

Polygenic influences on networks of psychopathology symptoms: Unweaving the web

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Key Points

Question: Which individual symptoms of psychopathology does genetic risk associate with?

Findings: Combining psychological network and polygenic score approaches, this observational study (N=5,521) shows polygenic scores for psychopathology-related traits are primarily associated with a restricted number of trait-relevant and cross-trait symptoms. Results replicate in an independent sample following preregistered analyses (N=4,625).

Meaning: A shift from thinking of psychopathology at the disorder level to thinking about individual transdiagnostic symptoms may be beneficial to uncover novel insights in the development and comorbidity of psychopathology. Symptom-level analyses may be valuable in unravelling the complex (genetic) aetiology of psychiatric conditions and avoiding pitfalls resulting from disorder heterogeneity.

Abstract

Importance: Studies on polygenic risk for psychiatric traits commonly employ a disorder-level approach to phenotyping, implicitly considering disorders as homogenous constructs. However, symptom heterogeneity is ubiquitous, with many possible combinations of symptoms falling under the same disorder umbrella. Focusing on individual symptoms may shed light on the role of polygenic risk in psychopathology.

Objective: To determine (i) whether polygenic scores associate with all symptoms of psychiatric disorders, or with a subset of indicators and (ii) whether polygenic scores associate with comorbid phenotypes via specific sets of relevant symptoms.

Design: Data from two population-based cohort studies were used in the study. Data from children in the Avon Longitudinal Study of Parents and Children (ALSPAC) were included in the primary analysis, and data from children in the Twins Early Development Study (TEDS) were included in confirmatory analyses. Data analysis was conducted from October 2021 to January 2024.

Setting: Pregnant women based in the Southwest of England due to deliver in 1991-1992 were recruited in ALSPAC. Twins born in 1994-1996 were recruited in TEDS from population-based records.

Participants: Participants with available genetic data and whose mothers completed the Short Mood and Feelings Questionnaire and the Strength and Difficulties Questionnaire when children were 11 years of age were included.

Main outcomes and measures: Psychopathology relevant symptoms, such as hyperactivity, pro-sociality, depression, anxiety and peer and conduct problems at 11 years of age. Psychological networks were constructed including individual symptoms and polygenic scores for depression, anxiety, attention deficit-hyperactivity disorder (ADHD), Body Mass Index (BMI) and educational attainment (EA) in ALSPAC. Following a preregistered confirmatory analysis, network models were cross-validated in TEDS.

Results: We included 5,521 participants from ALSPAC (50.3% female) and 4,625 participants from TEDS (53.2% female). Polygenic scores associate preferentially with restricted subsets of core symptoms and indirectly associate with other, more distal symptoms of psychopathology (network edges range between $r=-0.074$ and $r=0.073$). Psychiatric polygenic scores associate with specific cross-disorder symptoms, and non-psychiatric polygenic scores associate with a variety of indicators across disorders, suggesting a contribution of non-psychiatric traits to comorbidity. For example, the polygenic score for ADHD associates with a core ADHD symptom, being easily distracted ($r=0.07$), and the polygenic score for BMI associates with symptoms across disorders, including being bullied ($r=0.053$), and not thinking things out ($r=0.041$).

Conclusions and relevance: Genetic associations observed at the disorder level may hide symptom-level heterogeneity. A symptom-level approach may enable a better understanding of the role of polygenic risk in shaping psychopathology and comorbidity.

Genetic studies have consistently shown that many genetic variants, each exerting a small effect, are involved in complex human traits, and together contribute to the likelihood of developing psychiatric disorders.¹ This polygenicity can be leveraged to compute polygenic scores (PGS), weighted sums of risk variants carried by an individual.^{2,3} PGS are a useful research tool indexing the genetic propensity to develop a particular psychiatric disorder, and have become instrumental in investigating the relationship between polygenic risk and psychiatric traits.

Findings based on PGS partly depend on the operationalisation of heterogeneous phenotypes. Notably, psychiatric disorders include a broad variety of symptoms, which, in combination, lead to numerous clinical presentations. This heterogeneity in psychiatric symptoms may bias genetic findings.⁴ In fact, evidence shows that symptoms have different heritability estimates, with some genetic effects specific to individual symptoms.⁵⁻⁷ Similarly, symptoms are differentially impacted by environmental risk factors and treatment, and contribute differently to relapse risk.⁸⁻¹⁰ In addition, some frequently comorbid disorders share a number of symptoms. For example, depression and anxiety frequently co-occur, and both feature insomnia, concentration problems and fatigue.¹¹ Findings on the shared genetic liability between comorbid disorders may therefore partly reflect a shared liability to transdiagnostic disorder features, such as endophenotypes or shared symptoms.

