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# Common genetic and environmental risk for personality disorders and psychotic-like experiences in young adult twins

**Martin Tesli**[1,2] | **Ragnar Nesvåg**[1] | **Unn K. Haukvik**[2,3] | **Kristin Gustavson**[1,4] | **Natalia Tesli**[2,3] | **Christine Friestad**[2,5] | **Torbjørn Skardhamar**[6] | **Øyvind Næss**[1,7] | **Nikolai Czajkowski**[1,4] | **Kenneth S. Kendler**[8,9] | **Ted Reichborn-Kjennerud**[1,3] | **Eivind Ystrom**[1,4,10]

[1]Department of Mental Disorders, Norwegian Institute of Public Health, Oslo, Norway

[2]Centre for Research and Education in Forensic Psychiatry, Department of Mental Health and Addiction, Oslo University Hospital, Oslo, Norway

[3]Institute of Clinical Medicine, University of Oslo, Oslo, Norway

[4]Department of Psychology, University of Oslo, Oslo, Norway

[5]University College of Norwegian Correctional Service, Lillestrøm, Norway

[6]Department of Sociology and Human Geography, University of Oslo, Oslo, Norway

[7]Department of Community Medicine and Global Health, Institute of Health and Society, University of Oslo, Oslo, Norway

[8]Virginia Institute for Psychiatric and Behavioral Genetics, Virginia Commonwealth University, Richmond, Virginia, USA

[9]Department of Psychiatry, Virginia Commonwealth University, Richmond, Virginia, USA

[10]PharmacoEpidemiology and Drug Safety Research Group, School of Pharmacy, University of Oslo, Oslo, Norway

**Correspondence**
Martin Tesli, Department of Mental Disorders, Norwegian Institute of Public Health, PO Box 222 Skøyen, N-0213 Oslo, Norway.
Email: martin.tesli@fhi.no

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## Abstract

**Introduction:** Psychotic-like experiences (PLE) have been associated with the subsequent emergence of psychotic disorders as well as several other domains of psychopathology. In this twin study, we estimated the genetic and environmental correlations between PLE and 10 personality disorders (PD).

**Methods:** Diagnoses of 10 PDs according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) and PLE from the Composite International Diagnostic Interview (CIDI) were retrieved for 2793 young adult twins from the Norwegian Twin Registry. Risk for having a PD and PLEs was modeled using item response theory. Biometric twin models were fitted to estimate the genetic and environmental correlations between PDs and PLEs. Co-twin control analysis was performed to estimate additional within-family risk for PLEs when having a PD.

**Results:** Phenotypic overlap between PDs and PLEs ranged from 14% to 44% in males and from 11% to 39% in females, with the highest overlap for borderline PD in both sexes. In general, we found higher genetic correlations

($r = 0.14$–$0.72$) than environmental correlations ($r = 0.06$–$0.28$) between PDs and PLEs. The highest genetic correlations between PLE and PDs were found for borderline ($r = 0.72$), paranoid ($r = 0.56$), schizotypal ($r = 0.56$) and antisocial PD ($r = 0.49$).

**Conclusion:** We found that the co-occurrence between PDs and PLE is the best explained by shared genetic determinants, with minor contributions from environmental factors. Interestingly, borderline PD was highly genetically correlated with PLE, warranting molecular genetic studies of this association.

**KEYWORDS**

genetic, personality disorders, psychosis, twin study

## 1 | INTRODUCTION

Personality disorders (PD) are a class of conditions characterized by enduring maladaptive patterns of cognition, emotional regulation, inner experience and social relations, and are estimated to affect up to 10% of the global population.[1–4] Psychotic disorders, which have a lifetime prevalence of ~1% worldwide,[5] are also characterized by distorted cognition, emotional dysregulation and social dysfunction, in addition to hallucinations and delusions. Despite overlapping symptoms and reports of co-morbidity between PDs and psychotic disorders,[6] these diagnostic groups are often treated as distinct entities in clinical practice. For example, while clinical guidelines recommend mainly psychosocial interventions to patients suffering from PDs,[7] antipsychotic medication is the treatment of choice for psychotic disorders. Such a distinction might have historical as well as empirical reasons—from the introduction of DSM-III in 1980 till the DSM-5 system in 2013, PDs were categorized under "axis II," whereas other severe mental disorders such as schizophrenia, bipolar disorder and major depressive disorder were categorized under "axis I." Although axis I and II were not conceptualized as mutually exclusive, different research approaches have to a large extent been applied to these two nosological domains. This dichotomy might have hampered investigations of shared features and common underlying mechanisms. In recent years, evidence of overlap between PDs and psychotic disorders have emerged, from both epidemiological[6] and clinically oriented[8,9] studies. However, it is unknown to which extent this co-morbidity results from shared genetic and environmental factors.

