Risk of Mental Disorders in Adolescence Associated with Peers' Diagnoses and Genetic Predispositions

Jussi Alho, PhD[1], Mai Gutvilig, MSc[1], Ripsa Niemi, MSc[1], Kimmo Suokas, MD[1], Kaisla Komulainen, PhD[1], Petri Böckerman, PhD[2,3], Marko Elovainio, PhD[1,4], Roger T. Webb, PhD[5], Christian Hakulinen, PhD[1,4]

[1]Department of Psychology, Faculty of Medicine, University of Helsinki, Helsinki, Finland

[2]School of Business and Economics, University of Jyväskylä, Jyväskylä, Finland

[3]Labour Institute for Economic Research LABORE, Helsinki, Finland

[4]Finnish Institute for Health and Welfare, Helsinki, Finland

[5]Centre for Mental Health and Safety, Division of Psychology & Mental Health, University of Manchester, Manchester Academic Health Sciences Centre (MAHSC), Manchester, UK

Corresponding author: Jussi Alho, University of Helsinki, P.O. Box 21, 00014, Helsinki, Finland. Email: jussi.alho@helsinki.fi, Phone: +358 40 322 8770

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Key Points

Question: Are exposures to peers' diagnosed mental disorders and peers' genetic predispositions associated with adolescents' subsequent risk of developing the same mental disorders?

Findings: In this nationwide cohort study of more than 600 000 Finnish adolescents, both peers' genetic predispositions – inferred from relatives' diagnoses – and peers' own mental disorder diagnoses were associated with adolescents' subsequent diagnosis of the same disorder. These associations were not explained by probands' own genetic predisposition or parental socioeconomic position.

Meaning: Peer networks, particularly in school settings, may influence mental health risk during adolescence and represent potential targets for preventive interventions.

Abstract

Importance: Peer networks may influence early psychiatric vulnerability, both through direct exposure to peers' mental disorders and via peers' genetic predispositions. Clarifying these peer influences could inform preventive strategies aiming to improve adolescent mental health.

Objective: To determine whether adolescents' exposure to peers' diagnosed mental disorders or genetic predispositions is associated with subsequent risk of the same disorders, and whether these associations differ across disorders or peer network contexts.

Design, Setting, and Participants: Nationwide register-based cohort study including 604 819 Finnish residents born 1985–2000. Participants were assigned to four peer network contexts: lower secondary school, upper secondary school, 1000-m grid, and postal code area.

Follow-up extended from age 17 until first mental disorder diagnosis, emigration, death, or December 31, 2023.

Exposures: (1) Peers' family-based genetic risk scores (FGRS) for mental disorders, estimated from diagnoses in first- through fifth-degree relatives; and (2) peers' own diagnoses of the same disorders. Both exposures were analyzed by quintiles and as continuous measures.

Main Outcomes and Measures: Time to an incident diagnosis of the same disorder after age 17. Associations were estimated using Cox proportional hazards models adjusted for sex, birth year, individual FGRS, and parental education and income level.

Results: Of 604 819 cohort members (50.3% male), 234 117 received a mental disorder diagnosis over a median 11.7-year follow-up. High vs low peer FGRS was associated with higher risk of the same disorder across all disorders and peer networks, although the associations were stronger for externalizing vs internalizing disorders and for school-based vs geographically defined peers, with the greatest associations observed for substance use disorders in upper secondary school (HR 1.41, 95% CI 1.35–1.47). In contrast, peer diagnoses showed stronger associations for internalizing disorders and were primarily observed in school-based peer networks, with the strongest association for mood disorders in upper secondary school (HR 1.21, 95% CI 1.19–1.24). These associations were not explained by probands' own FGRS or parental socioeconomic status.

Conclusions and Relevance: Our findings point to peer environments, particularly in upper secondary school settings, as important influences on mental health trajectories. More research is needed to elucidate the mechanisms underlying these findings.

Introduction

Adolescence is a critical period when many mental disorders emerge[1-3] and enduring peer networks, social behaviors, and coping patterns form.[4-6] Given the developmental salience of peer relationships, peer networks may influence early-life psychiatric vulnerability.[7-10] In addition to direct peer transmission of mental health, it has been suggested that peers' genetic predispositions may also shape adolescent mental health trajectories.[11-13]

In a nationwide registry study, exposure to school peers with a diagnosed mental disorder was associated with higher risk of the same disorder, suggesting social transmission of mental disorders within adolescent peer networks.[14] This association was most evident for mood and anxiety disorders. In parallel, another register-based study found that peers' family-based genetic predispositions for psychiatric disorders during adolescence were associated with higher risk of the same disorder, particularly drug and alcohol use disorders.[15] It remains unclear, however, how these two peer exposures – peers' diagnosed disorders and genetic predispositions – compare in their associations with mental disorder trajectories, and how these associations differ across disorders or peer network contexts.

