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Comorbidity aggregation models in children and adolescents with ADHD and direct and moderator effects of familial clinical history and psychosocial factors

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ABSTRACT

ADHD condition occurs with an extensive variety of comorbid mental disorders This study aims to individuate models of comorbidity aggregation in 1086 probands with ADHD and the direct and moderator effect of First Degree Relatives (FDRs) Mental Disorders and socio-environmental factors. Principal Component Analysis individuated four models of highly related comorbidities in affected probands. Inter-psychiatric and inter-neurodevelopmental comorbidities associations, with higher loading of Anxiety-Depressive-Conduct and "Intellectual-Language-Motor" subgroups of disorders were distinctly collocated within first and second components respectively. Regression and moderator analyses highlighted the effect of familiarity ADHD among siblings and socio-environmental factors in both components; in addition, FDRs psychiatric disorders worked as direct factors in the first component. Tic-Tourette and Autism vs. Bipolar Disorder and Intellectual Disabilities; Opposition-Conduct vs. Learning Disabilities subgroups in the third and fourth components, respectively, defined contraposed comorbidity profiles. Mothers with ADHD moderated by lower rates of ADHD siblings; high negative parenting and lower FDRs neurodevelopmental disorders represented the direct effect for the third and fourth components, respectively. Proband's age as well as disorders loading within more components contribute to better define these comorbidities subgroups. The findings suggest to consider a nosology of ADHD that takes in account of comorbidity profiles and their co-related familial and environmental risk factors

1. Introduction

The literature on children and adolescents with Attention Deficit Hyperactivity Disorder (ADHD) reports that this condition rarely presents itself on its own; it occurs with an extensive variety of comorbid psychiatric and neurodevelopmental disorders (Inci et al.,

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2019; Jensen & Steinhausen, 2015; Spencer, 2006). The range fluctuates from 15 % to 80 % and from 13 % to 50 % for psychiatric and neurodevelopmental disorders, respectively, depending 1) on the syndromic comorbidity and developmental stages (Keresztény et al., 2012); 2) by child's sex (Tung et al., 2016); 3) by clinic-referred versus community-referred samples (Abikoff & Klein, 1992); 4) by structure of the study and clinical practice sensibility to the ADHD. Some studies support an early co-occurrence onset of these disorders since they did not substantially find a qualitative difference between younger vs older children; however, the latter reported higher frequency and number of comorbidities (Keresztény et al., 2012). In parallel, longitudinal studies emphasised more internalising disorders among girls and externalising disorders among males (Biederman et al., 2008, 2010), although others documented increased ODD, CD and aggressive behaviours also in girls (Tung et al., 2016). Despite different approaches and results, there is unanimous agreement that the comorbidity condition plays a crucial role in the heterogeneity, the developmental trajectories, the treatment and the lifetime persistence and outcome of ADHD itself (Biederman et al., 2008; Jensen & Steinhausen, 2015; Spencer, 2006).

Genetic studies support a biological basis of ADHD and its comorbidity, reporting that 1) ADHD shares common genetic risk variants with other psychiatric or neurodevelopmental disorders, indicating that "the current clinical boundaries do not reflect distinct underlying pathogenetic processes at least on the genetic level" (Anttila et al., 2018; Lee et al., 2019); 2) A considerable portion of the genetic variants associated with ADHD polygenic risk scores (PRS) reflects a non-specific genetic liability toward broad childhood psychopathology (Brikell, Larsson, & Lu, 2020); 3) Part of the heritability of mental traits as well genetic overlap between disorders is due to shared genetic variation with socio-economic status (SES) (Marees et al., 2021).

Based on the mutual interaction of hereditary and socio-environmental factors in promoting a child's ADHD symptoms (Faraone & Doyle, 2001), clinical research has increased the use of pedigree data (parents, twins, siblings and half-siblings) for investigating the link between familial characteristics and proband's comorbidity.

Previous studies have documented the following: 1) Certain comorbid disorders with ADHD co-aggregate within families (Del'Homme et al., 2007; Faraone et al., 1997; Faraone & Doyle, 2001); 2) First Degree Relatives (FDRs) of children with ADHD are at a higher risk for major psychiatric disorders, with this risk increasing significantly if the FDRs also have ADHD (Hidalgo-López et al., 2019; Jokiranta-Olkoniemi et al., 2019; Wei et al., 2019; Yang et al., 2011; Young, 2011); 3) Low socio-economic status and environmental adversities in the household contribute to the onset of ADHD and comorbid disorders in offspring (Björkenstam et al., 2018; Choi et al., 2017; Keilow et al., 2020; McRae et al., 2020; Weissenberger et al., 2017). Collectively, these studies indicate that family history and socio-environmental characteristics are crucial areas of research to better understand the factors underlying comorbidity in children with ADHD.

Given that the presence of comorbidities among children with ADHD is the norm rather than the exception, the current study aims to delineate highly related comorbidity subgroups within a large cohort of Italian probands with ADHD, encompassing a broad spectrum of psychiatric and neurodevelopmental disorders. Additionally, the study seeks to investigate associated familial mental disorders and socio-environmental risk factors.

More specifically, this study aims to individuate: 1) which disorders are more associated with ADHD than others. Since it is presumable that our sample will show a great number of different combinations among co-occurrent comorbidity disorders, we are interested in individuate models of co-aggregation of specific comorbidity disorders which are mostly each other associated; 2) which familial risk factors, including parenting strategies, mother's education and family income, differentiate comorbidity subgroups.

2. Methods

2.1. Participants

Data reported in this study are extracted from an online cross-sectional survey filled out by parents and targeted to 5–18-year-old children with ADHD. The survey included a wide collection of data used to develop distinct research directions, some of which had been presented in previous studies (Bruni et al., 2021; Melegari et al., 2021, 2023). Parents' informed consent represented an obligatory field for proceeding to the compilation of the questionnaire.

Specifically for this study, parents were asked to respond to the following items: 1) diagnosed comorbidities of own affected child with ADHD, subject of the whole survey by us identified as "proband"; 2) probands' FDRs psychiatric or neurodevelopmental disorders specifying the relative (mother, father or sibling) whom the data referred. Probands' and FDRs' diagnoses were certified by child and adolescent and adult Mental Health Services, respectively; 3) SES and environmental characteristics of familial context.

A total of 1086 probands with ADHD (M = 925 (85.2 %); F = 161 (14.8 %) mean age 11.47, S.D.3.19) participated in the study. Adopted children were excluded.

A consistent percentage of the probands with ADHD were registered to the National ADHD Patient Register, and medication prescriptions were reported in 438 (40.4 %) patients; 70.3 % of them were treated with Methylphenidate, and 26.7 % with atomoxetine, antipsychotics, mood stabilisers, and other drugs. 2.2 % of caregivers did not specify the treatment.

The total sample was characterised by a significant representation of the Italian territory with the participation of all regions, 20 metropolitan cities and 78.3 % (72/92) of Italian provinces.

The study was approved by the Ethics Committee of the Department of Developmental and Social Psychology at Sapienza University and was conducted in accordance with the Declaration of Helsinki (October 2013).

2.2. Procedures and materials

2.2.1. Probands' comorbidities

Parents were asked to select from or to add to the list of 13 disorders certified proband's comorbidities coded with 1 = present and 0 = absent.