In general, twin studies have provided high heritability estimates for psychotic disorders such as schizophrenia (70%–80%),[10] whereas PDs have been found to be modestly to moderately heritable (30%–50%).[11] In the latest genome-wide association study (GWAS) of schizophrenia, which included more than 30,000 cases and 110,000 controls, over 100 genetic variants were discovered.[12] This stands in contrast to the largest GWAS of a PD (antisocial), including >16.000 individuals with no genome-wide significant results.[13] Molecular genetic studies of PDs appear to be a decade behind those of severe mental disorders like schizophrenia. Lack of well-powered genetic studies might give rise to the impression that there is a smaller genetic component to PDs than other mental disorders, which in turn might limit initiatives for further genetic investigations, thereby creating a vicious cycle.

Hitherto, indications of shared genetic and/or environmental underpinnings of PDs and psychotic disorders stem from family studies, which have found

### Significant Outcomes

- We found an overlap between personality disorders and psychotic-like experiences in young adults.
- Shared genetic factors explained more of the co-occurrence between personality disorders and psychotic-like experiences than environmental factors.
- Among the personality disorders, borderline personality disorder has the highest genetic correlation with psychotic-like experiences.

### Limitation

- As psychotic-like experiences might not necessarily be specific precursors to psychotic disorders, the current results should be replicated in clinical samples.
- Attrition rate is a potential source of bias in this sample.

familial co-aggregation of schizophrenia and several PDs, including schizotypal, schizoid, paranoid and avoidant PDs.[11] However, family studies are not able to distinguish between genetic and environmental effects to the same extent as twin studies. We have previously quantified shared genetic and environmental factors between personality and psychopathological symptoms in the Norwegian Institute of Public Health Twin panel[14,15] as well as between psychotic-like experiences (PLE) and cannabis use disorders.[16]

In the current twin study, we investigate common genetic and environmental risk underlying the relationship between 22 self-reported PLE and 10 PDs according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). PLEs, such as hallucinatory and delusional experiences, have been hypothesized to exist on a continuum from healthy individuals to patients with a diagnosis of schizophrenia.[17] Subclinical symptoms are defined as PLEs that do not cross the symptomatic threshold or imply sufficient functional impairment to warrant a diagnosis of a psychotic disorder. According to the psychosis continuum model[18] subclinical psychotic symptoms are markers for genetic vulnerability to psychotic illness. General population studies have demonstrated that 5–10% of presumably healthy people have experienced psychotic symptoms, and persistence of symptoms in adolescence may predict a later psychotic disorder.[19] PLEs have also been found to be associated with a range of other psychiatric symptoms and disorders, hereunder depression and anxiety.[20] Previously, twin studies have found high concordance between monozygotic twins for psychotic experiences among women,[21] and more recently molecular genetic studies have found PLEs to share genetic risk factors with psychotic disorders.[22] Earlier work using twin samples has mostly focused on selected positive psychotic symptoms, like hallucinations[23] and delusions[24] and the samples have not been population-based. In this population-based twin study, we include a wide range of PLEs.

In order to obtain a broad overview of patterns of shared and distinct genetic and environmental underpinnings between PDs and psychotic symptoms, we also investigate a range of PDs across the three different clusters. Cluster A comprises the "odd-eccentric," B the "dramatic-emotional," and C the "anxious-fearful" disorders, of which all categories either bear similarities at phenotypic level or have been found to co-occur in individuals and families with psychotic disorders.[11]

In the current study we aim to (1) estimate the overlap in genetic factors for 10 PDs and PLEs in a population-based sample of young adult twins, and (2) estimate within-family risk for PLEs when having PD symptoms.

In general, we hypothesize to find genetic and environmental correlations between the PLEs and PDs, albeit higher genetic than environmental, keeping in mind the high heritability estimates for psychotic disorders[25] and few established common environmental risk factors. Specifically, we expect to find high genetic correlations between PLE and the cluster A schizotypal and paranoid personality disorders, as well as the cluster B borderline and antisocial personality disorders, as indicated in previous family reports.[26,27] Further, schizotypal, paranoid and borderline personality disorders are defined by psychotic-like features in DSM-IV, such as paranoid ideations and unusual perceptional experiences.

