) or genetic variants associated with a trait or condition, and the weights are the estimated effect size of the genetic variant on the trait of interest obtained from a Genome-Wide Association Study (GWAS). Specifically, the PGS for each individual *i* is calculated as:

$$PGS_i = \sum_{j=1}^{J} \beta_j SNP_{i,j} \qquad (i = 1 \ldots . N)\ (j = 1 \ldots . J) \qquad (1)$$

where $SNP_{i,j}$ is the allele frequency (0,1 or 2) for the *j-th* SNP of individual *i* and β_j is the meta-analysis effect size for SNP *j* and the trait.[9] To facilitate the interpretation

[6] Five response options are offered: 1 'yes', 2 'no', 3 'one or both parents died before respondent was 16', 4 'parents never lived together during respondent's lifetime, 5 'never lived with parents/don't know'.

[7] Information on self-reported ADHD diagnosis and/or symptoms is not included in the dataset. However, we take advantage of the fact that ELSA contains the PGS for a number of behavioural, emotional and health-related phenotypes instead.

[8] For instance, the PGS for ADHD has previously been used as the key explanatory variable in studies of its impact on later life labour outcomes (Rietveld & Patel, 2019) or on educational and cognitive outcomes (Stergiakouli et al., 2016).

[9] For a detailed description of how the PGS for ADHD is constructed see NatCen Social Research (2022) and Demontis et al. (2019).

of results, the PGS for ADHD is further standardised to have mean 0 and a standard deviation of 1.

Summary Statistics

Table 1 presents summary statistics of the sample of interest. Notably, 5% of the respondents' parents divorced before the respondents were 16. The main regressor of interest, the polygenic score for ADHD, had a mean value of -876.55, with a minimum of -1061 and a maximum of -679. As mentioned above, to ease the interpretation of the results, in the analysis that follows we will use the standardised polygenic score.[10]

In terms of sociodemographic characteristics, the average age of the sample respondents was around 68 years (standard deviation, SD, = 9.36); 55% were women and 69% were married; the average family was composed of two individuals (mean = 1.97; SD = 0.77). Only 17% achieved higher education and the (log-equivalised) household income was 5.65 (SD= 0.64). Note that 20% of respondents were wage employed, 5% were self-employed and 1% were unemployed.

The wealth of information reported in ELSA allows the retrieval of retrospective information on the respondents' parents. This is relevant because it allows us to account for a set of parental variables that could plausibly influence whether respondents' parents divorced during the respondent's childhood/adolescence. Specifically, we include variables that capture whether, when the respondent was younger than 16 years, one of their parents experienced major events such as financial hardship, a prolonged period of unemployment (exceeding six months), or had an addiction to tobacco or alcohol, or experienced mental health problems. Table 1 shows that 18% of individuals stated their parents had experienced severe financial hardship in the past, while 6% reported that during their childhood/adolescence, their parents were addicted to drugs or alcohol or had mental health problems. Moreover, a total of 7% reported that their parents had experienced long periods of unemployment when they were younger than 16 years.

Finally, we also include a set of variables describing the living conditions of the respondents when they were ten years old, including the number of books and bedrooms at home, as well as the family size during that period. On average, there were roughly 2.5 books in the family home, which had three bedrooms. During this period, the average family had five members.

[10] Figures A1 and A2 in the Appendix describe the full distribution of the polygenic score.

[TABLE 1 HERE]

Empirical framework

To estimate the effect of genetic risk for ADHD on parental outcome we use the following ordinary least-squares (OLS) model:[11]

$$Y_i = \alpha + \beta PGS_i + X_i'\gamma + \varepsilon_i \qquad (2)$$

where Y_i is the outcome of interest, a dummy variable taking the value of one if the respondents' parents divorced before the respondent was 16 years old. PGS_i is the polygenic score for ADHD, and β is the main term of interest, i.e. the impact of the genetic risk of ADHD on parental divorce. X_i stands for the vector of controls (some of which are time varying, while others are time invariant) and ε_i is the error term.[12]

Note that reverse causality is not an issue here, as the decision of parental divorce/separation cannot affect the PGS, which is determined at conception. This is consequence of the random assignment of an individual's genotype at conception (Davey Smith & Ebrahim, 2005). In other words, the allocation of genetic variants from parents to children at the time of conception is as good as random. Genetic variants are largely unrelated to the many socioeconomic and behavioural characteristics that tend to confound observational studies. Hence, this genetic covariate may be understood as reflecting exogenous influences, uncorrelated with unobserved family or environmental factors. Note that coefficient β can be interpreted as representing *intention-to-treat* (ITT) effects, as the individual's diagnosis and/or symptoms are not observed directly (Davey Smith & Ebrahim, 2005).

As control variables, X_{it}, we include a set of indicators, namely the respondents' age and gender, that might have affected parental divorce. We also incorporate the following parental variables: whether the parents had experienced financial hardship; and whether either or both had experienced prolonged unemployment, were addicted to

[11] Note, for the sake of keeping the notation simple, here and below we omit time subscripts, but in our context these equations hold only conditioning on time periods.