The list reported: a) seven psychiatric comorbid disorders: Oppositional Defiance Disorder (ODD), Generalized Anxiety (GAD), Separation Anxiety (SAD), Social Phobias (SP), Depressive Disorders (DEP), Bipolar Disorder (BD), Conduct Disorder (CD); b) seven neurodevelopmental disorders: Intellectual Disability (ID), Specific Language Disorder (SLangD), Specific Learning Disorder (SLearnD), Autism Spectrum Disorder (ASD), Developmental Coordination Disorder (DCD), Tic (TIC) and Tourette (TS) Disorders. Because the rates of SAD (2.6 %), SP (3.6 %), and Tourette disorder (2.5 %) were low—and their associations with GAD (30 % and 27.5 % for SAD and SP, respectively) and Tic disorders (37 % for Tourette disorder) were significant—we combined them into broader categories: Anxiety Disorders and Tic-TS, improving statistical power. Diagnoses added by parents (epilepsy, OCD: <1 %) were not included in the study due to their low frequency.

2.2.2. FDRs psychiatric and neurodevelopmental disorders

The frequencies of psychiatric or neurodevelopmental disorders among First Degree Relatives (FDRs) were scored as present (1) and absent (0). These scores were included in the statistical analyses without distinction between FDRs, given their low occurrence rates. ADHD in fathers, mothers, and siblings were classified as either absent (0) or present (1) and incorporated into the analyses as separate variables.

2.2.3. Familial socio-environmental factors

We asked the parents:

- to choose one or more of eleven parenting strategies derived from the Alabama Parenting Questionnaire (APQ; Essau et al., 2006) to use with their children, scored on a binary scale (e.g., "Yes"/"No"). The strategies described in the questionnaire comprise two subscales: supportive/preventive strategies defined as positive (e.g., "I listen to his/her request,; "to find a compromise"), and authoritarian harsh patterns defined as negative strategies (e.g., "I scream, I lose control"; "I punish him/her excessively"). The internal consistency in the sample of the present study was adequate for both positive (Cronbach's $\alpha = 0.64$) and negative parenting strategies (Cronbach's $\alpha = 0.58$).to define the presence of one or both parents in the household, indicating with which caregiver(s) the offspring lived, scored as: both parents = 1; mother = 2; father = 3; other caregiver = 4.
- to indicate the number of offspring in the household, coded as: 1, 2, 3, 3 + .
- We asked parents to categorise their family income into one of three classes: "low," "medium," or "high." We chose to use a subjective evaluation rather than an objective one because it aligns better with the goals of our study. Previous research has shown that individuals' perceptions of their socio-economic status, or where they believe they rank in the socio-economic hierarchy, are more closely linked to both adult and child mental health outcomes, even after accounting for objective economic status (McLaughlin et al., 2012; Pickett, 2006; Drukker et al., 2003; Singh-Manoux et al., 2003; Adler et al., 2000).

Finally, data referred to educational level were coded as: 1 = ≤ secondary school, 2 = high school, 3 = graduate, based on the reporting parent (95 % mothers). We decided to utilise these data for three reasons: 1) the mother is universally recognised as the principal caregiver within the family context; 2) several studies highlight the significant impact of maternal educational attainment, compared to that of the father, on child health and development outcomes (Geoffroy et al., 2010; Murata et al., 2023; Ye et al., 2019); 3) we expected a considerable percentage of single-parent households, and children predominantly continue to live with their mother following parental separation (Agha et al., 2013).

2.3. Statistical analyses

The binary responses of parents were coded as binomial variables (1 = yes, 0 = no). The effects of probands' age and sex on the total number of comorbidities were analysed using Generalised Linear Models with a Poisson distribution. Further comparisons on the presence/absence of each comorbidity were analysed using Chi-square tests.

Chi-square tests were used to determine group differences in the following variables: FDRs with or without ADHD or with psychiatric or developmental comorbidities. ANOVA followed by post hoc LSD tests were used for ordinal variables. Means and standard deviations were calculated for each variable.

To group parenting strategies and probands' comorbidities, the following data reduction strategy was used: first, Parallel Analysis (PCA) with 1000 iterations was performed to determine the ideal number of components; then, a PCA with Varimax rotation was conducted.

The resulting PCA components were used as dependent variables in ANCOVAs to determine the extent to which clinical familial history, socio-economic and environmental conditions, and data reduction of parenting strategies (independent variables) impact them. Additionally, age, sex, and drug treatment were included as covariates. Therefore, in each ANCOVA, we considered as independent variables: i) if the mother has ADHD; ii) if the father has ADHD; iii) if other siblings have ADHD; iv) if there are first-degree relatives with psychiatric problems; v) if there are first-degree relatives with neurodevelopmental disorders; vi) the self-reported family income; vii) the level of education of the mother; viii) if the family is single-parent or two-parents; ix) the number of sib-

lings; x and xi) the score regarding the negative and positive parenting strategies; xii) if the proband is taking drugs for his/her condition; xiii and xiv) the proband's sex and age. All results report the η_p^2 as effect size and the E-Value as sensitivity analysis for selection bias (Smith & VanderWeele, 2019).

Finally, bootstrapped moderation analyses were conducted to evaluate if the effect of direct variables, identified through regression analyses, on each specific component is conditional on the values of a third variable. Moderation analyses are reported with the ΔR_{mo}^2 effect size (Liu & Yuan, 2021), and they are graphically reported by means of Tumble graphs, a graphical approach that minimises the risk of misleading interpretations by ensuring that the endpoints of plotted line segments are selected based on actual data rather than arbitrary standard deviations (Bodner, 2016). Moderation analysis was performed following the Memon and colleagues (2019) recommendations and considering the variables that represent the socioeconomic familial status, because they predominantly play a significant role as moderators in the development of ADHD in children, interacting with genetic predisposition (Björkenstam et al., 2018; Choi et al., 2017; Keilow et al., 2020; McRae et al., 2020; Weissenberger et al., 2017). Therefore, we computed the moderation for each statistically significant regressor by considering the effects of other significant regressors, as well as variables representing socio-economic familial status. These include: a) perceived family income, b) whether the household is single- or two-parent, and c) the number of children in the household, even if these factors are not statistically significant.

The decision to use perceived family income as a moderator is supported by the strong association between parental socio-economic status and ADHD disorders in offspring (Larsson, Asherson, & Chang, 2013). Research indicates that children from low socio-economic status (SES) families are at a 1.85–4.52 times higher risk of developing ADHD compared to those from high-SES families (Hegelund et al., 2019; Russell et al., 2016).

The moderating effect of household structure, whether single- or two-parent, is also backed by empirical evidence, which shows that children from single-parent households have an odds ratio of 1.76 for ADHD diagnoses (Rethemiotaki, 2020). Lastly, the number of children in the household was included as an additional socio-economic moderator because larger families are often linked to increased socio-economic disadvantage. Moreover, larger families can also be linked to rivalry, aggression, and avoidance among siblings, which are factors correlated with ADHD symptoms (Ayyash et al., 2023).

All statistical analyses were conducted using the R statistical software (R Core Team, 2022), employing the R package 'psych' (Revelle, 2022) for PCA and parallel analyses, the package 'stdmod' (Cheung et al., 2022) for moderation analysis and the package 'EValue' (Smith & VanderWeele, 2019) for sensitivity analysis.