In the present study, we used Finnish nationwide registry data to analyze whether peers' diagnosed internalizing and externalizing disorders, as well as their genetic predispositions, are associated with adolescents' later risk of the same disorders. We examined differences across disorder categories and between school-based and geographically defined peer networks. Genetic predispositions were quantified using family genetic risk scores (FGRS), which estimate liability based on information from first- through fifth-degree relatives.[16,17] Drawing on prior evidence,[14,15] we hypothesized that the risk of internalizing disorders would

be more strongly associated with peers' diagnoses, whereas the risk of externalizing disorders would be more strongly associated with peers' genetic predispositions.

Methods

Study population

The study population consisted of all Finnish residents born between January 1, 1985, and December 31, 2000. We linked information from several national administrative registers using pseudonymized personal identification numbers assigned to all Finnish residents since 1969. For school-based peers, we used information from two Statistics Finland national school registers (National Joint Application Register and Students data module) to assign all individuals to their final year of lower secondary school (9th grade) and first year of upper secondary school. For geographically defined peers, we used the INFRA Spatial data module of Statistics Finland to map residents in a nationwide grid of one-square kilometer frames and in statistical postal code areas.

Of all Finnish residents born in 1985–2000 (n=1 427 498), we excluded individuals who did not apply to upper secondary education; died or emigrated between their application and the start of upper secondary school; were in the same school year but belonged to a different birth cohort; had missing school or geographical information; had no registered relatives; or were born outside Finland and immigrated after the official school starting age (August 1 of the year of their 7th birthday). Additionally, individuals with no peers in their network (e.g., in sparsely populated 1000-meter grids) were excluded and, from each multiple birth (e.g., twins, triplets), one individual was randomly selected to avoid bias from first-degree relatives in the same peer network.

Of the remaining 648 459 cohort members, 43 640 were diagnosed with one of the examined mental disorders (ICD-10 diagnoses F10–F19, F30–F48, F90–F94) before age 17 and were therefore excluded from follow-up. Thus, the final analytical cohort consisted of 604 819 individuals.

Peer networks

Peer network levels included lower secondary school (9th grade), upper secondary school (first year), one-square kilometer grid, and statistical postal code area. All peer networks were defined in the calendar year of each cohort member's 16th birthday. Lower secondary school information was taken from the spring of that year and upper secondary school information from the fall, while residential location was based on the registered address at the end of the year. Each individual belonged to all four peer networks, which were coded independently within each birth cohort (e.g., individuals in the same school, school year, and birth year formed a peer network).

Measures

Diagnoses of mental disorders were extracted from the Finnish Care Register for Health Care and the Register of Primary Health Care Visits. In these registers, psychiatric inpatient care has been reliably recorded since 1975, secondary outpatient care since 1998, and primary care since 2011.[18] Diagnostic coding followed the ICD-8 between 1969 and 1986, the Finnish implementation of ICD-9 between 1987 and 1995, and ICD-10 from 1996 onward. In some primary care settings, International Classification of Primary Care, 2nd edition (ICPC-2) codes are used instead of ICD-10. ICD-8, ICD-9, and ICPC-2 codes were converted to corresponding ICD-10 subchapter categories,[1] and overlapping register entries were resolved using a predefined algorithm.[18]

The primary outcome measures were the time to an incident first diagnosis of externalizing and internalizing disorders occurring after age 17. Externalizing disorders included substance use disorders (F10–F19) and disruptive behavior disorders, including hyperkinetic, conduct, and mixed conduct-emotional disorders (F90–F92). Internalizing disorders included mood (F30–F39) and anxiety disorders (F40–F48), as well as childhood-onset emotional and social functioning disorders (F93–F94). In addition, we separately analyzed first diagnoses of substance use disorder (F10–F19), mood disorder (F30–F39), and anxiety disorder (F40–F48). Exposure variable was peer FGRS for the same disorder. We calculated the FGRS based on diagnoses in the probands' first- through fifth-degree relatives (**Supplementary Table S1**) following a previous study.[16] The final FGRS, accounting for relatives' risk-period completion, genetic relatedness, cohort- and sex-specific liability, shared environmental effects of cohabitation (**Supplementary Table S2)**, number of relatives, and regional and temporal variation, reflects an individual's mean familial genetic risk for the disorder relative to the Finnish general population (see **Supplementary Methods S1** for more details).

The individual FGRS values were then used to estimate exposure to peer FGRS within each peer network, with residuals derived from cross-classified multilevel models representing the exposure variable (see **Supplementary Methods S2** for more details). Additionally, we applied a similar approach to estimate exposure to peers' mental disorder diagnoses received before the peers turned 17. We included the following covariates in the analytical models: sex, birth year, individual FGRS, parental education level (primary, upper secondary, or higher education), and parental income level in quintiles at the end of the year when probands reached age 16.