[12] The standard errors are clustered at the individual level, as some individuals may appear in the regression during multiple time periods, but the results are consistent for different cluster types.

tobacco or alcohol, or had experienced mental health problems when the respondent was under 16 years of age. Additionally, in order to control for the family's earlier economic situation and cultural capital, we included several proxy variables referring to when the child was 10 years old, i.e., the number of bedrooms in the family house, the number of books possessed and the number of family members. The right-hand side in equation (2) also incorporates the top ten principal components (PC) of the genetic relationship matrix as additional regressors to account for potential population stratification (Price et al., 2006; NatCen Social Research, 2022). This stratification might bias the relationship between genetic factors (PGS) and parental divorce if genetic differences between subpopulations in the sample are related to unobserved factors in the error term. Dummy variables for each wave of the panel capture time-fixed effects.

4. Results

Baseline Results

Table 2 presents the primary results of the paper. Specifically, Table 2 displays the ITT effects of the PGS for ADHD on parental divorce/separation. Column (1) reports the unconditional estimates, column (2) includes a set of predetermined covariates, column (3) adds the ten principal components of the genetic relationship matrix, and column (4) includes year-fixed effects (preferred specification). Interestingly, these estimates indicate a positive and significant effect of the genetic predisposition of children to have ADHD on parental divorce/separation. Specifically, a one standard deviation increase in the propensity of having a child with ADHD increases the chances of marital breakdown by 1.4%. Moreover, the estimates are stable in each of the four alternative specifications.[13]

[TABLE 2 HERE]

Sensitivity Analysis

In Table 3 we show the robustness of our findings by means of a series of robustness checks. Specifically, column (1) presents the benchmark estimates and column (2) incorporates a set of current respondent characteristics, including marital status,

[13] Figure A2, in the Appendix, displays the unconditional relationship between the PGS for ADHD and parental divorce/separation.

education background, income and labour market status (to control for the respondent's socio-economic environment). Column (3) includes the PGS for the autism spectrum disorder (ASD) to control for potential genetic overlap between ADHD and ASD, which is one of the most common comorbidities associated to ADHD. Moreover, children and adolescents with both ADHD and ASD have more severe symptoms, which might affect our outcome of interest (Gnanavel et al., 2019). This helps isolate the specific genetic contributions of ADHD. Column (4) adds-in a set of parenting-style variables, to refine our understanding of environmental influences, particularly how different parenting styles might mitigate or exacerbate the genetic risks associated with ADHD. Finally, in Column (5) we take into account the panel nature of the data and allow for a random effects (RE) model. The coefficients obtained are remarkably consistent across different specifications, which confirms the robustness of our results.

[TABLE 3 HERE]

Potential Mechanisms

Figure 1 presents the results of the heterogeneity analysis conducted to investigate the interplay of the severity of the genetic predisposition to ADHD with the respondent's i) gender (Panel A) and ii) age when the parents divorced (Panel B).[14] The analysis by gender is grounded in the assumption that child gender might be a significant moderator of parenting stress, since there is evidence that parents of girls with ADHD experience lower levels of parental stress than those of boys with this condition (Theule et al., 2013). On the other hand, as discussed in Section 2, childhood age is an important predictor of marital stability.[15] For parents of children with no significant disability, the probability of divorce falls as the children grow up and become more independent, but the parents of children with a disability or health condition are exposed to a greater chance of divorce, due to the more severe parenting demands imposed on them, together with more disruptive or challenging behaviour by the children (Hartley et al., 2010).

Our results show that, as expected, the probability of parental divorce/separation increases with the severity of the genetic risk for ADHD (shown in both Panels). Thus,

[14] The severity of the genetic predisposition (shown in Figure 1) is measured by calculating quintiles of the standardised PGS distribution.

[15] We consider that parents broke up their marital relationship at three different children age groups: under 5 years, 10 years and 16 years.

parents of children in the top quintile of the PGS distribution experience a greater probability of marital dissolution. Moreover, this probability also rises as children enter adolescence (Panel B), suggesting that their disruptive behaviour tends to accumulate over time, adversely affecting marital functioning. However, no significant differences were found on the basis of gender (Panel A).[16]

[FIGURE 1 HERE]

Regarding other potential mechanisms for our findings, we examined whether a genetic predisposition for ADHD had a significant impact on respondents' health and health-related limitations when they were aged under 16 (see Table A2 in the Appendix). Our estimations show that individuals with a higher PGS for ADHD seem to be more likely to have restricted physical activities and to be hospitalised for lengthy periods due to health-related issues. This may partially explain why the parents of children with ADHD tend to be less occupationally active (Kvist et al., 2013) and more subject to stress (Kvist et al., 2013; Leitch et al., 2019; Schermerhorn et al., 2012; Theule et al., 2013). Moreover, in general they report lower levels of satisfaction and have a poorer health-related quality of life (Peasgood et al., 2021). All of these factors ultimately affect marital stability.