3. Results

Probands' demographic, socio-economic, and environmental characteristics, as well as FDRs' psychiatric and neurodevelopmental characteristics and probands' comorbidities and their interactions, are reported in Table 1 (A-B-C). Of the probands, 90.8 % showed at least one comorbidity; specifically, 47.4 % had one comorbidity, while 43.4 % had two or more comorbidities. Males vs. females differed in the number of mental comorbid disorders (Table 2A). The interaction between sex and #Comorbidities showed that more male adolescents had 1, 2, or 3 + comorbidities than male children, while more female adolescents had 1 comorbidity than female children (Table 2B-C).

Frequencies of socio-environmental variables are reported in Table 1A. The 30 % of parents had only the proband, whereas 70 % had two, three or more offspring. 80.2 % of probands lived with both parents, while 19.5 % lived with a single parent (100 % mother) and 0.3 % with other caregivers (e.g. foster grandmother). Moreover, 19.5 % of mothers had low education, and 22.7 % of families had low income. $\chi 2$ tests were used to study the interaction between socio-environmental factors. Specifically, probands with one parent showed: 1) proportionally more low-income whereas more medium-income was in two-parents household ($\chi 2(1) = 43.062$, p Bonferroni corrected < 0.001); 2) higher frequencies of one or two offsprings compared to those with two parents household ($\chi 2(1) = 26.98$, p Bonferroni corrected < 0.001 and $\chi 2(1) = 15.297$, p Bonferroni corrected < 0.001, respectively) (Table 1A); 3) a significant positive relationship between mothers with $\leq$ secondary educational level and number of offspring compared to those with high school ($p < 0.001$), and graduate ($p = 0.029$), indicating that mothers with a low educational level had more offspring than mothers with higher educational level.

Frequencies of FDR mental disorders and their interactions are reported in Table 1B. 12.8 % of probands had at least one FDR with ADHD; among those with siblings (757/1086; 69.7 %), 11.1 % had at least one sibling with ADHD. Siblings with ADHD were related to probands' higher number of comorbidities (binomial model $\chi 2(3) = 12.417$, $p = 0.006$) since probands with 3 or more comorbidities had proportionally more siblings with ADHD than probands with one comorbidity ($p = 0.043$). FDRs with ADHD had higher frequencies of psychiatric and neuro-developmental disorders when compared to those without ADHD (Table 1B). Moreover, we found a negative significant relationship between father with ADHD and income ($\chi 2(2) = 10.196$, $p = 0.006$) and a positive significant relation between father ADHD and rates of household with one parent (binomial model $\chi 2(1) = 12.066$, $p = 0.001$), indicating that more the fathers are ADHD more showed low income and higher frequency of families with one parent household compared to fathers without ADHD.

Parenting strategies PCA showed the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy of 0.7, and the Bartlett Sphericity test was statistically significant ($\chi 2(55) = 3815.62$, $p < 0.001$). Parallel analysis suggested a 2-component solution, and the results were able to explain the 52 % variability. The two components, named "positive" and "negative" parenting strategies" were computed by adding the presence = 1 / absence = 0 responses weighted for the loadings.

Probands' comorbidities PCA showed the Kaiser-Meyer-Olkin (KMO) measure of sampling adequacy was 0.53, and the Bartlett Sphericity test was statistically significant ($\chi 2(55) = 2595.44$, $p < 0.001$), suggesting that these data can be used for PCA.

Table 1
Social, demographic and clinical characteristics of the probands and probands' families. A) Income = the self-evaluation of the family income. Mother's Education = the self-report of the education level of the mother (primary = primary and secondary school degree; high school = high school degree; graduate = college-level education). Within rounded brackets is reported the mean and standard deviation of offsprings, divided by education level. Number of Offsprings = the number of offsprings and its percentage B) Mother ADHD and Father ADHD = the number and percentage of parents with ADHD. Siblings ADHD = the number and percentage of probands' with at least one sibling with ADHD. # siblings with ADHD = the number and percentage of siblings with ADHD. FDRs psychiatric = the number and percentage of probands' with at least one first degree relative with psychiatric diseases. FDRs Neurodevelopmental = the number and percentage of probands' with at least FDRs with neurodevelopmental diseases. C) Probands' characteristics and comorbidities, divided between children and adolescents (age > 11 y.o.). * = percentage referred to the total number of FDRs without ADHD; ** = percentage referred to the total number of FDRs with ADHD; $ = statistically significant difference between adolescents and children in the frequency of comorbidities.

A) Family characteristics				
		Single-Parent	**Two-Parent**	**Overall Total**
Income	Low	84 (40 %)	158 (18 %)	244 (22.7 %)
	Medium	124 (58 %)	677 (79 %)	803 (74.6 %)
	High	4 (2 %)	26 (3 %)	30 (2.8 %)
	Missing data	-	8 (missing)	9(missing)
Mother's Education	Primary School (#Offsprings: 2.1 (SD 0.87))	36 (17 %)	173 (20 %)	212 (19.5 %),
	High School (#Offsprings: 1.8 (SD 0.73))	116 (55 %)	447 (51 %)	564 (51.9 %)
	Graduate (#Offsprings: 1.93 (SD 0.71))	60 (28 %)	249 (29 %)	310 (28.5 %)
Number of Offsprings	1 Child	95 (45 %)	232 (27 %)	329 (30 %)
	2 Children	85 (40 %)	481 (55 %)	569 (52 %)
	3 Children	26 (12 %)	121 (14 %)	147 (13.5 %)
	More than 3 Children	6 (3 %)	35 (4 %)	41 (3.8 %)
	Missing data	-	10 (missing)	-

B) Relatives' Psychiatric and Neurodevelopmental Characteristics		
		N (%)
Mother ADHD		28 (2.6 %)
Father ADHD		42 (3.8 %)
Siblings ADHD		84 (7.7 %)
# Siblings with ADHD	1 sibling	65 (6 %)
	2 siblings	13 (1.2 %)
	3 siblings	6 (0.5 %)
FDRs Psychiatric $		50 (4.6 %)
	Without ADHD	28 (2.9 %*)
	With ADHD	22 (15.7 %**)
FDRs Neurodevelopmental $		54 (5 %)
	Without ADHD	39 (4.1 %*)
	With ADHD	15 (10.7 %**)

C) Probands' characteristics and comorbidities		
Gender		
	Male	925 (age: 11.5, SD 3.21)
	Female	161 (age: 11.4, SD 3.13)
Conduct Disorder		88 (8.11 %)
	Adolescents	66 (10.26 %)
	Children	22 (4.98 %)
	FDRs without ADHD	70 (7.41 %*)
	FDRs with ADHD	18 (12.86 %**)
Oppositional Defiant Disorder		547 (50.41 %)
	Adolescents	326 (50.7 %)
	Children	221 (50 %)
	FDRs without ADHD	469 (49.63 %*)
	FDRs with ADHD	78 (55.71 %**)
Anxiety		163 (15.02 %)
	Adolescents	121 (18.82 %)
	Children	42 (9.5 %)
	FDRs without ADHD	129 (13.65 %*)
	FDRs with ADHD	34 (24.29 %**)
Bipolar Disorder		26 (2.4 %)
	Adolescents	21 (3.28 %)
	Children	5 (1.13 %)
	FDRs without ADHD	19 (2.02 %*)
	FDRs with ADHD	7 (5 %**)
Depressive Disorders		42 (3.87 %)
	Adolescents	38 (5.91 %)
	Children	4 (0.9 %)
	FDRs without ADHD	34 (3.6 %*)
	FDRs with ADHD	8 (5.71 %**)
Specific Language Disorder		482 (44.46 %)
	Adolescents	316 (49.22 %)