Statistical analysis

We fitted Cox proportional hazards models to separately estimate associations between peer FGRS and peer diagnoses with adolescents' subsequent risks of developing the same mental disorder across four peer network levels. Probands without a mental disorder diagnosis before age 17 were followed from age 17 until first mental disorder diagnosis, emigration, death, or December 31, 2023, whichever occurred first. We categorized the residuals from the cross-classified multilevel models representing peer FGRS and peer diagnoses (**Supplementary Methods S2**) into quintiles to define exposure groups and report hazard ratios (HRs) and 95% confidence intervals (CIs) for low vs high peer exposure contrast. As a secondary exposure specification, the exposure variables were standardized (z-scored) and modeled linearly as continuous predictors.

For both exposure types, we estimated three models: Model 1 adjusted for sex and birth year; Model 2 additionally adjusted for individual FGRS; and Model 3 further adjusted for parental education and income level. To account for differences in the baseline risk between academic and vocational schools at the upper secondary school level, Cox proportional hazards models were stratified by school type for the upper secondary peer networks. In separate sensitivity analyses, we censored follow-up at ages 20, 25, or 30; quantified exposure to peer diagnoses using only diagnoses occurring after age 15 (and before age 17); and recalculated peer FGRS exposure using only peers without a mental disorder diagnosis by age 17.

Results

Descriptives of the cohort are reported in **Table 1** (for additional descriptives, see **Supplementary Table S3**). During 7.2 million person-years of follow-up, with a median of 11.7 years (IQR 7.5–16.5), 234 117 (38.7%) cohort members were diagnosed with any of the examined mental disorders. **Table 2** reports HRs for associations between the highest vs

lowest quintile of the two exposure types and subsequent diagnoses of externalizing and internalizing disorders across peer networks. **Figure 1** shows the corresponding HRs across all exposure quintiles (lowest quintile as reference). HRs for the associations between the exposure types and subsequent diagnoses of substance use disorder, mood disorder, and anxiety disorder are shown in **Supplementary Table S4** and **Supplementary Figure S1**.

Exposure to the highest vs lowest quintile of peer FGRS was associated with higher risk of proband diagnosis of the same disorder across all examined disorders and peer network levels in all three analytical models. Additional adjustment for individual FGRS, parental education, and parental income (Models 2 and 3) attenuated but did not eliminate these associations. Upper secondary school networks showed the strongest associations between peer FGRS and proband diagnosis across all disorders, with exposure quintiles displaying a clear dose–response gradient. Across both school-based peer networks, peer FGRS for externalizing disorders showed stronger associations with proband diagnosis (lower secondary: HR 1.12, 95% CI 1.09–1.15; upper secondary: HR 1.36, 95% CI 1.31–1.41) than peer FGRS for internalizing disorders (lower secondary: HR 1.06, 95% CI 1.04–1.07; upper secondary: HR 1.14, 95% CI 1.13–1.16) in the fully adjusted model (Model 3). Similarly stronger associations were observed for substance use disorders (lower secondary: HR 1.15, 95% CI 1.11–1.19; upper secondary: HR 1.41, 95% CI 1.35–1.47) compared to mood disorders (lower secondary: HR 1.06, 95% CI 1.04–1.08; upper secondary: HR 1.21, 95% CI 1.19–1.24) or anxiety disorders (lower secondary: HR 1.07, 95% CI 1.06–1.09; upper secondary: HR 1.13, 95% CI 1.11–1.15). The associations for externalizing disorders were also generally stronger in school-based than in geographically defined peer networks. For geographically defined peers, the associations were comparable across all disorders.

Exposure to the highest vs lowest quintile of peer diagnoses was associated with a higher risk of proband diagnosis for both externalizing and internalizing disorders across all three analytical models only in school-based peer networks (**Table 1**; **Figure 1**). In contrast to peer FGRS, the HRs for the associations between peer diagnoses and proband diagnosis in school-based networks were slightly larger for internalizing disorders (lower secondary: HR 1.05, 95% CI 1.03–1.06; upper secondary: HR 1.17, 95% CI 1.15–1.19) than for externalizing disorders (lower secondary: HR 1.03, 95% CI 1.01–1.06; upper secondary: HR 1.12, 95% CI 1.09–1.16). This difference was particularly evident between mood disorders (lower secondary: HR 1.06, 95% CI 1.04–1.08; upper secondary: HR 1.21, 95% CI 1.19–1.24) and substance use disorders (lower secondary: HR 0.99, 95% CI 0.96–1.03; upper secondary: HR 1.12, 95% CI 1.08–1.16). In geographical peer networks, significant associations in the fully adjusted model (Model 3) were observed only in postal code areas for internalizing disorders (HR 1.02, 95% CI 1.01–1.04) and mood disorders (HR 1.02, 95% CI 1.00–1.04).

When treated as a continuous predictor, peer FGRS was associated with a proband diagnosis of the same disorder across all disorders and peer network levels in the fully adjusted models (**Figure 2**; **Supplementary Table S5; Supplementary Figure S2**). Peer diagnoses were associated with a proband diagnosis of internalizing disorders, including mood and anxiety disorders, across all four peer network levels. For externalizing and substance use disorders, peer diagnoses were associated with a proband diagnosis only in school-based peer networks, yet less strongly than peer FGRS. For internalizing disorders, peer diagnoses were more strongly associated with a proband diagnosis than peer FGRS in upper secondary school networks.