5. Conclusion and Discussion

This paper advances on previous studies by investigating the causal impact of the genetic risk of ADHD on marital conflict resulting in divorce. Our results show that, on average, a one-point increase in the standard deviation of the propensity to have a child with ADHD raises the probability that the parents' marital relationship will end by 1.4 percentage points. This is a sizable impact, roughly 28% with respect to the mean parental divorce/separation rate. The magnitude of the estimated effect increases with the severity of the condition and as the children enter adolescence, but does not substantially differ on the basis of gender. We also explore alternative channels for our findings, and conclude that children with a higher genetic predisposition for ADHD are also at greater risk of

[16] The set of estimates underlying Figure 1 are given in Table A.3 in the Appendix.

severe health-related limitations, which may aggravate parental stress and make marital dissolution more likely.

These findings are policy-relevant in several dimensions. From the perspective of the child, the traumatic event of divorce may exacerbate the severity of existing ADHD symptoms (Heckel et al., 2009), heightening pressures on the health system and on families confronting the disorder (Peñuelas-Calvo et al., 2021). Moreover, if these marital breakdowns increase the number of single-parent families, this would give policy makers additional cause for concern, as recent research has highlighted the existence of higher levels of child ADHD medication and diagnosis, especially among boys, within these non-traditional families impacting negatively their treatment (Bedard & Witman, 2020). Further, our findings suggest that ADHD in girls may also be associated with relevant disruptive behaviour within the family, which could differ from that of boys with the same condition and which should be analysed further in future studies.

It is reassuring to note that the cohorts analysed in this study and the findings obtained have not been affected by the policy changes introduced by the different divorce reform acts implemented since 1970 in England and Wales. Similarly, the data reveal that divorce rates remained roughly constant and low until the late 1960s, rising then until the mid-1990s to fall thereafter, although without returning to the low levels of the 1950s (ONS, 2024).

The results presented in this paper are in line with those of previous research, and support calls for a paradigm shift in managing this disorder, with the adoption of a broader and more comprehensive perspective that places the family at the centre of interventions. This need is accentuated by the fact that the behaviour of children/adolescents with ADHD tends to affect multiple dimensions of the lives of parents and siblings. Interventions focused on families, aimed at improving family relationships and caregivers' wellbeing, should be considered an essential aspect in the clinical management of ADHD (Peñuelas-Calvo et al., 2021). However, this broader approach would require the collaboration of mental health and social care services, with significant organisational and budgetary implications.

The results shown in this paper are subject to some limitations. Firstly, we do not have data on ADHD diagnosis, which could have been useful to investigate the effect of potential ADHD treatment as a mediating factor. Nevertheless, this information might not be especially informative, in any case, since our analysis concerns a cohort of individuals for whom the diagnosis and treatment of the ADHD condition was little known, differing

considerably from current procedures. Secondly, we cannot control for the ADHD genetic risk of parents, which could interact with that of their offspring and result in a stronger effect on parental divorce. If this were the case, our findings could be interpreted as representing a lower bound effect. Notwithstanding these limitations, we believe this paper sheds significant light on the important effect of children's ADHD on their parents' marital stability, highlighting the need, in treating ADHD, to design mental health policies that consider its impact on the entire family.

References

Adhvaryu, A., Daysal, N. M., Gunnsteinsson, S., Molina, T., & Steingrimsdottir, H. (2022, marzo). *Impacts of Child Health on Families: Evidence from Childhood Cancers*. 5th IZA Workshop on Gender and Family Economics: Families as an Insurance Mechanism. https://research.cbs.dk/en/publications/impacts-of-child-health-on-families-evidence-from-childhood-cance

Anchesi SD., Corallo F., Di Cara M., Quartarone A., Catalioto R., Cucinotta F., & Cardile D. (2023). Autism and ADHD: A literature review regarding their impacts on parental divorce. *Children (Basel)*, *10*(3). https://doi.org/10.3390/children10030438

Balbo, N., & Bolano, D. (2024). Child disability as a family issue: A study on mothers' and fathers' health in Italy. *European Journal of Public Health*, *34*(1), 79-84. https://doi.org/10.1093/eurpub/ckad168

Barkley, R. A., Fischer, M., Edelbrock, C., & Smallish, L. (1991). The adolescent outcome of hyperactive children diagnosed by research criteria—iii. Mother–child interactions, family conflicts and maternal psychopathology. *Journal of Child Psychology and Psychiatry*, *32*(2), 233-255. https://doi.org/10.1111/j.1469-7610.1991.tb00304.x

Bedard, K., & Witman, A. (2020). Family structure and the gender gap in ADHD. *Review of Economics of the Household*, *18*(4), 1101-1129. https://doi.org/10.1007/s11150-019-09476-9

Bitsko RH., Claussen AH., Lichstein J., Black LI., Jones SE, Danielson ML., et al. (2022). Mental health surveillance among children—United States, 2013–2019. *MMWR Suppl.*, *71*(2), 1-42. https://doi.org/10.15585/mmwr.su.7102a1

Brown, R. T., & Pacini, J. N. (1989). Perceived family functioning, marital status, and depression in parents of boys with attention deficit disorder. *Journal of Learning Disabilities*, *22*(9), 581-587. https://doi.org/10.1177/002221948902200911

Burt SA., Krueger RF., McGue M., & Iacono W. (2003). Parent-child conflict and the comorbidity among childhood externalizing disorders. *Archives of General Psychiatry*, *60*(5), 505-513. https://doi.org/10.1001/archpsyc.60.5.505