Therefore, analysing unidimensional phenotypes, such as symptoms, can be more informative to uncover relationships between biology and psychopathology¹² by better capturing the heterogeneity of psychiatric traits.¹³ Psychological network modelling is a recently developed statistical framework used to explore relationships between individual symptoms.¹⁴ Modelling observed variables as nodes (e.g., individual items on psychological scales), and their statistical associations as edges (e.g., partial correlations), networks allow for the visualisation of reciprocal dependencies between symptoms, as well as exploratory and confirmatory analyses.¹⁵ By focusing on a more granular, symptom-based phenotype, incorporating PGS in psychopathology networks can show whether PGS broadly associate

with all facets of a trait or relate specifically to a restricted set of symptoms, and whether PGS are associated with comorbid disorders via individual symptoms.

Here, we aimed to investigate how polygenic risk for psychopathology-related traits associates with individual symptoms of childhood psychopathology. Firstly, we examined the network structure of childhood behavioural and emotional symptoms, in combination with PGS for depression, anxiety, ADHD, as well as Body Mass Index (BMI) and Educational Attainment (EA). Secondly, we tested how well our initial exploratory findings replicated in an independent sample with a preregistered confirmatory network analysis.

Methods

Sample

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a large birth cohort study based in the Southwest of England which includes data on mothers, fathers, and children.^{16,17} Pregnant women residing in Avon and expected to deliver between 1991 and 1992 were recruited in the core sample (N=14,541), followed by additional recruitment waves adding 906 pregnancies (14,901 children alive at 1 year of age). Ethical approval for the study was obtained from the ALSPAC Ethics and Law Committee and the Local Research Ethics Committees. Informed consent was obtained following the recommendations of the ALSPAC Ethics and Law Committee. The study website contains details of data that is available through a fully searchable data dictionary (<http://www.bristol.ac.uk/alspac/researchers/our-data/>).

For primary analyses, a sample of children with available genome-wide data was selected (N=8,365). Genotyping, imputation, and quality control steps for ALSPAC data are detailed in eMethods. Questionnaires sent out when children were 11 years old were selected (N_{ALSPAC}=5,521, 50.3% female, mean age 11.8 years old).

For replication analyses, a sample was selected from the Twins Early Development Study (TEDS), a large UK-based longitudinal study of families of twins born between 1994 and 1996 (N=13,732).¹⁸ Identical selection steps were followed to match ALSPAC (N_{TEDS}=4,625, 53.2% female, mean age 11.27 years old). Information on TEDS quality control is detailed by Selzam and colleagues.¹⁹

In both cohorts, only genotyped participants whose mothers responded to at least 75% of questionnaire items were included in the final analytical sample, retaining N=5,521 ALSPAC children and N=4,625 TEDS children out of the initial cohorts. Among these included individuals, we imputed remaining missing items using multiple imputation by predictive mean matching via the R package *mice* (version 3.14.0)²⁰. Of the maximum

possible number of item data points (number of items x number of individuals) we imputed 0.73% of data points that were missing in ALSPAC and 0.1% in TEDS.

Measures

Questionnaires

Mother-rated reports of the Short Mood and Feelings Questionnaire (SMFQ, 13 items) and the Strength and Difficulties Questionnaire (SDQ, 25 items) were available in both ALSPAC and TEDS and were selected.^{21,22} Both are reliable and valid measures of, respectively, depression symptoms and social and emotional wellbeing, rated on a 3-point scale, 0 (“Not true”), 1 (“Sometimes”), or 2 (“True”). The SDQ is divided in five subscales: ‘Emotional problems’, ‘Peer problems’, ‘Hyperactivity’, ‘Conduct problems’ and ‘Pro-sociality’. Following scoring guidelines, five SDQ items were reverse coded (Items 7, 11, 14, 21, 25). eTable 1 contains mean values and endorsement rates of SDQ and SMFQ (hereafter referred to as scale items). Item 1 and 4 of the SMFQ (“*Miserable/unhappy*” and “*Restless*”) were not present in TEDS and were therefore excluded in ALSPAC to match datasets, leaving 11 items of the SMFQ in the analysis.