Limiting the follow-up period to younger ages led to modest but consistent increases in HRs for both exposure types across most disorders, most notably in school-based peer networks (**Supplementary Table S6**). The largest HR increases were observed for peer diagnoses of internalizing disorders in school-based networks. The corresponding increase for peer FGRS was from 1.06 (95% CI 1.05–1.08) to 1.09 (95% CI 1.06–1.13). Restricting peer diagnoses exposure to diagnoses occurring after age 15 attenuated HRs across disorders (**Supplementary Table S7**). When peer FGRS was recalculated using only peers without a mental disorder diagnosis by age 17, the results remained similar, with only minor deviations (**Supplementary Table S8**).

Discussion

In this analysis of nationwide registry data from Finland including more than 600 000 persons, we found that both peers' genetic predispositions, inferred from relatives' diagnoses, and peers' own mental disorder diagnoses were associated with probands' subsequent diagnosis of the same disorder. These associations were not explained by probands' own genetic predisposition or parental socioeconomic position. The two exposure types showed distinct predictive patterns: peers' genetic predispositions were associated with proband diagnosis across all disorder categories and in both school-based and geographically defined peer networks, with particularly strong associations for substance use and other externalizing disorders in school-based networks. In contrast, peers' diagnoses showed the strongest associations for internalizing rather than externalizing disorders, with the clearest associations in school-based peer networks.

To our knowledge, this study is the first to directly compare associations of peer genetic predispositions and peer diagnoses with subsequent proband diagnosis. Consistent with

Salvatore et al.[15], associations between peers' genetic predispositions and proband diagnosis were stronger for substance use disorders than for mood or anxiety disorders, stronger for school-based than geographically defined peers, and stronger in upper than lower secondary school. When exposure to peers' genetic predispositions was restricted to peers without a mental disorder diagnosis by age 17, our findings remained similar, suggesting that these associations were not driven by peers' own diagnoses.

In our earlier study, classmates' mental disorder diagnoses in lower secondary school were associated with subsequent diagnosis, particularly for mood and anxiety disorders.[14] The present study extends these findings by showing that exposure to peers with a mood or anxiety disorder diagnosis within the same school year in both lower and upper secondary schools was associated with subsequent diagnosis of the same disorder, while no such associations were observed for geographically proximal peers. As with peers' genetic predispositions, these associations were stronger in upper than lower secondary school. Taken together, our findings provide further support for peer genetic effects[11-13,15] and social transmission of psychiatric risk in adolescence[14,19].

Consistently stronger associations in school-based than geographically defined peer networks suggest that schools are a primary setting for adolescent socialization and peer influence. Stronger associations in upper vs lower secondary school peer networks may reflect developmental specificity, with environmental exposures differing in their impact by developmental stage.[20] In adolescence, peer relationships become increasingly central, with late adolescence characterized by greater autonomy, closer peer interactions, and higher susceptibility to peer influence.[21-23] It is also worth noting that at follow-up start (age 17), probands had completed lower secondary school and were in the first year of the typically

three-year upper secondary school, which may partly explain the stronger associations observed in upper vs lower secondary school.

The contrasting predictive patterns of the two exposure types across disorder categories may reflect underlying mechanisms. Stronger associations of peer genetic predispositions with externalizing disorders—especially substance use—and stronger associations of peer diagnoses with internalizing disorders could result from differences in thresholds for help-seeking, treatment, and diagnosis. For example, substance use problems in adolescence are often under-recognized and undertreated, causing peer diagnoses to underestimate exposure prevalence whereas genetic predispositions capture latent risk regardless of diagnosis. A large, nationally representative U.S. study found that 51% of adolescents with depression or suicidality-related conditions received treatment, compared with only 10% of those meeting criteria for substance use disorder.[24] This disparity aligns with earlier studies showing that adolescents are less likely to recognize substance use problems as requiring professional help and expect greater stigma and negative social consequences compared with depression or anxiety.[25,26] For internalizing disorders, having peers with diagnosed depression or anxiety may reduce stigma, increase symptom awareness, and encourage treatment seeking, thereby promoting clinical detection and facilitating clustering of diagnoses.[27] The attenuation of hazard ratios observed when exposure to peer diagnoses was restricted to diagnoses occurring after age 15 – more likely to reflect contemporaneous symptom exposure – supports this normalization mechanism rather than direct symptom contagion as normalization does not require peers to be actively symptomatic. At the same time, this attenuation may also reflect the exclusion of cumulative exposure to peers' symptoms earlier in adolescence.