Chen, C., Bailey, C., Baikie, G., Dalziel, K., & Hua, X. (2023). Parents of children with disability: Mental health outcomes and utilization of mental health services. *Disability and Health Journal*, *16*(4), 101506. https://doi.org/10.1016/j.dhjo.2023.101506

Currie, J., & Stabile, M. (2006). Child mental health and human capital accumulation: The case of ADHD. *Journal of Health Economics*, *25*(6), 1094-1118. https://doi.org/10.1016/j.jhealeco.2006.03.001

Davey Smith, G., & Ebrahim, S. (2005). What can mendelian randomisation tell us about modifiable behavioural and environmental exposures? *BMJ (Clinical Research Ed.)*, *330*(7499), 1076-1079. https://doi.org/10.1136/bmj.330.7499.1076

Demontis, D., Walters, R. K., Martin, J., Mattheisen, M., Als, T. D., Agerbo, E., Baldursson, G., Belliveau, R., Bybjerg-Grauholm, J., Bækvad-Hansen, M., Cerrato, F.,

Chambert, K., Churchhouse, C., Dumont, A., Eriksson, N., Gandal, M., Goldstein, J. I., Grasby, K. L., Grove, J., … Neale, B. M. (2019). Discovery of the first genome-wide significant risk loci for attention deficit/hyperactivity disorder. *Nature Genetics*, *51*(1), Article 1. https://doi.org/10.1038/s41588-018-0269-7

Eriksen, T. L. M., Gaulke, A., Skipper, N., & Svensson, J. (2021). The impact of childhood health shocks on parental labor supply. *Journal of Health Economics*, *78*, 102486. https://doi.org/10.1016/j.jhealeco.2021.102486

Fletcher, J. M. (2014). The effects of childhood ADHD on adult labor market outcomes. *Health Economics*, *23*(2), 159-181. https://doi.org/10.1002/hec.2907

Fulton BD., Scheffler RM., Hinshaw SP., Levine P., Stone S., Brown TT., & Modrek S. (2009). National variation of ADHD diagnostic prevalence and medication use: Health care providers and education policies. *Psychiatric Services*, *60*(8), 1075-1083. https://doi.org/10.1176/ps.2009.60.8.1075

Furzer, J., Dhuey, E., & Laporte, A. (2022). ADHD misdiagnosis: Causes and mitigators. *Health Economics*, *31*(9), 1926-1953. https://doi.org/10.1002/hec.4555

Gnanavel, S., Sharma, P., Kaushal, P., & Hussain, S. (2019). Attention deficit hyperactivity disorder and comorbidity: A review of literature. *World Journal of Clinical Cases*, *7*(17), 2420-2426. https://doi.org/10.12998/wjcc.v7.i17.2420

Hartley, S. L., Barker, E. T., Seltzer, M. M., Floyd, F., Greenberg, J., Orsmond, G., & Bolt, D. (2010). The relative risk and timing of divorce in families of children with an autism spectrum disorder. *Journal of Family Psychology : JFP : Journal of the Division of Family Psychology of the American Psychological Association (Division 43)*, *24*(4), 449-457. https://doi.org/10.1037/a0019847

Heckel, L., Clarke, A., Barry, R., McCarthy, R., & Selikowitz, M. (2009). The relationship between divorce and the psychological well-being of children with ADHD: Differences in age, gender, and subtype. *Emotional and Behavioural Difficulties*, *14*(1), 49-68. https://doi.org/10.1080/13632750802655695

Kvist, A. P., Nielsen, H. S., & Simonsen, M. (2013). The importance of children's ADHD for parents' relationship stability and labor supply. *Social Science & Medicine*, *88*, 30-38. https://doi.org/10.1016/j.socscimed.2013.04.001

Leitch, S., Sciberras, E., Post, B., Gerner, B., Rinehart, N., Nicholson, J. M., & Evans, S. (2019). Experience of stress in parents of children with ADHD: A qualitative study. *International Journal of Qualitative Studies on Health and Well-being*, *14*(1), 1690091. https://doi.org/10.1080/17482631.2019.1690091

Lynch, F. L., Bulkley, J. E., Varga, A., Crawford, P., Croen, L. A., Daida, Y. G., Fombonne, E., Hatch, B., Massolo, M., & Dickerson, J. F. (2023). The impact of autism spectrum disorder on parent employment: Results from the r-Kids study. *Autism Research*, *16*(3), 642-652. https://doi.org/10.1002/aur.2882

NatCen Social Research. (2022). *ELSA Polygenic Scores, 2022. 2nd Edition.* UK Data Archive Study Number 8773. https://beta.ukdataservice.ac.uk/datacatalogue/studies/study?id=8773

Office for National Statistics (ONS). *Divorces in England and Wales: 2022*. Released 22 February 2024. Consulted on 25 May 2024. Available at https://www.ons.gov.uk/peoplepopulationandcommunity/birthsdeathsandmarriages/divorce/bulletins/divorcesinenglandandwales/2022