Statistical analyses

All analyses were carried out with R version 4.2.0, outlined in Figure 1, and reported following STROBE guidelines (eTable15).²³⁻²⁴ Example code is available on GitHub (github.com/giuliapiazza18/Unweaving-the-polygenic-web-pipeline).

PGS calculation

PGS for depression (based on GWAS summary statistics²⁵), anxiety,²⁶ ADHD,²⁷ BMI,²⁸ and EA²⁹ were calculated using LDpred2 in both cohorts.³⁰ To ensure no overlap between target and base data, we selected summary statistics from large GWAS that did not include ALSPAC and/or TEDS in their samples. PGS were generated by using the option

'LDPred2-auto' with default parameters (using the R package *bigsnpr* version 1.10.8),³¹ limited to HapMap3 variants³² and using target data as reference Linkage Disequilibrium (LD) panels. Recommended quality control steps on GWAS summary statistics were performed prior to generating the scores³³ (eMethods).

Covariates

To adjust for the effects of covariates on symptoms, age- and sex-regressed standardised residuals for each symptom were obtained from linear regressions and used as input data for networks in both cohorts. Scale items were adjusted for child age (around 11 years old) and sex. PGS were adjusted for the first 10 genetic principal components, child age, sex, and genotyping chip and batch.

Exploratory network estimation (N≈5,521)

Five cross-sectional networks with scale items and an individual PGS were estimated in ALSPAC (either depression, anxiety, ADHD, BMI or EA). Additional networks with all PGS and scale items and scale items only are available in the online material (eResults, eFigure 3).

Unregularised model search was used for network estimation via the R package *qgraph* (version 1.9.2) and its 'ggmModSelect' function,³⁴ shown to perform optimally in large samples ($N > 5,000$) compared to other network estimation techniques³⁵ (eMethods).

The resulting networks were visualised using the Fruchterman-Reingold algorithm.³⁶ The accuracy of network parameters was investigated with the R package *bootnet* (version 1.5).³⁷ One thousand nonparametric bootstraps were calculated for all network edge weights. Network weights matrices are reported in eTables 7-13. Additionally, we report covariate-adjusted correlations between PGS and scale items (i.e., correlations between each PGS and each scale item, only adjusted for covariates but not adjusted for all other relationships between nodes, in contrast with network analyses) in eTable 14.

Confirmatory Network Estimation (N≈4,625)

We conducted a preregistered confirmatory analysis (osf.io/7y2g8) using the R package *psychometrics* (version 0.10) (figure 1).¹⁴ First, we tested whether the pattern of presence or absence of associations between items (network structure) was replicated in the secondary sample (model 1). Second, we tested whether the estimates of these associations (network edges) were comparable across samples (model 2). Third, we repeated these steps focusing particularly on associations between PGS and symptoms (models 3-5).

Specifically, in model 1, we assessed how well network structures derived in the primary sample fit in our secondary sample using standard fit indices (Root Mean Square Error of Approximation, RMSEA; Comparative Fit Index, CFI). In model 2, in a combined dataset, we evaluated the fit of a model with equality constraints on network edges across cohorts, i.e., a model in which all ALSPAC and TEDS edges were set to be equal. For example, we extracted the structure of the network with the ADHD PGS derived in ALSPAC and, in model 1, we tested the fit of this structure in TEDS. In model 2, we set all edges in the ADHD PGS network to have equal weights in ALSPAC and TEDS and evaluated model fit.

In model 3, we tested the overall significance of all edges connecting to the PGS node in a combined dataset. First, we estimated a model where all edges connecting the PGS were set to zero (model 3). For example, if the ADHD PGS was connected to items '*Easily distracted*' and '*Child cheats*' in primary results, both edges were set to zero. Second, we compared this to the original model, where these edges were retained as non-zero. In model 4, these steps were repeated on each edge connecting to PGS. For example, we set the edge connecting the ADHD PGS to item '*Easily distracted*' to zero and compared this to the original model, which included the non-zero edge. Lastly, in model 5, individual edges connecting to PGS were free to vary between cohorts. For example, the edge connecting the ADHD PGS to item '*Easily distracted*' was allowed to freely vary between ALSPAC and TEDS. We compared this to a model where this edge was set to be equal.