(*continued on next page*)

Table 1 (*continued*)

A) Family characteristics				
		Single-Parent	**Two-Parent**	**Overall Total**
	Children	166 (37.56 %)		
	FDRs without ADHD	414 (43.86 %*)		
	FDRs with ADHD	68 (48.57 %**)		
Tic and Tourette Syndrome		77 (7.11 %)		
	Adolescents	47 (7.32 %)		
	Children	30 (6.8 %)		
	FDRs without ADHD	64 (6.79 %*)		
	FDRs with ADHD	13 (9.29 %**)		
Autistic Spectrum Disorder		64 (5.9 %)		
	Adolescents	30 (4.67 %)		
	Children	34 (7.69 %)		
	FDRs without ADHD	58 (6.14 %*)		
	FDRs with ADHD	6 (4.29 %**)		
Intellectual Disabilities		79 (7.28 %)		
	Adolescents	42 (6.53 %)		
	Children	37 (8.37 %)		
	FDRs without ADHD	60 (6.35 %*)		
	FDRs with ADHD	19 (13.57 %**)		
Specific Learning Disorder		106 (9.78 %)		
	Adolescents	34 (5.29 %)		
	Children	72 (16.33 %)		
	FDRs without ADHD	93 (9.85 %*)		
	FDRs with ADHD	13 (9.29 %**)		
Developmental Coordination Disorder		79 (7.28 %)		
	Adolescents	40 (6.22 %)		
	Children	39 (8.82 %)		
	FDRs without ADHD	66 (6.98 %*)		
	FDRs with ADHD	13 (9.29 %**)		

Table 2

Results of the Generalised Linear Models on the Poisson family using as dependent variable the frequency of probands, and as independent variables the number of comorbidities (#Comorbidities), biological sex, and class age (adolescent / child). A) The results from the Generalised Linear Models on the Poisson family (LR $\chi 2$ = log-ratio chi-squared score; Df = degrees of freedom). B) The post-hoc tests on the #Comobilities X Sex interaction Bonferroni corrected, comparing the frequencies of children and adolescents grouped per each number of comorbidities and sex. Post-hoc comparisons among sexes in adolescents and children are always statistically significant ($p < .001$ in all cases), with greater comorbidity for males than females in all cases. C) The table shows the raw frequencies divided by class age, sex and number of comorbidities.

A)				LR $\chi 2$	Df	p-value
Adolescent_Children				2.8892	1	0.0892
#Comorbidities				157.7023	3	0.0000
Sex				441.6092	1	0.0000
Adolescent_Children X #Comorbidities				14.4407	3	0.0024
Adolescent_Children X Sex				1.5055	1	0.2198
#Comorbidities X Sex				9.8519	3	0.0199
Adolescent_Children X #Comorbidities X Sex				12.3326	3	0.0063
B)	***Females***					
	Number of comorbidities	estimate	SE	z.ratio	p.value	
	0	−1.099	0.667	−1.648	0.795	
	1	0.860	0.254	3.382	0.006	
	2	−0.189	0.276	−0.686	1.000	
	3 +	0.693	0.463	1.497	1.000	
	Males					
	Number of comorbidities	estimate	SE	z.ratio	p.value	
	0	−0.069	0.215	−0.322	1.000	
	1	0.297	0.096	3.084	0.016	
	2	0.533	0.140	3.807	0.001	
	3 +	0.651	0.158	4.124	0.000	
C)		0	1	2	3 +	
Adolescent	Female	3	52	24	14	
Adolescent	Male	42	253	138	117	
Adolescents total	*45*	*305*	*162*	*131*	*643*	
Children	Female	9	22	29	7	
Children	Male	45	188	81	61	
Children total	*54*	*210*	*110*	*68*	*442*	
Total	*99*	*515*	*272*	*199*	*1085*	

Parallel analysis suggested 4 components. PCA with Varimax rotation with Kaiser normalisation showed the best adaptability and accounted for 61 % of the variance (Table 3).

The components, defined by comorbid disorders with higher values were characterized by: 1°) "Anxiety -DEP-CD" (Anxiety disorders, Depressive Disorders and Conduct Disorder); 2°) "ID-SLangD-DCD" (Intellectual Disability, Specific Language Disorder and Developmental Coordination Disorder); 3°) "TIC-TS-ASD-vs.BD" (Tic and Tourette Disorders, Autistic Spectrum Disorder, v. Bipolar Disorder) 4°) "ODD vs. SLearnD disorders" (Oppositional Defiance Disorder v. Specific Learning Disorder) (Table SM1). The continuous values of the four components were computed as the sum of the presence of each comorbidity, weighted for the loadings (Table 3).We conducted ANCOVAs on the four components of PCA relative to proband comorbidities. In the Anxiety-DEP-CD component the effects of mother's educational level ($F(1, 1058) = 17.762$, $p < 0.001$; $\eta_p^2 = 0.017$, E-value = 1.213), FDRs with psychiatric disorders ($F(1, 1058) = 13.640$, $p < 0.001$; $\eta_p^2 = 0.013$, E-value = 1.931),Siblings with ADHD ($F(1, 1058) = 7.742$, $p = 0.005$; $\eta_p^2 = 0.007$, E-value = 1.272) and parental positive strategies ($F(1, 1058) = 4.919$, $p = 0.027$; $\eta_p^2 = 0.005$, E-value = 1.124) were statistically significant, indicating that the risk of probands with these comorbidity disorders was higher in those who had siblings with ADHD and FDRs psychiatric disorders, and mothers with low educational attainment and in those experiencing high positive parenting strategies. Moreover, this component shows higher values among probands adolescents with ADHD ($F(1, 1058) = 50.627$, $p < 0.001$; $\eta_p^2 = 0.046$, E-value = 1.146) and in those with pharmacological treatment ($F(1, 1058) = 18.540$, $p < 0.001$; $\eta_p^2 = 0.017$, E-value = 1.81). Moderation analysis on interactions regarding parental positive strategies x educational attainment, positive parenting × siblings with ADHD, positive parenting x FDRs psychiatric, educational attainment and siblings with ADHD, and educational attainment x siblings with ADHD did not reach statistical significance (all $t < 1.775$ and $p > 0.075$). A moderation effect was observed between one vs. two parents household x Siblings with ADHD ($t = -2.743$, $p = 0.006$, $b = -0.094$, 95 %CI = −0.171, 0.019, $\Delta R^2_{mo} = 0.68$), showing that mood and conduct comorbidities are more present in probands when siblings had ADHD and when lived with one-parent household, while there was no difference when siblings had not ADHD between one-parent and two-patents households ($\beta = 0.046$; M = 0.21, SD = 0.40; M = 0.17, SD = 0.35; respectively). Moreover one vs. two parents household x mother's education moderation effect was found ($t = 2.162$; $p = 0.031$, $b = 0.064$, 95 %CI = 0.007, 0.120, $\Delta R^2_{mo} = 0.63$), showing that probands with one-parent households and with mother's low education have more mood and CD comorbidities than those with two-parents households ($\beta = -0.11$; M = 0.41, SD =