Peasgood, T., Bhardwaj, A., Brazier, J. E., Biggs, K., Coghill, D., Daley, D., Cooper, C. L., De Silva, C., Harpin, V., Hodgkins, P., Nadkarni, A., Setyawan, J., & Sonuga-Barke, E. J. S. (2021). What is the health and well-being burden for parents living with a child with ADHD in the United Kingdom? *Journal of Attention Disorders*, *25*(14), 1962-1976. https://doi.org/10.1177/1087054720925899

Peñuelas-Calvo, I., Palomar-Ciria, N., Porras-Segovia, A., Miguélez-Fernández, C., Baltasar-Tello, I., Colmenero, S. P.-, Delgado-Gómez, D., Carballo, J. J., & Baca-García, E. (2021). Impact of ADHD symptoms on family functioning, family burden and parents' quality of life in a hospital area in Spain. *European Journal of Psychiatry*, *35*(3), 166-172. https://doi.org/10.1016/j.ejpsy.2020.10.003

Price AL., Patterson NJ., Plenge RM., Weinblatt ME., Shadick NA., & Reich D. (2006). Principal components analysis corrects for stratification in genome-wide association studies. *Nature Genetics, 38*(8), 904-909. https://doi.org/10.1038/ng1847

Rajah, N., Mattock, R., & Martin, A. (2023). How do childhood ADHD symptoms affect labour market outcomes? *Economics & Human Biology*, *48*, 101189. https://doi.org/10.1016/j.ehb.2022.101189

Rietveld CA, & Patel PC. (2019). ADHD and later-life labor market outcomes in the US. *European Journal of Health Economics*, 949-967.

Ronald, A., Bode, N. de, & Polderman, T. J. C. (2021). Systematic review: how the attention-deficit/hyperactivity disorder polygenic risk score adds to our understanding of ADHD and associated traits. *Journal of the American Academy of Child & Adolescent Psychiatry*, *60*(10), 1234-1277. https://doi.org/10.1016/j.jaac.2021.01.019

Schachar, R. J., & Wachsmuth, R. (1991). Family dysfunction and psychosocial adversity: Comparison of attention deficit disorder, conduct disorder, normal and clinical controls. *Canadian Journal of Behavioural Science / Revue canadienne des sciences du comportement*, *23*(3), 332-348. https://doi.org/10.1037/h0079018

Schermerhorn AC., D'Onofrio BM., Slutske WS., Emery RE., Turkheimer E., Harden KP., Heath AC., & Martin NG. (2012). Offspring ADHD as a risk factor for parental marital problems: controls for genetic and environmental confounds. *Twin Research and Human Genetics*(15), 700-713. https://doi.org/10.1017/thg.2012.55

Stergiakouli E., Martin J., Hamshere ML., Heron J., St Pourcain B., Timpson NJ., Thapar A., & Davey Smith G. (2016). Association between polygenic risk scores for attention-deficit hyperactivity disorder and educational and cognitive outcomes in the general population. *International Journal of Epidemiology*, *46*(2). https://doi.org/10.1093/ije/dyw216

Theule, J., Wiener, J., Tannock, R., & Jenkins, J. M. (2013). Parenting stress in families of children with ADHD: a meta-analysis. *Journal of Emotional and Behavioral Disorders, 21*(1), 3-17. https://doi.org/10.1177/1063426610387433

Vos, M., & Hartman, C. A. (2022). The decreasing prevalence of ADHD across the adult lifespan confirmed. *Journal of Global Health, 12*, 03024. https://doi.org/10.7189/jogh.12.03024

Ward K., Medina J., Mo M., & Cox K. (2009). *ELSA Wave Three: Life History Interview. A User Guide to the Data. Version 1*. IFS.

Wymbs, B. T., Pelham Jr., W. E., Molina, B. S. G., Gnagy, E. M., Wilson, T. K., & Greenhouse, J. B. (2008). Rate and predictors of divorce among parents of youths with ADHD. *Journal of Consulting and Clinical Psychology*, *76*(5), 735-744. https://doi.org/10.1037/a0012719

Tables and Figures

6th June 2024

Table 1: Summary Statistics

	Mean	S.D.	Min	Max
Outcome Variable:				
Parents Divorced Age < 16	0.05	0.22	0	1
Main Regressor:				
PGS for ADHD	-876.55	56.07	-1061	-679
Standardisd PGS for ADHD	-0.00	1.00	-3	4
Demographics:				
Years of Age	68.17	9.36	31	99
Female [0,1]	0.55	0.50	0	1
Married [0,1]	0.69	0.46	0	1
Family Size	1.97	0.77	0	8
Socio-Economics:				
Higher Education [0,1]	0.17	0.38	0	1
Household Income	5.65	0.64	-4	9
Employed [0,1]	0.20	0.40	0	1
Self Employed [0,1]	0.05	0.22	0	1
Unemployed [0,1]	0.01	0.07	0	1
Parental Conditions Age < 16:				
Financial Hardship [0,1]	0.18	0.38	0	1
Parents Addicted [0,1]	0.06	0.23	0	1
Parents Unemployed [0,1]	0.07	0.26	0	1
Living Conditions Age 10:				
No. of Books at Home	2.54	1.21	1	5
No. of Bedroom at Home	2.94	0.88	1	15
Family Size	4.90	1.74	2	22
Observations	26235			

Source: English Longitudinal Study of Ageing (ELSA), Wave 1-9.