P-values were adjusted for multiple comparisons with False Discovery Rate correction (FDR) using the Benjamini-Hochberg method (alpha = 5%) and the R package *stats* (version 4.2.0) in model 4 (34 tests) and model 5 (35 tests).^{24,38}

Results

Exploratory analyses

PGS preferentially associated with specific items of their corresponding traits. For example, the ADHD PGS (figure 2c) was only associated with one hyperactivity item: '*Easily distracted*' (HYP.3) and the depression PGS (figure 2a) was associated with depression symptom '*Not enjoying anything*' (DEP.2).

Additionally, psychiatric PGS did not associate only with trait-concordant items but showed cross-trait associations. For example, in addition to its within-trait associations, the ADHD PGS also associated with the item '*Child cheats*' (COND.4) in the conduct problems subscale, and the depression PGS also associated with '*Being bullied*' (PEER.4) in the peer problems subscale. Similarly, the anxiety PGS was associated with depression node '*Feeling lonely*' (DEP.10) (figure 2b). Moreover, PGS associated with a broader set of items based on covariate-adjusted correlations (i.e. adjusted for covariates, but not adjusted for all relationships between nodes as in network analyses) (eTable 14).

Lastly, non-psychiatric traits were associated with symptoms across disorders. The BMI PGS (figure 3a) associated positively with conduct, peer, pro-sociality, and hyperactivity problems and negatively with emotional issues, and the EA PGS negatively associated with items belonging to most subscales, as well as most hyperactivity (figure 3b). Nonparametric bootstraps showed edges were estimated accurately, as sample values were comparable to bootstrap mean edge weights (eFigure 2).

Confirmatory analyses

Overall, networks replicated well across datasets. Model 1 and 2 indicated network models were successfully replicated in the secondary sample. All network structures derived in ALSPAC showed good model fit in TEDS based on standard fit indices in model 1 (Table 1). Similarly, when setting equality constraints between ALSPAC and TEDS edges (model 2), model fit was good across all networks (eTable 3). Although standard fit indices were comparatively better when edges were not constrained to be equal across samples, indices

accounting for model complexity (e.g., the Bayesian Information Criterion) consistently favoured models with constrained edges.

Edges connecting PGS were statistically significant in all networks, as models including these edges (models 3 and 4) fit better than models that excluded them (eTables 4-5). In addition, results from model 5 show that PGS had similar associations with items across cohorts. Models constraining PGS edges to be equal in ALSPAC and TEDS were preferred to models which lifted these equality constraints, except the edge between the EA PGS and item '*Child cheats*' (COND.4). This difference, however, did not survive corrections for multiple comparisons.

Discussion

We examined the associations between childhood psychopathology symptoms and PGS for psychiatric disorders and relevant traits using a network approach. We found that (i) psychiatric PGS are associated with a core subset of indicators of their corresponding traits and (ii) PGS are not only associated with symptoms of their respective trait but show direct cross-trait associations. These findings were replicated in an independent sample and, as discussed below, suggest that the relationship between (non-)psychiatric polygenic risk and psychopathology traits may be mediated by specific factors or other symptoms.

Trait-relevant associations between PGS and symptoms

PGS associated with a selection of items measuring their corresponding trait. For example, the ADHD PGS was only positively associated with one item in the hyperactivity subscale, '*Easily distracted*' (HYP.3). This result suggests the association between ADHD and the polygenic risk for ADHD might be preferentially explained by the association with cognitive-attentional elements of the disorder. Similarly, the depression PGS was associated positively with anhedonia ('*Not enjoying anything*', DEP.2), suggesting the polygenic risk for depression might primarily influence prominent features of the disorder, associated with the greatest impairment.³⁹ When associations between items and PGS were not adjusted for all relationships between network nodes (i.e., in covariate-adjusted correlations, eTable14), PGS associated with a broader set of items than those identified by network analysis.

Taken together, these results suggest that associations between PGS and psychiatric traits might be preferentially explained by the association with core symptoms, rather than reflect uniform associations with all symptoms as commonly implied by disorder-level analyses. These core symptoms may be key mediators in the relationships between PGS and other, more distal symptoms of psychopathology.

Cross-trait associations between PGS and symptoms

PGS for psychiatric disorders were also found to be associated with items that did not directly measure PGS-concordant phenotypes. Notably, the anxiety PGS was associated with depression symptom '*Feeling lonely*' (DEP.10). This may indicate that a shared genetic influence on individual symptoms of depression and anxiety contributes to their frequent co-occurrence.