This study's primary strength was its use of nationwide population data and the inclusion of diagnoses from both primary and secondary health care. Nonetheless, several limitations warrant consideration. First, the FGRS approach relies on the accuracy of register-based family linkages and diagnoses. Although both primary and secondary health care data were examined, individuals who did not seek help or were diagnosed in primary care before 2011 were classified as unaffected. Moreover, because psychiatric inpatient care has been reliably recorded only since 1975, registry coverage for older relatives may be incomplete. We therefore set relatives' time at risk according to national registry availability. Importantly, previous studies have demonstrated the validity of FGRS.[16,17,28,29] Second, as with all observational cohort studies, we cannot rule out residual confounding due to unmeasured covariates. Third, although school-based and geographically defined peer networks provide reasonable proxies for adolescents' social contexts, we cannot verify how extensively probands interacted with all individuals in these networks. Finally, because Finland has a free education system and universal health care with minimal financial barriers, these findings may not generalize to countries with different institutional arrangements.

In conclusion, this nationwide study of over 600,000 Finnish residents suggests that peers' diagnoses and genetic predispositions for a mental disorder are associated with adolescents' risk of developing the same disorder, highlighting the importance of peer contexts in the etiology of mental disorders. Associations with peers' genetic predispositions were evident across all contexts, particularly for externalizing disorders in school-based networks, whereas associations with peers' diagnoses were more confined to school-based peer networks and strongest for internalizing disorders. These findings support the view that peer environments during adolescence—especially in upper secondary school—may play an important role in shaping subsequent mental health trajectories. Further research is needed to elucidate the

mechanisms underlying these associations and to inform the development of preventive interventions.

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Conflicts of Interest Disclosures

None reported.

Author Contributions

Dr. Alho has full access to all the data in the study and takes full responsibility for the integrity of the data and accuracy of the data analysis.

Study concept and design: Alho, Elovainio, Gutvilig, Hakulinen, Niemi

Acquisition, analysis or interpretation of data: All authors

Drafting of the manuscript: Alho, Hakulinen

Critical revision of the manuscript for important intellectual content: All authors

Statistical analysis: Alho, Gutvilig, Niemi

Obtained funding: Hakulinen, Elovainio

Study supervision: Hakulinen

Data Sharing Statement

Data available: No

Additional Information

Explanation for why data not available: Data for the present study is property of Statistics Finland and Finnish Institute of Health and Welfare. The data are available from these authorities, but restrictions apply.

References

1. Suokas K, Niemi R, Gutvilig M, et al. Lifetime incidence and age of onset of mental disorders, and 12-month service utilization in primary and secondary care: a Finnish nationwide registry study. *Epidemiol Psychiatr Sci*. Jun 6 2025;34:e31. doi:10.1017/S2045796025100061
2. Solmi M, Radua J, Olivola M, et al. Age at onset of mental disorders worldwide: large-scale meta-analysis of 192 epidemiological studies. *Molecular psychiatry*. 2022;27(1):281-295.
3. Kieling C, Buchweitz C, Caye A, et al. Worldwide prevalence and disability from mental disorders across childhood and adolescence: evidence from the global burden of disease study. *JAMA psychiatry*. 2024;81(4):347-356.
4. Sawyer SM, Azzopardi PS, Wickremarathne D, Patton GC. The age of adolescence. *The lancet child & adolescent health*. 2018;2(3):223-228.
5. Patton GC, Sawyer SM, Santelli JS, et al. Our future: a Lancet commission on adolescent health and wellbeing. *The Lancet*. 2016;387(10036):2423-2478.
6. Dahl RE, Allen NB, Wilbrecht L, Suleiman AB. Importance of investing in adolescence from a developmental science perspective. *Nature*. 2018;554(7693):441-450.
7. Rubin KH, Bukowski WM, Bowker JC. Children in peer groups. *Handbook of child psychology and developmental science*. 2015;4:175-222.
8. Giletta M, Choukas-Bradley S, Maes M, Linthicum KP, Card NA, Prinstein MJ. A meta-analysis of longitudinal peer influence effects in childhood and adolescence. *Psychological bulletin*. 2021;147(7):719.
9. Watts LL, Hamza EA, Bedewy DA, Moustafa AA. A meta-analysis study on peer influence and adolescent substance use. *Current psychology*. 2024;43(5):3866-3881.
10. Prinstein MJ, Giletta M. Peer relations and developmental psychopathology. *Developmental psychopathology*. 2016:1-53.
11. Baud A, Mulligan MK, Casale FP, et al. Genetic variation in the social environment contributes to health and disease. *PLoS genetics*. 2017;13(1):e1006498.
12. Domingue BW, Belsky DW. The social genome: Current findings and implications for the study of human genetics. *PLoS genetics*. 2017;13(3):e1006615.
13. Domingue BW, Belsky DW, Fletcher JM, Conley D, Boardman JD, Harris KM. The social genome of friends and schoolmates in the National Longitudinal Study of Adolescent to Adult Health. *Proceedings of the National Academy of Sciences*. 2018;115(4):702-707.
14. Alho J, Gutvilig M, Niemi R, et al. Transmission of Mental Disorders in Adolescent Peer Networks. *JAMA Psychiatry*. 2024;81(9):882-888. doi:10.1001/jamapsychiatry.2024.1126
15. Salvatore JE, Ohlsson H, Sundquist J, Sundquist K, Kendler KS. Peer Social Genetic Effects and the Etiology of Substance Use Disorders, Major Depression, and Anxiety Disorder in a Swedish National Sample. *Am J Psychiatry*. Sep 1 2024;181(9):824-833. doi:10.1176/appi.ajp.20230358
16. Kendler KS, Ohlsson H, Sundquist J, Sundquist K. Family genetic risk scores and the genetic architecture of major affective and psychotic disorders in a Swedish national sample. *JAMA psychiatry*. 2021;78(7):735-743.
17. Kendler KS, Ohlsson H, Sundquist J, Sundquist K. The patterns of family genetic risk scores for eleven major psychiatric and substance use disorders in a Swedish national sample. *Translational Psychiatry*. 2021;11(1):326.
18. Suokas K, Gutvilig M, Lumme S, Pirkola S, Hakulinen C. Enhancing the accuracy of register-based metrics: Comparing methods for handling overlapping psychiatric register