Note: The Table reports the mean, standard deviation, minimum and maximum values of the main variables used in the analysis.

Table 2: Estimates of the Effect of the ADHD Polygenic Score on Parental Divorce

	(1) Parents Divorced Age < 16	(2) Parents Divorced Age < 16	(3) Parents Divorced Age < 16	(4) Parents Divorced Age < 16
PGS for ADHD	0.015***	0.014***	0.014***	0.014***
	(0.004)	(0.004)	(0.004)	(0.004)
Demographics:				
Years of Age		-0.001***	-0.001***	-0.001***
		(0.000)	(0.000)	(0.000)
Female [0,1]		-0.013*	-0.014*	-0.014*
		(0.008)	(0.008)	(0.008)
Parental Conditions Age < 16:				
Financial Hardship [0,1]		0.046***	0.046***	0.045***
		(0.012)	(0.012)	(0.012)
Parents Addicted [0,1]		0.062***	0.063***	0.062***
		(0.024)	(0.024)	(0.024)
Parents Unemployed [0,1]		-0.012	-0.011	-0.010
		(0.015)	(0.015)	(0.015)
Living Conditions Age 10:				
No. of Books at Home		-0.004	-0.004	-0.004
		(0.003)	(0.003)	(0.003)
No. of Bedroom at Home		-0.004	-0.005	-0.005
		(0.005)	(0.004)	(0.004)
Family Size		-0.005*	-0.004*	-0.004*
		(0.003)	(0.003)	(0.003)
Principal Components			✓	✓
Time FE				✓
Observations	26235	26235	26235	26235

Source: English Longitudinal Study of Ageing (ELSA), Wave 1-9.

Note: The Table reports estimates of the effect of the ADHD polygenic score on parental divorce. Standard errors are clustered at the respondent level. Observations are at the respondent-year level. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$.

Table 3: Estimates of the Effect of the ADHD Polygenic Score on Parental Divorce - Sensitivity Analysis

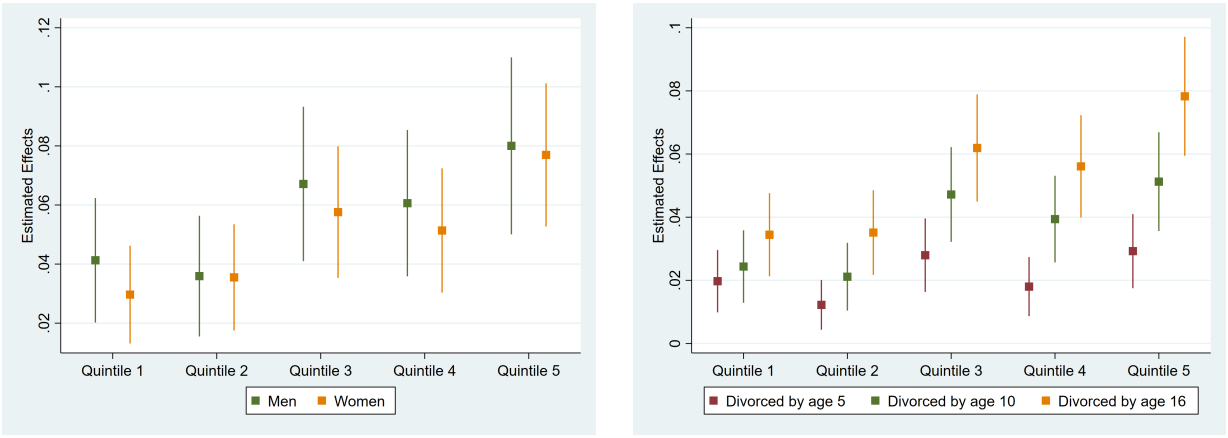
	(1) Parents Divorced Age < 16	(2) Parents Divorced Age < 16	(3) Parents Divorced Age < 16	(4) Parents Divorced Age < 16
PGS for ADHD	0.014***	0.014***	0.014***	0.013***
	(0.004)	(0.004)	(0.004)	(0.004)
Covariates	✓	✓	✓	✓
Principal Components	✓	✓	✓	✓
Time FE	✓	✓	✓	✓
Additional Covariates	✓			
Poligenic Score for ASD		✓		
Parenting Styles			✓	
RE Model				✓
Observations	25859	26235	25761	26235

Source: English Longitudinal Study of Ageing (ELSA), Wave 1-9.

Note: The Table reports estimates of the effect of the ADHD polygenic score on parental divorce. Standard errors are clustered at the respondent levels. Observations are at the respondent-year level. *$p < 0.1$, **$p < 0.05$, ***$p < 0.01$.