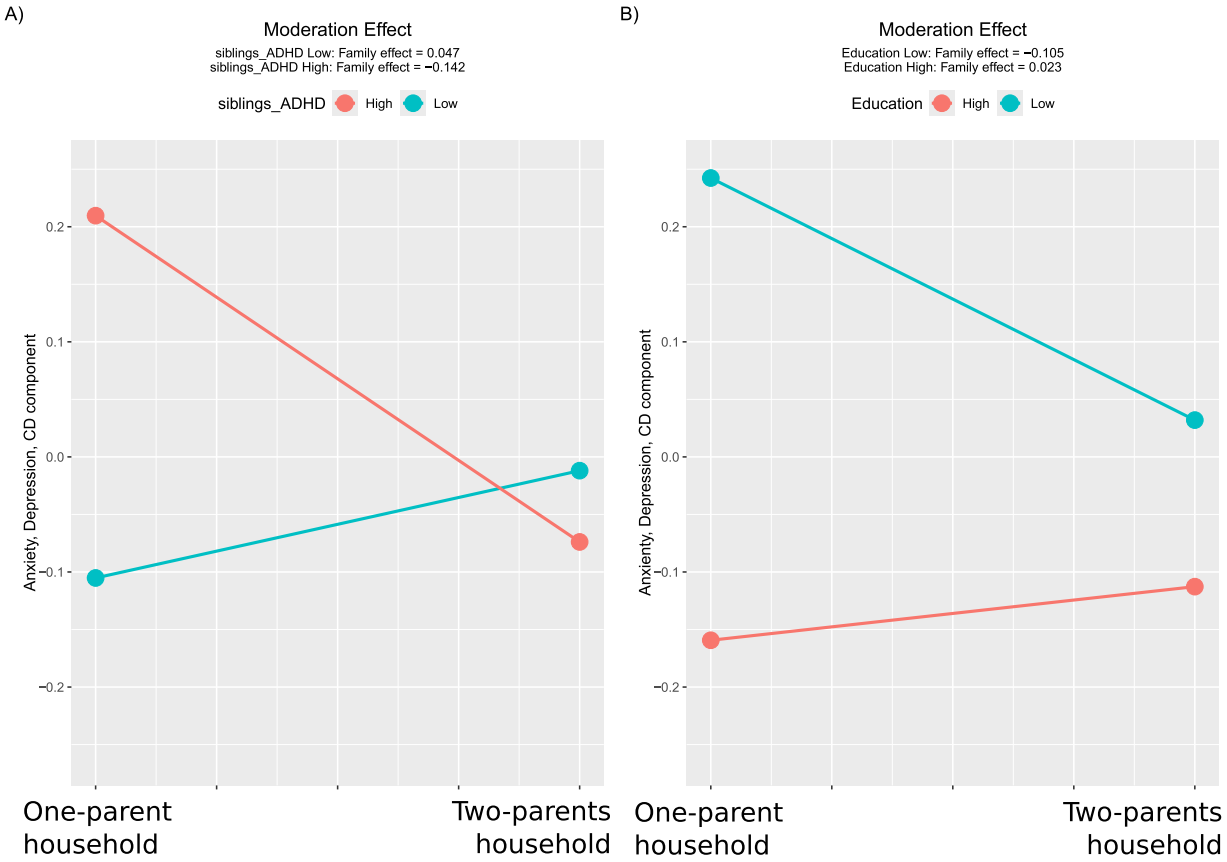

Fig. 1. Tumble graphs for the moderation effects on the Anxiety-DEP-CD component. A) Interaction between family and siblings with ADHD, where the family effect differs based on the presence of siblings with ADHD; B) Interaction between mother's education level and one vs. two parents household.

0.52; M = 0.13, SD = 0.32, respectively) while no difference was observable for mother's with high education level (β = −0.02; M = 0.13, SD = 0.33; M = 0.13, SD = 0.31). See Fig. 1 for graphical representations of the moderation effects.

In the "ID-SLangD-DCD" component we found the effects of the number of offspring (F(1, 1057) = 3.898, p = 0.049; $\eta_p^2 = 0.004$; E-value = 1.298), familial income (F(1, 1057) = 4.392, p = 0.036; $\eta_p^2 = 0.004$; E-value = 1.283) and age (F(1, 1057) = 32.468, p < 0.001; $\eta_p^2 = 0.03$; E-value = 1.254). A greater number of offspring was linked to higher scores in this component, while greater age and family income were linked to lower scores in this component. Siblings with ADHD approached statistical significance (F(1, 1057) = 2.922, p = 0.088; $\eta^2 = 0.003$; E-value = 1.170). We explored how the number of siblings might moderate this effect for two contrasting reasons. First, having other siblings may have a "buffering" or "diluting" effect, reducing the severity of ADHD symptoms. Second, larger families with multiple siblings can create an environment that could exacerbate symptoms of ADHD and its comorbidities. Research suggests that siblings in ADHD-affected families often experience varied situations, leading to different behavioural outcomes. These nonshared environmental influences may help lessen the severity of ADHD symptoms in siblings (Buschgens et al., 2008). Conversely, larger families can also foster rivalry, aggression, and avoidance among siblings—all factors that are correlated with ADHD symptoms (Ayyash et al., 2023). Moderation analysis showed a significant interaction between the number of offspring and siblings with ADHD (t = 3.365, p = 0.001, b = −0.107, 95 %CI = 0.006, 0.193, $\Delta R_{mo}^2 = 0.94$, see Fig. 2). In particular, this component was more present when among offspring the number of siblings with ADHD is greater (β = −0.146, M = 0.19, sd = 0.37) compared to the number of offspring with low or absent sibling with ADHD (M = 0.37, sd = 0.55).

Moderation analysis between "siblings with ADHD" and familial income, and between "siblings with ADHD" and one vs. two parents household were not statistically significant (all |t| < 1.034 and all p > 0.3).

In the "TIC-TS-ASD vs. BD disorders" third component, the effects of age (F(1, 1053) = 5.144, p < 0.024; $\eta_p^2 = 0.005$, E-value = 1.230), siblings with ADHD (F(1, 1053) = 13.500, p < 0.001; $\eta_p^2 = 0.013$; E-value = 1.154) and mothers with ADHD (F(1, 1053) = 14.341, p < 0.001; $\eta_p^2 = 0.013$; E-value = 2.548) were statistically significant. The age effect showed that older probands had lower scores in this component (standardized β = −2.359). The siblings with ADHD effect showed that siblings without ADHD had higher scores (M = 0.04, sd = 0.21) than those with ADHD (M = −0.05, sd = 0.24). Finally, the mothers with ADHD effect showed that probands with mothers with ADHD had higher scores (M = 0.17, sd = 0.36) than those without ADHD (M = 0.03, sd = 0.21) in this component.