entries in Finnish healthcare registers. *International Journal of Methods in Psychiatric Research*. 2024;33(2):e2029.
19. Alho J, Webb RT, Gutvilig M, et al. Examination of self-harm clustering in adolescent peer networks: a nationwide registry cohort study in Finland. *Lancet Reg Health Eur*. Jan 2026;60:101517. doi:10.1016/j.lanepe.2025.101517
20. Cicchetti D, Rogosch FA. A developmental psychopathology perspective on adolescence. *Journal of consulting and clinical psychology*. 2002;70(1):6.
21. Steinberg L. Cognitive and affective development in adolescence. *Trends in cognitive sciences*. 2005;9(2):69-74.
22. Brown BB, Larson J. Peer relationships in adolescence. *Handbook of adolescent psychology*. 2009;2(Pt I):74-104.
23. Giordano PC. Relationships in adolescence. *Annu Rev Sociol*. 2003;29(1):257-281.
24. Lee D, Dusetzina SB, Patrick SW, Graves JA, Fry CE. Adolescent Treatment Landscape of Depression, Suicidality, and Substance Use Disorder in the US. *JAMA Health Forum*. Aug 1 2025;6(8):e252647. doi:10.1001/jamahealthforum.2025.2647
25. Lemmer D, Moessner M, Arnaud N, et al. An online vignette experiment on stigma and help-seeking attitudes towards five mental health problems in adolescents and emerging adults. *Scientific Reports*. 2025;15(1):29956.
26. Lubman D, Cheetham A, Jorm AF, et al. Australian adolescents' beliefs and help-seeking intentions towards peers experiencing symptoms of depression and alcohol misuse. *BMC public health*. 2017;17(1):658.
27. Marcotulli D, Foulkes L, Stringaris A. Editorial Perspective: How spreading mental health information can be (un-) helpful–a dynamic systems approach. Wiley Online Library; 2025.
28. Kendler KS, Ohlsson H, Sundquist J, Sundquist K. Relationship of family genetic risk score with diagnostic trajectory in a Swedish national sample of incident cases of major depression, bipolar disorder, other nonaffective psychosis, and schizophrenia. *JAMA psychiatry*. 2023;80(3):241-249.
29. Kendler KS, Ohlsson H, Bacanu S, Sundquist J, Sundquist K. Differences in genetic risk score profiles for drug use disorder, major depression, and ADHD as a function of sex, age at onset, recurrence, mode of ascertainment, and treatment. *Psychological Medicine*. 2023;53(8):3448-3460.

Table 1: Descriptive statistics of the analytical cohort (n=604 819).

Characteristic	No. or median	% or IQR
Diagnosis during follow-up		
Externalizing disorders	51 431	8.50
Internalizing disorders	219 560	36.30
Substance use disorders	32 169	5.32
Mood disorders	113 135	18.71
Anxiety disorders	188 982	31.25
Age at diagnosis		
Externalizing disorders	25.2	21.7–29.7
Internalizing disorders	25.0	21.6–29.3
Substance use disorders	23.7	20.7–27.8
Mood disorders	24.8	21.4–29.1
Anxiety disorders	25.7	22.2–29.9
Peer network size		
Lower secondary school	97	71–125
Upper secondary school	78	34–124
1000-m grid	11	5–20
Postal code area	55	32–85

Table 2: Associations of the two exposure types (peer FGRS and peer diagnoses) with subsequent risk of externalizing and internalizing disorders across peer networks.