Figure 1: Heterogeneity Analysis

Panel A: by Gender **Panel B: by Age of Divorce**

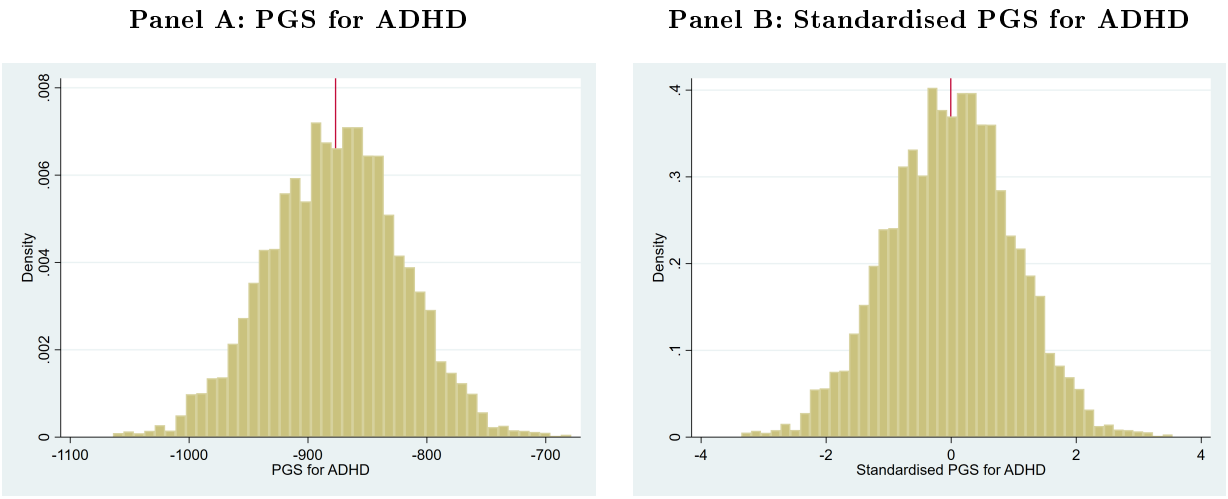


Note: The Figures provide a graphical representation of the heterogeneity analysis. In Panel A, we report the estimated coefficients, with associated standard error, by gender and by quintiles of the polygenic score for ADHD. Similarly, in Panel B, we report the estimated coefficients, and associated standard errors, by quintiles of the polygenic score for ADHD and by respondent's age when parents divorced.

A Appendix

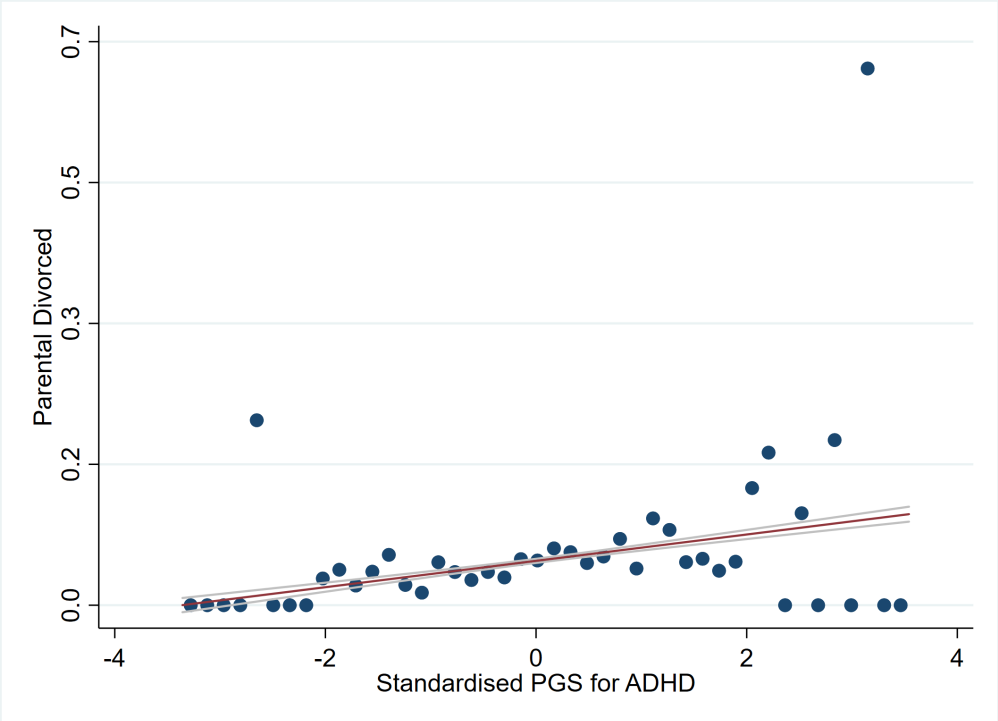
A.1 Additional Tables and Figures

Figure A.1: Histogram Polygenic Score



Note: The Figures show the distribution of the poligenic score (PGS) for ADHD.

Figure A.2: Effect of PGS for ADHD on Parental Divorce



Note: The figure plots the relationship between the standardised PGS for ADHD and parental divorce when the respondent was 16 or below.