Similarly, the EA PGS was negatively associated with individual hyperactivity items. Previous evidence suggests higher EA PGS predict lower ADHD symptoms and better inhibitory control.⁴⁰ Indeed, our networks showed the EA PGS was negatively associated with cheating (COND.4), having poor attention (HYP.5) and being easily distracted (HYP.3) and impulsive (HYP.5), and also positively associated with internalising and peer problems, such as being solitary (PEER.1), having many worries (EMO.2), not being liked (PEER.3), and not volunteering to help others (PRO.5). This may suggest that childhood EA is a reflection of social and cognitive processes that also play a part in most internalising and externalising disorders.

Furthermore, the BMI and depression PGS associated with peer problems, specifically with being bullied (PEER.4). In turn, being bullied was positively associated with being lonely (DEP.10) and often unhappy (EMO.3), suggesting being bullied may mediate the relationship between these PGS and depression symptoms. This is also consistent with recent evidence showing the genetic predisposition to higher BMI, depression and ADHD is associated with bullying victimisation in children.⁴¹ Pre-existing vulnerability to mental illness might lead to exposure to bullying in childhood, which in turn exacerbates emotional difficulties in adolescence,⁴² hyperactivity and impulsivity, inattention, and conduct problems.⁴³ This represents a pattern of evocative gene-environment correlation: children who are predisposed to developing a high BMI might, in some contexts, evoke particular reactions in their environment, such as bullying.⁴⁴ Unfavourable environments, in turn, affect mental and physical health. This can have cascading effects, as stress in early life mediates the association between the genetic predisposition to high BMI and later depression.⁴⁵

In sum, adopting a network approach to phenotyping can suggest potential pathways to developing psychiatric traits by highlighting indirect paths from polygenic risk to later psychopathology via intermediate phenotypes. Taking a dimensional view of psychopathology, we investigated the extent to which common genetic variation in the population (indexed by PGS) associates with individual differences in symptoms. Findings should be replicated in high-risk or clinical cohorts.

Limitations

A few limitations of this study merit comment. First, the partial correlations evidenced in our study cannot be assumed to reflect causal mechanisms.

Second, results derived from our discovery cohort (ALSPAC) may be affected by overfitting, which, consequently, could affect results in the combined sample of both cohorts (models 3 and 4). As such, edges between PGS and scale items derived in the confirmatory sample are the most conservative estimates (Table 2). Models investigating differences in edges between cohorts (model 5) were implemented to minimise this issue. In fact, we did not observe any systematic deflation of estimates in the second cohort, reducing the likelihood of inflated estimates in the discovery cohort.

Third, polygenic scoring is a proxy for individual genetic liability, and it does not capture the full heritability of a trait (SNP-heritability) due to measurement error, meaning there are likely associations between genetic liabilities and symptoms that our analysis was not able to detect. The PGS calculated in this study vary in predictive power, in accordance with the GWAS they were derived from. This may explain some findings, such as the EA PGS associating with more symptoms of ADHD than the ADHD PGS itself.

Lastly, ALSPAC and TEDS are affected by attrition¹⁶⁻¹⁸. Therefore, replications of these findings in representative cohorts with high retention rates are warranted. Similarly, this analysis was limited to participants of European descent. As more diverse samples are being made available for genetic research, it will be important to verify whether our findings

hold true in those samples. Replication studies would also benefit from using more normally distributed item data and more granular genetic data (e.g. symptom-level GWAS, see online Supplement).

Conclusion

Modelling polygenic risk in networks of psychological variables showed previously unreported patterns of associations that replicated across samples. Relationships between psychopathology-related PGS and childhood psychological difficulties suggest PGS are preferentially associated with specific trait-relevant and cross-trait symptoms. Introducing genetic data into psychological networks can provide new insights into the aetiology of comorbidity as well as identify potential pathways to the development of psychiatric traits.

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Data sharing statement

The ALSPAC resource is owned and provided by the University of Bristol. Data can be made available and accessed upon request, as detailed at

<https://www.bristol.ac.uk/alspac/researchers/access/>.

The TEDS resource is held by King's College London. Data can be made available, subject to a data sharing agreement, as detailed at [https://www.teds.ac.uk/researchers/teds-data-](https://www.teds.ac.uk/researchers/teds-data-access-policy)

[access-policy](https://www.teds.ac.uk/researchers/teds-data-access-policy).