	Peer FGRS						Peer diagnoses					
	Model 1		Model 2		Model 3		Model 1		Model 2		Model 3	
Diagnosis category and peer network level	**HR**	**95% CI**	**HR**	**95% CI**	**HR**	**95% CI**	**HR**	**95% CI**	**HR**	**95% CI**	**HR**	**95% CI**
Externalizing disorders												
Lower secondary school	1.22	1.18, 1.25	1.15	1.12, 1.18	1.12	1.09, 1.15	1.06	1.03, 1.09	1.05	1.02, 1.07	1.03	1.01, 1.06
Upper secondary school	1.47	1.42, 1.52	1.37	1.32, 1.42	1.36	1.31, 1.41	1.15	1.12, 1.19	1.13	1.09, 1.16	1.12	1.09, 1.16
1000-meter grid	1.13	1.10, 1.16	1.08	1.05, 1.11	1.05	1.02, 1.07	1.01	0.98, 1.04	1.00	0.97, 1.03	0.99	0.96, 1.02
Postal code area	1.14	1.11, 1.18	1.09	1.06, 1.12	1.06	1.03, 1.09	1.01	0.98, 1.04	1.00	0.97, 1.03	0.99	0.96, 1.02
Internalizing disorders												
Lower secondary school	1.09	1.08, 1.11	1.07	1.05, 1.08	1.06	1.04, 1.07	1.06	1.04, 1.07	1.05	1.03, 1.06	1.05	1.03, 1.06
Upper secondary school	1.18	1.16, 1.20	1.15	1.14, 1.17	1.14	1.13, 1.16	1.20	1.18, 1.21	1.18	1.16, 1.19	1.17	1.15, 1.19
1000-meter grid	1.08	1.06, 1.09	1.06	1.04, 1.07	1.04	1.03, 1.06	1.02	1.00, 1.03	1.01	1.00, 1.03	1.01	0.99, 1.02
Postal code area	1.08	1.06, 1.09	1.05	1.04, 1.07	1.04	1.03, 1.06	1.03	1.02, 1.05	1.03	1.01, 1.04	1.02	1.01, 1.04

Hazard ratios (HRs) and 95% confidence intervals (CIs) correspond to the association between the highest vs lowest quintile of the exposure and the subsequent risk of being diagnosed with the same disorder between age 17 and the end of follow-up, estimated across the different peer network levels. Model 1 was adjusted for sex and birth year. Model 2 was adjusted for sex, birth year, and individual FGRS. Model 3 was adjusted for sex, birth year, individual FGRS, parental education level, and parental income level.