Table A.1: Summary Statistics - by Gender

	(1) **Men**	(2) **Women**	(3) ***p*-value**
Outcome Variable:			
Parents Divorced Age < 16	0.06	0.05	0.000
	(0.236)	(0.213)	
Main Regressor:			
PGS for ADHD	-879.20	-874.41	0.000
	(55.303)	(56.595)	
Standardisd PGS for ADHD	-0.05	0.04	0.000
	(0.990)	(1.013)	
Demographics:			
Years of Age	68.15	68.17	0.873
	(9.254)	(9.451)	
Married [0,1]	0.79	0.61	0.000
	(0.410)	(0.487)	
Family Size	2.08	1.88	0.000
	(0.762)	(0.762)	
Socio-Economics:			
Higher Education [0,1]	0.23	0.12	0.000
	(0.419)	(0.329)	
Household Income	5.71	5.60	0.000
	(0.653)	(0.630)	
Employed [0,1]	0.22	0.19	0.000
	(0.413)	(0.394)	
Self Employed [0,1]	0.08	0.03	0.000
	(0.272)	(0.175)	
Unemployed [0,1]	0.01	0.00	0.000
	(0.089)	(0.056)	
Parental Conditions Age < 16:			
Financial Hardship [0,1]	0.16	0.20	0.000
	(0.367)	(0.398)	
Parents Addicted [0,1]	0.05	0.06	0.173
	(0.226)	(0.233)	
Parents Unemployed [0,1]	0.08	0.06	0.000
	(0.271)	(0.244)	
Living Conditions Age 10:			
No. of Books at Home	2.42	2.64	0.000
	(1.194)	(1.220)	
No. of Bedroom at Home	2.92	2.96	0.001
	(0.874)	(0.887)	
Family Size	4.84	4.96	0.000
	(1.732)	(1.741)	
Observations	11740	14495	26235

Source: English Longitudinal Study of Ageing (ELSA), Wave 1-9.

Note: The Table compares the mean values of the main variables used in the paper by gender. There is a total of 30748 respondent-year level observations, covering 4261 respondents.

Table A.2: Estimates of the Effect of the ADHD Polygenic Score on Respondent Health during Childhood/Adolescence

	(1) **Restricted Physical Activities**	(2) **Confined in Bed Due to Health**	(3) **Hospitalised > 1 Month**
PGS for ADHD	0.018***	0.012*	0.011**
	(0.006)	(0.006)	(0.005)
Observations	26223	26193	26171

Source: English Longitudinal Study of Ageing (ELSA), Wave 1-9.

Note: Dependent variables: dummy variables describing the health of the respondent during childhood/adolescence (before the age of 16). Key regressor: (standardized) polygenic score (PGS) for ADHD. Standard errors are clustered at the respondent levels. Observations are at the respondent-year level. All models control for the covariates explained in Section 4. ** $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.*

Table A.3: Estimates of the ADHD Polygenic Score on Parental Divorce - Heterogeneity Analysis

	(1) **Full-Sample**	(2) **Men**	(3) **Women**
Panel A: Parents Divorced by Age of 5			
Quintile 1	0.019***	0.023***	0.016**
	(0.005)	(0.009)	(0.007)
Quintile 2	0.012***	0.008	0.015**
	(0.004)	(0.006)	(0.007)
Quintile 3	0.029***	0.030***	0.029***
	(0.007)	(0.010)	(0.009)
Quintile 4	0.017***	0.022**	0.012**
	(0.005)	(0.009)	(0.006)
Quintile 5	0.032***	0.041***	0.026***
	(0.007)	(0.012)	(0.008)
Observations	26235	11740	14495
Panel B: Parents Divorced by Age of 10			
Quintile 1	0.023***	0.028***	0.019**
	(0.006)	(0.010)	(0.008)
Quintile 2	0.021***	0.018**	0.024***
	(0.006)	(0.008)	(0.009)
Quintile 3	0.046***	0.050***	0.043***
	(0.008)	(0.013)	(0.011)
Quintile 4	0.036***	0.048***	0.025***
	(0.007)	(0.013)	(0.008)
Quintile 5	0.055***	0.053***	0.056***
	(0.009)	(0.014)	(0.012)
Observations	26235	11740	14495
Panel C: Parents Divorced by Age of 16			
Quintile 1	0.032***	0.040***	0.025***
	(0.007)	(0.011)	(0.009)
Quintile 2	0.034***	0.033***	0.036***
	(0.007)	(0.011)	(0.010)
Quintile 3	0.056***	0.064***	0.051***
	(0.009)	(0.014)	(0.012)
Quintile 4	0.050***	0.059***	0.043***
	(0.009)	(0.014)	(0.011)
Quintile 5	0.078***	0.084***	0.073***
	(0.010)	(0.017)	(0.013)
Observations	26235	11740	14495

Source: English Longitudinal Study of Ageing (ELSA), Wave 1-9.

Note: Key regressors: dummies for each quantile of the PGS distribution. Standard errors are clustered at the respondent level. Observations are at the respondent-year level. In Column (1), we report estimates for the full sample, while in Columns (2) and (3), we distinguish between men and women. In Panel A, the dependent variable is a dummy variable for whether the respondent's parents divorced before the respondent turned 5. In Panel B, the dependent variable is a dummy variable for whether the respondent's parents divorced before the respondent turned 10. In Panel C, the dependent variable is a dummy variable for whether the respondent's parents divorced before the respondent turned 16.* $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$