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Figure 1: Analysis flow of the study, including network analysis in ALSPAC (a) and replication in TEDS (b)

ALSPAC: Avon Longitudinal Study of Parents and Children; TEDS: Twin Early Development Study; PGS: Polygenic Score; BMI: Body Mass Index; DEP: Depression; ANX: Anxiety; ADHD: attention deficit-hyperactivity disorder; EA: educational attainment; SDQ: Strength and Difficulties Questionnaire; SMFQ: Short Mood and Feelings Questionnaire; gLASSO: graphical least absolute shrinkage and selection operator; PC: principal component.

Figure 2 a-c: Networks of psychiatric polygenic scores and psychopathology symptoms

Plots of networks with depression PGS (a), anxiety PGS (b), ADHD PGS (c). Partial correlations between scale items are drawn in the plot when $|r| > 0.1$ for clarity (i.e., the threshold for *qgraph* visualisation of edges connecting scale items is 0.1). All partial correlations between PGS nodes and scale items are drawn (i.e., *qgraph* visualisation threshold is 0 for edges connecting PGS). All edges connecting PGS are blue when positive and red when negative. All edges connecting scale items are solid grey when positive and dotted grey when negative. Bold items in the legend indicate nodes connected to a PGS. PGS are in the centre of each graph and all other nodes are positioned according to an average layout obtained with the Fruchterman-Reingold algorithm. eFigure 1 includes all networks without thresholds and common layout.

Figure 3 a-b: Networks of non-psychiatric polygenic scores and psychopathology symptoms

Plots of networks with BMI PGS (a) and EA PGS (b). As in Figure 2, partial correlations between scale items are drawn in the plot when $|r| > 0.1$ for clarity, and all partial correlations between PGS nodes and scale items are drawn. All edges connecting PGS are blue when positive and red when negative. All edges connecting scale items are solid grey when positive and dotted grey when negative. Bold items in the legend indicate nodes connected to a PGS. PGS are in the centre of each graph and all other nodes are positioned according to an average layout obtained with the Fruchterman-Reingold algorithm.

Table 1: Model fit indices from model 1, testing the model fit of ALSPAC networks in TEDS.

CFI above 0.95 and RMSEA below 0.05 were considered indicators of good model fit and of successful replication of ALSPAC networks in TEDS. CFI: Comparative Fit Index; RMSEA: Root Mean Square Error of Approximation.

Fit index	ADHD PGS network	Depression PGS network	Anxiety PGS network	EA PGS network	BMI PGS network
CFI	0.977	0.979	0.978	0.978	0.977
RMSEA	0.021	0.020	0.021	0.021	0.021

Table 2. Weights (partial correlations) of the edges of interest in PGS networks.

These were derived from primary analyses (ALSPAC weights), confirmatory model 1 (TEDS weights) and confirmatory model 2 (constrained model weights). †:significantly different weight estimates in TEDS and ALSPAC based on uncorrected *p*-values in model 5. When correcting for multiple comparisons, the difference is non-significant. All other estimates are not significantly different in TEDS and ALSPAC based on both uncorrected and corrected *p*-values.

Network and items	Edge	ALSPAC weight	TEDS weight	Constrained model weight
EA PGS network				
Cheats†	COND.4--EA†	-0.049	-0.098	-0.072
Complained of sickness	EMO.1--EA	-0.044	-0.014	-0.031
Many worries	EMO.2--EA	0.040	0.053	0.046
Easily distracted	HYP.3--EA	-0.062	-0.044	-0.054
Does not think things out	HYP.4--EA	-0.052	-0.028	-0.040
Bad attention	HYP.5--EA	-0.048	-0.069	-0.058
Solitary	PEER.1--EA	0.037	0.010	0.025
Not generally liked	PEER.3--EA	0.036	0.027	0.033
Volunteers to help	PRO.5--EA	-0.078	-0.069	-0.074
BMI PGS network				
Steals	COND.5--BMI	0.048	0.039	0.044
Many fears	EMO.5--BMI	-0.039	-0.011	-0.026
Does not think things out	HYP.4--BMI	0.043	0.038	0.041
Bullied	PEER.4--BMI	0.051	0.054	0.053
Volunteers to help	PRO.5--BMI	0.074	0.073	0.073
ADHD PGS network				
Cheats	COND.4--ADHD	0.048	0.040	0.044
Easily distracted	HYP.3--ADHD	0.070	0.069	0.070
Depression PGS network				
Not enjoying anything	DEP.2--DEP	0.037	0.037	0.038
Bullied	PEER.4--DEP	0.055	0.036	0.047
Anxiety PGS network				
Felt lonely	DEP.10--ANX	0.040	0.014	0.028