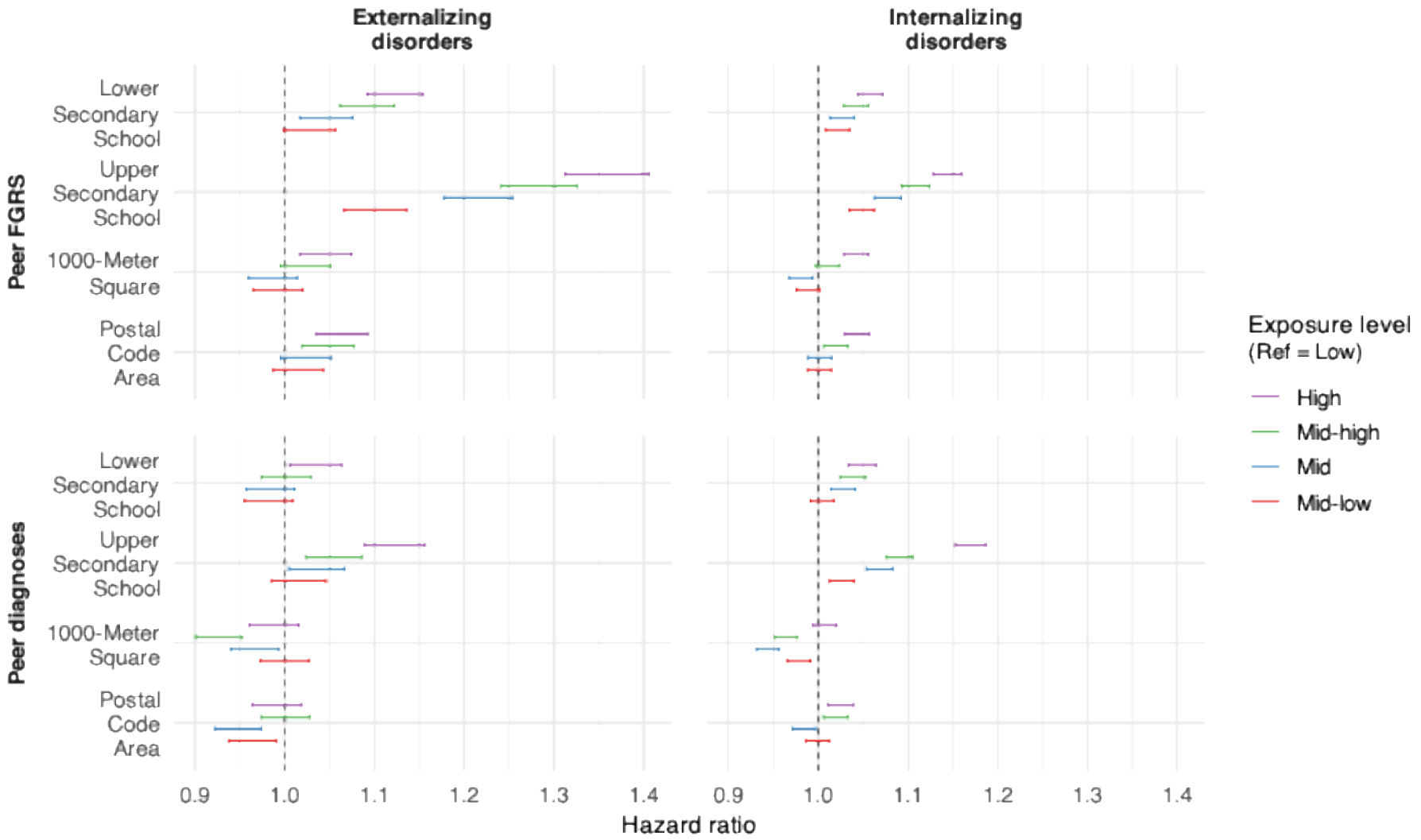

Figure 1: Associations of the two exposure types (peer FGRS and peer diagnoses) with subsequent risk of externalizing and internalizing disorders across peer networks. The plot shows the 95% CIs of the HRs from Cox proportional hazards models for the Mid-low (red), Mid (blue), Mid-high (green), and High

(purple) peer FGRS quintiles, with the Low quintile as reference. The models were adjusted for sex, birth year, individual FGRS, parental education level, and parental income level.

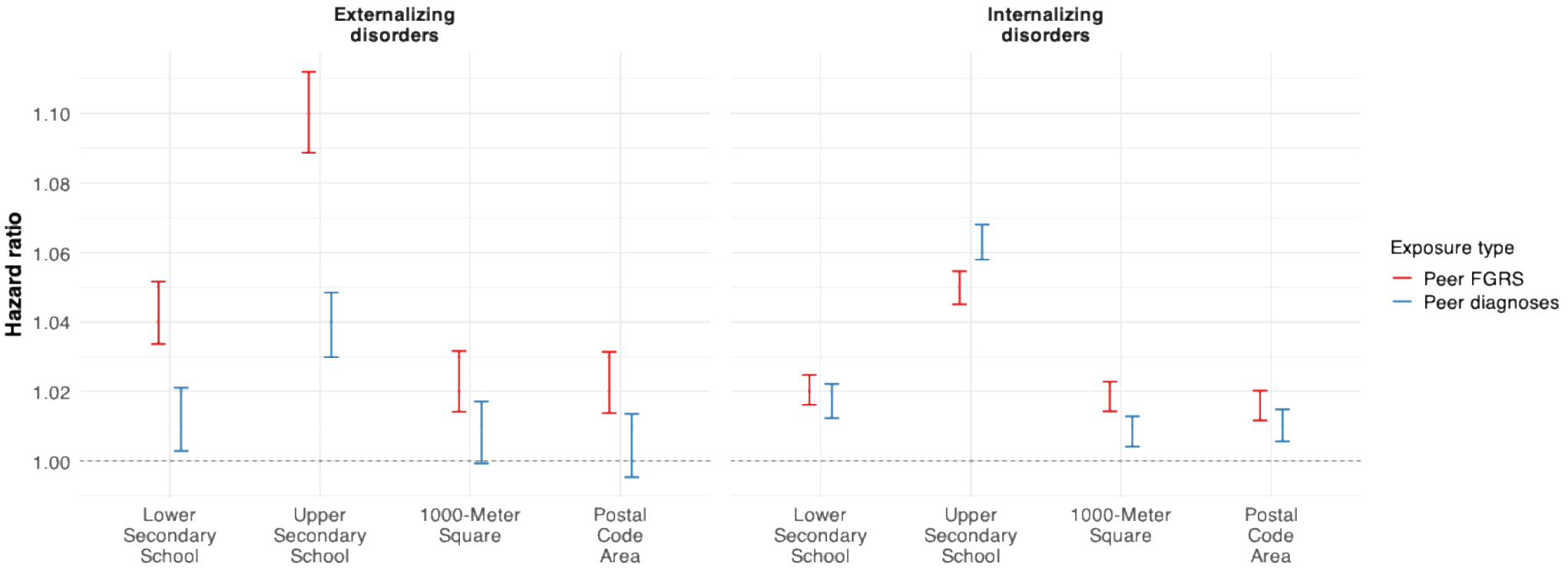

Figure 2: Associations of the two exposure types (peer FGRS and peer diagnoses) with subsequent risk of externalizing and internalizing disorders across peer networks, using a continuous predictor. The plot shows the 95% confidence intervals of the HRs from the Cox proportional hazards model for each association. The models were adjusted for sex, birth year, individual FGRS, parental education level, and parental income level.