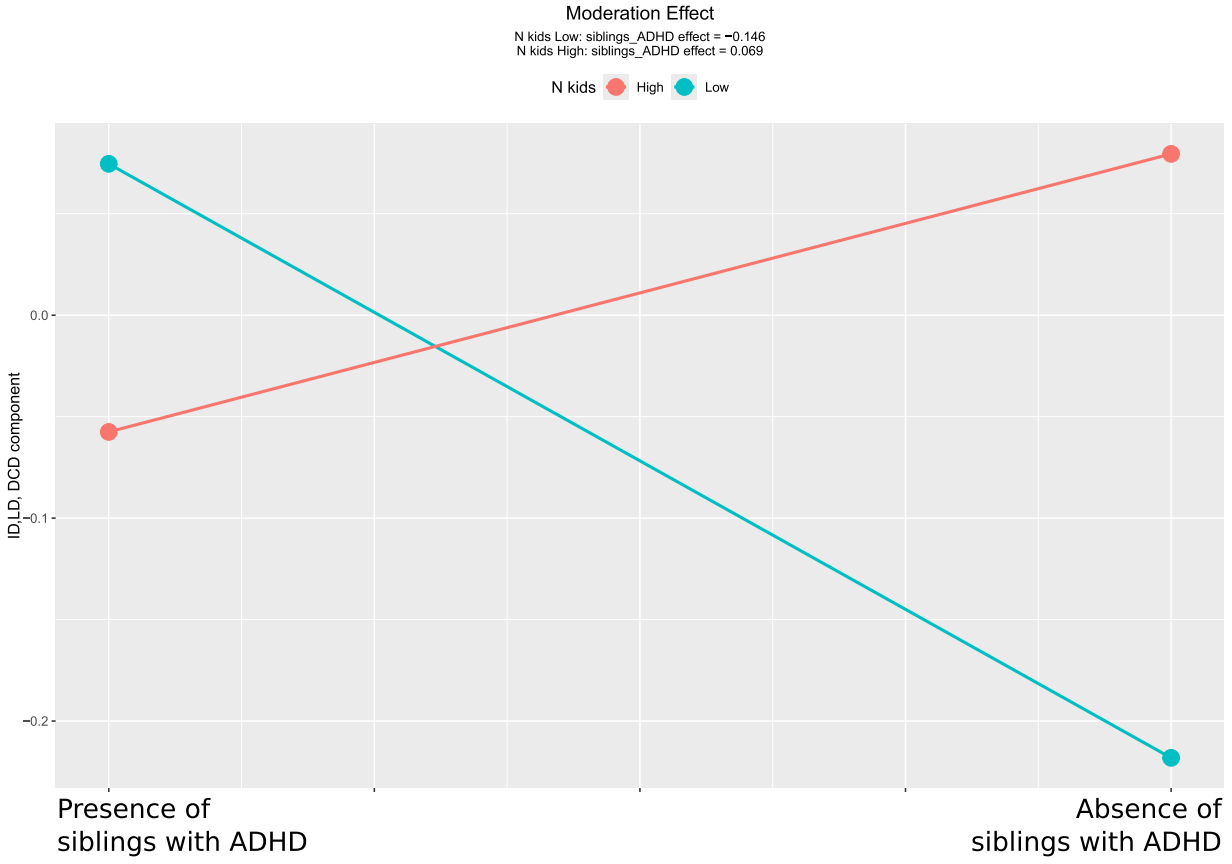

Fig. 2. Tumble graph for the moderation effect on the ID-SLangD-DCD component. Interaction between the number of offsprings moderated and the presence of siblings with ADHD.

Moderation analyses computed between significant regressors linked aspects related to hereditary factors of ADHD (siblings with ADHD, mother with ADHD, but also age, because hereditary and pre-natal factors are associated to earlier onset (Tobarra-Sanchez et al., 2022; Ayubi et al., 2021)). Moderation analysis regarding probands' age × mother with ADHD, and age × siblings with ADHD, did not reach statistical significance (all $t < 1.49$, all $p > 0.13$). When the interaction "mother with ADHD" and "siblings with ADHD" was evaluated ($t = -2.882$, $p = 0.004$, $b = -0.062$, 95 %CI = −0.139, −0.001, $\Delta R^2_{mo} = 0.94$), a significant effect emerged. Because the "TIC-TS-ASD vs. BD disorders" component has positive loadings with Tics-TS-ASD disorders, but negative loadings with BD disorders, two additional moderation analyses were computed. In the first one, the moderation between mother with ADHD and sibling with ADHD was computed on Tics-TS disorders, showing a significant effect ($t = -2.372$, $p = 0.018$, $b = -0.051$, 95 %CI = −0.088, −0.012, $\Delta R^2_{mo} = -0.26$), while the same moderation analysis on the BD disorder was not significant ($t = 1.791$, $p = 0.074$, $b = 0.038$, 95 %CI = −0.037, 0.170, $\Delta R^2_{mo} = 0.05$). In particular, in low frequency/absence of siblings with ADHD, the presence of the mother with ADHD led to a higher presence of probands' Tics-TS disorders (35 %) than in the presence of siblings with ADHD (0 %) ($\beta = 0.201$), while this difference was not present when the mother didn't have ADHD (siblings without ADHD = 7 %, siblings with ADHD = 3 %). The same moderation analysis was not statistically significant for ASD ($p > 0.05$). See Fig. 3 for the moderation analyses.

Finally, in the ODD vs SLearnD disorders component the effects of age ($F(1, 1057) = 13.566$, $p < 0.001$; $\eta^2_p = 0.013$; E-value = 1.803), sex ($F(1, 1057) = 10.098$, $p < 0.001$; $\eta^2_p = 0.009$; E-value = 1.667), drugs ($F(1, 1057) = 8.645$, $p = 0.003$; $\eta^2_p = 0.008$; E-value = 1.906), parental negative strategies ($F(1, 1057) = 4.155$, $p = 0.042$; $\eta^2_p = 0.004$; E-value = 1.141) and FDRs with neurodevelopmental disorders ($F(1, 1057) = 3.902$, $p = 0.048$; $\eta^2_p = 0.004$; E-value = 1.444), were statistically significant, indicating that this component was higher when probands with ADHD were males (males M = −0.085, sd = 0.53; females M = −0.237, sd = 0.55), in those using drugs (drugs M = −0.06 sd = 0.53; no drugs M = −0.14, sd = 0.53), when they were younger, and parents used higher negative parenting strategies and FDRs showed lower neurodevelopmental disorders. Moderation analysis did not show any statistically significant effect (all $t < 0.7$, all $p > 0.3$).

Analyzing the ODD alone comorbidity, age, sex or FDRs with neurodevelopmental disorders effects were not statistically significant, while parental negative and positive strategies ($\chi2(1) = 8.995$, $p = 0.003$; $\chi2(1) = 9.926$, $p = 0.002$) and the drugs ($\chi2(1) = 13.528$, $p < 0.001$) were statistically significant.

Analysing SLearnD alone, effects of sex $\chi2(1) = 7.767$, $p = 0.005$), age ($\chi2(1) = 24.861$, $p < 0.001$) and parental positive strategies

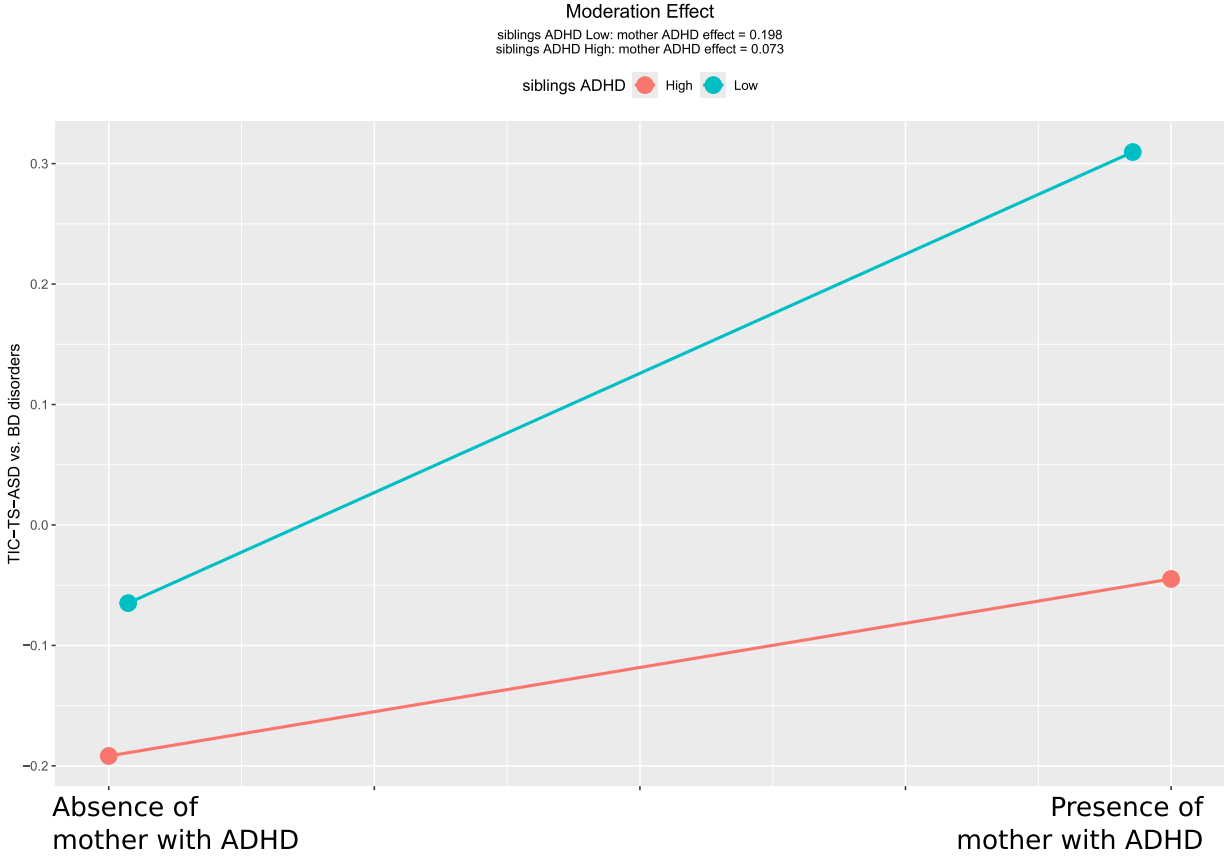

Fig. 3. Tumble graph for the moderation effect on the TIC-TS-ASD vs. BD disorders. Interaction between mother with/without ADHD and the presence of siblings with ADHD.

were statistically significant ($\chi2(1) = 9.054$, $p = 0.003$).

4. Discussion

Preliminarily, the findings highlight a wide range of psychiatric and neurodevelopmental comorbid disorders in subjects with ADHD, with a high percentage of two or more comorbid mental disorders. The findings support the notion that ADHD is most likely a multi-heterogeneous condition rather than a single homogeneous clinical entity (Inci et al., 2019).

The FDRs with ADHD are significantly related to the subjects' increased co-occurrence of comorbid disorders, as well as their own co-occurrence with other psychiatric or neurodevelopmental disorders (Yang et al., 2011). In addition, we find either a strong relationship between affected FDRs and socio-economic and environmental factors or between these factors themselves. These findings highlight that ADHD condition represents a biological substrate promoting either the association with other mental disorders or high social and household dysfunction.

4.1. Mood (anxiety, depression) and conduct disorders

Regarding the aim of our study, the findings showed a positive correlation between all comorbid psychiatric disorders, with a stronger association between "mood (anxiety, depression) and conduct disorders" in the first component. Although shared genetic risk variants and strong correlations (Anttila et al., 2018) support the nature of their co-occurrence, very little is known about the processes and mechanisms underlying their association. A trans-diagnostic approach to common dimensions across these disorders, focused on exploring underlying neuropsychological processes and pathophysiological circuits, could significantly contribute to understanding their relationships, as their differences have already been documented (Sonuga-Barke et al., 2016).

An important contribution comes from studies that provide evidence of interactions between these disorders. They document that ADHD in the presence of ODD accelerates the onset and worsens the severity of CD (Loeber et al., 2000), while anxiety disorders seem to strengthen the association between ADHD and ODD-CD (Humphreys et al., 2012); in turn, CD increases the risk of depression in patients with ADHD along the developmental age (Fischer et al., 2002; M.-J. Lee et al., 2016).

This group of disorders, which is most common in adolescence (Kessler et al., 2005), mainly increases in the presence of psychiatric disorders of FDRs, low maternal education and familial ADHD among siblings, especially when the latter two factors were present in a single-parent household, suggesting that multifactorial conditions (genetic and socio-environmental) seem to play a role in their association with ADHD. The findings also highlight the need to consider environmental factors, which are largely uncharacterised (Pickett & Wilkinson, 2010; Rocha et al., 2023), as a potential direct and moderating effect of risk in patterns of association between disorders (Bornovalova et al., 2010, 2014; Silberg et al., 2012). Consistently, recent studies document that mental disorders in offspring are strongly associated with parental psychopathology, low SES and single-parent households (Kinge et al., 2021). Furthermore, the findings appear to be supported by research highlighting that the overlap between psychiatric disorders is due to shared genetic variation with SES (Marees et al., 2021). The hypothesised mechanism is that SES, particularly low educational attainment, can influence genetic variance and correlations between clinical phenotypes, thereby increasing susceptibility. This effect is especially pronounced if the child is also exposed to cumulative environmental adversities (Björkenstam et al., 2018; Keilow et al., 2020).

4.2. Intellectual disabilities and language-motor ADHD comorbidities

This second component represents a group of early-onset neurodevelopmental disorders. Several studies support their co-occurrence and document that 1) The correlation between ADHD and ID is due to shared genetic factors and interactive mechanisms, as the pathophysiology of ADHD would influence IQ levels, increasing the risk of ID, and conversely, low intelligence would predict a subsequent increase in ADHD symptoms (Faraone et al., 2017). 2) Impairments in pragmatic-linguistic, socio-cognitive and executive functions are reported in patients with ADHD and ID (Çiray et al., 2022). 3) Difficulties in attention regulation and motor and language skills are early signs of low intellectual functioning (Sätilä et al., 2022). The acquisition of these skills at an early age reflects interactive mechanisms of neuropsychological and multiple brain networks between motor-linguistic, cognitive, and executive domains, which mutually contribute, at different times of development, to the development of each domain, their interaction as well as those of higher order skills (e.g. emotional-behavioural self-regulation; socio-cognitive abilities) (Löytömäki et al., 2020; Mundy et al., 2007; Petersen et al., 2015; Sheinkopf et al., 2004; Vaughan Van Hecke et al., 2012).

In our findings, the association between these comorbid disorders increases when the offspring had high sibling familiarity with ADHD and decreases in families with high economic levels and in children with older age. Supported by previous studies (Yochman et al., 2006), the findings seem to suggest that deficits in neuropsychological and pathophysiological brain circuits associated with ADHD, in the context of familiarity with the disorder, affect the interactive mechanisms between the above-mentioned domains, increasing the risk of association between these disorders. Moreover, the negative association between this group of disorders and depression supports that the effect of this risk factor is already present at a very early age.

The results are also consistent with studies that highlight that children from families with high household incomes in early childhood are less likely to have ADHD at 9–11 years of age (Lung et al., 2022; N. J. Spencer et al., 2022). Conversely, lower income is correlated with lower neurocognitive skills (Strenze, 2007), directly or indirectly increasing the risk of ADHD, ID and learning disabilities in offspring. As low income was correlated with paternal ADHD, although the effect of the latter on these disorders was lacking, we can hypothesise that it may represent a mediating factor in the association between income and ADHD condition in

offspring.

4.3. TIC/TS and ASD neurodevelopmental disorders

In the third component, we found that Tic/TS and ASD neurodevelopmental disorders, which are highly heritable and childhood-onset, were opposed to BD. ASD features often co-occur with ADHD and Tic/TS without representing complete comorbidity (Ronald et al., 2014). Genetic and clinical studies document that they are highly correlated (Canitano & Vivanti, 2007; P. H. Lee et al., 2019). A recent review concerning twins (see Tistarelli et al., 2020) reported that 1) autistic traits correlate with TS and ADHD, supporting that the origins of their co-occurrence could be attributable to genetic factors; 2) ADHD is a better predictor of ASD traits than vice versa and also plays a crucial role in the Tic/TS sub-phenotype that meets criteria for ASD (Darrow et al., 2017; Tylee et al., 2018). Despite interesting documentation, the nature of their relationship remains uncertain (Clarke et al., 2012). It is noteworthy that DCD loaded weakly but positively with Tic/TS-ASD disorders associated with ADHD, suggesting that altered neuropsychological processes and pathophysiological circuits linked to movement self-control could underlie their association.

Our findings showed that the association of Tic/TS-ASD comorbid disorders increases in the presence of maternal ADHD compared to low ADHD presence among siblings. The critical role of the mother with ADHD in increasing the risk of Tourette's (TS) and ASD in offspring is documented (Mathews & Grados, 2011; Musser et al., 2014). Nevertheless, recent studies suggested that pregnancy-related factors (PRF) such as smoking and alcohol are implicated in the pathogenesis of Tic/TS and ADHD in offspring (Ayubi et al., 2021), although Havdahl et al. (2022) cautioned against their causal association since PRF were strongly associated with polygenic ADHD mother scores. Similarly, other studies reported that maternal immune activation (MIA) in pregnancy could represent a causal pathway for fetal neuroinflammation, increasing the risk of ADHD-TS-ASD syndrome in offspring (Han et al., 2021). Despite several studies on this topic, most did not assess maternal ADHD (Kittel-Schneider et al., 2021). Conversely, we have not explored these factors, and although our findings highlight the direct effect of maternal ADHD, further studies are warranted to define better the interactions between maternal ADHD and pregnancy risk factors and their effect on ADHD, Tic/TS, and ASD disorders.

BD, and more weakly ID, defined a distinct opposing group within this component. Based on shared genetic risk factors and overlapping pathogenic mechanisms, some researchers suggest that ID, ASD, ADHD, schizophrenia, and BD disorders are better conceptualised as lying on an etiological and neurodevelopmental spectrum, with a gradient of decreasing impairment from severe intellectual disability, moderated in ADHD, and lower impairment in BD (Morris-Rosendahl & Crocq, 2020; Owen and O'Donovan, 2017). Consistently, some studies report a strong ID-BD association in adolescence after adjusting for socio-demographic confounders (Platt et al., 2019). BD and ID were separately and positively associated with psychiatric and neurodevelopmental disorders within the first and second components, respectively. Their collocation in two components and their negative association with ASD in the latter component might reflect biological heterogeneity within these disorders.

Finally, despite their respective and mutual high co-occurrence with ADHD, we found a negative relation between ODD and LD comorbidities loading within the fourth component. ADHD and LD share partial genetic influences, but they seem to involve opposite types of gene-environment interactions: bioecological for LD and diathesis-stress for ADHD (Rosenberg et al., 2012). Cross-lagged studies on twins suggest that ADHD symptoms may be considered a strong predictor of LD (Greven et al., 2012) and that comorbidity between ADHD inattentive type and LD is largely due to genetic influences (Wadsworth et al., 2015). On the other hand, longitudinal studies highlight a mutual influence between ADHD and ODD in increasing each other's risk along the continuum from childhood to adolescence or adulthood (Kuja-Halkola et al., 2015).

Together, these studies indicate that the covariance between ODD and LD may depend on different influences related to ADHD across developmental stages. Low rates of neurodevelopmental disorders among FDRs and high levels of negative parenting were identified as direct factors in this component. Negative parenting, when analysed separately, was associated with ODD comorbidity, aligning with the understanding that negative parenting strategies increase the risk of externalising behaviours in offspring (McRae et al., 2020) and potentially represent an important pathway for the co-occurrence of ADHD and ODD (Breaux et al., 2017).

In this component, CD was also positively associated with ODD, but their values loaded in a manner symmetrically opposed to those within the first component. Additionally, the age factor showed different trends within the two components. The risk of negative parenting increasing the presence of ODD (and CD) primarily in childhood supports the hypothesis that early maladaptive parenting practices may be exacerbated by a child's temperamental traits (e.g., explosive temperament), driving the association between the two disorders (Sonuga-Barke et al., 2016). Conversely, genetic and socio-environmental factors increase the risk of a CD (with ODD) and internalising disorders association mainly in adolescence.

Some limitations should be recognised: 1) Although we have explained that we used parents' self-reports of family economic status, this choice could represent a limitation of the study because it is not controlled for objective income measures. However, it is noteworthy that the percentage of adults with objective low (25 %) and high income (5 %), as reported by the National Institute of Statistics (ISTAT) in Italy in 2020 (the year of the study), is similar to that reported in our findings. It is documented that objective income does not always reflect the real familial economic condition and that subjective income is more closely linked to adult and child mental conditions, even after controlling for objective social status (Adler et al., 2000). 2) The rates of FDRs' ADHD and other mental disorders are likely underestimated for several reasons: a) only participants who sought psychiatric consultation were included in the study; b) few Italian adult mental health services are qualified for ADHD diagnosis and treatment (Zadra et al., 2020); and c) in Italy, free adult and child-adolescent mental health services have months-long waiting lists, meaning many patients were not captured through this cross-sectional study. 3) Other unexplored FDRs' demographic data and pregnancy risk factors would have enriched this research. Although the measures we used are those most commonly studied in ADHD conditions, we believe that, even in their absence, the results are worthy of interest.

5. Conclusions

The findings invite consideration of an ADHD nosology that accounts for highly associated comorbidity profiles and their related familial and environmental risk factors. This approach would contribute to identifying, at different stages of development, 1) mechanisms and risk factors underlying their association, 2) preventive strategies, and 3) targeted treatments for modifying the natural course of the ADHD condition.

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What this paper adds?

This paper demonstrates how the comorbidities of probands with ADHD are interconnected and examines the familial and environmental risk factors that impact them.

Declaration of Competing Interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.ridd.2025.105073.

Data availability

Data will be made available on request.

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