Our findings may inform clinicians and researchers on the relation between psychotic experiences and PDs, which in turn might have relevance for diagnostic systems and clinical guidelines.

## 2 | METHODS

### 2.1 | Participants

The Norwegian Institute of Public Health Twin panel is a population-based cohort of twins born between 1967 and 1979.[28] Since 1992, self-report questionnaire and face-to-face diagnostic interview data have been collected as part of a mental health research project. Details regarding recruitment and assessments have been published previously.[29,30] All complete pairs of 8045 twins who had completed a questionnaire in 1998 were invited to a diagnostic interview for DSM-IV mental disorders. Between 1999 and 2004, 2801 twins completed the interview, of which 2793 twins had full data on psychotic symptoms and PDs. Thus, the current sample includes information on 2793 twins (63.5% female, mean age 28.2 y, range 19–36 y). Zygosity classification was based on questionnaire data and validated by microsatellite analysis for 676 same-sex twin pairs, resulting in less than 1% misclassification.[29] The current sample consisted of 898 monozygotic (MZ) females, 444 MZ males, 532 same-sex dizygotic (DZ) females, 235 same-sex DZ males, and 684 DZ twins from opposite-sex pairs (344 females and 340 males). Participation in the interview study was predicted by higher age and monozygosity, but not by any mental health indicator from the previous questionnaire data.[31]

### 2.2 | Procedures

All participants were assessed for lifetime DSM-IV axis I disorders using the Norwegian version of the computerized Munich Composite International Diagnostic Interview (M-CIDI).[32] The interviewers were mostly senior

psychology students and psychiatric nurses trained by teachers certified by the World Health Organization and supervised closely during the data collection period. Most interviews were conducted face-to-face. For practical reasons, 231 (8.3%) of the interviews were conducted over telephone. Members of a twin pair were assessed by different interviewers.

The psychosis module of the M-CIDI includes a screening for 22 psychotic symptoms. Individuals endorsing at least one of the screening items were administered the full module. All items concerning delusions were rated with two thresholds, the first indicating that the item was endorsed, and the second that the content was definite psychotic based on responses to a probing question. When reviewing the verbal responses, three of the delusion items ("Have you ever believed people were spying on you?," "Was there ever a time when you believed people were following you?," and "Have you been convinced that people you saw talking to each other were talking about you or laughing at you?") had a high number of false positive responses. Thus, only definite psychotic responses were kept in the analyses for these items while responses to both thresholds were kept for the remaining items. The items kept in the analysis will in the following be referred to as PLEs.

The Norwegian version of the Structured Interview for DSM-IV Personality was used to assess PDs. This instrument is a comprehensive semi-structured diagnostic interview for the assessment of all DSM-IV Axis II diagnoses, and it includes nonpejorative questions organized into topical sections rather than by disorders. This allows for a more natural flow of the interview and increases the likelihood that useful information from related questions may be considered when rating-related criteria within that section. The specific DSM-IV criterion associated with each set of questions is rated according to the following scoring guidelines: 0 indicates that the criterion is not present or is limited to rare isolated examples; 1, subthreshold (some evidence of the symptom, but not sufficiently pervasive for the criterion to be considered present); 2, present (criterion clearly present for most of the last 5 years); and 3, strongly present (criterion is associated with subjective distress or some impairment in social or occupational functioning or intimate relationships). The structured interview for DSM-IV Personality is conducted after the Axis I interview, which helps the interviewer distinguish longstanding behavior reported by the subject from temporary states due to an episodic psychiatric disorder. Interrater reliability was assessed based on two raters' scoring of 70 audiotaped interviews. Intraclass correlations for the number of endorsed borderline personality disorder criteria at the subthreshold (≥1) and threshold (≥2) level were 0.93 and 0.92, respectively. The polychoric correlation was 0.94.

The study was approved by the Norwegian Data Inspectorate and the Regional Committee for Medical and Health Research Ethics, and written informed consent was obtained from all participants after complete description of the study.

## 2.3 | Statistical Analysis

The classical twin model partitions the variance of the observed variable into the effects of three latent variables: additive genetic effects (A), share environmental effects (C), and non-shared environmental effects (E). A are the linear combinations to the phenotype of independent genetic loci. As DZ twins on average share half their genes, and MZ twins have identical genotypes, additive genetic effects are correlated 0.5 in DZ twins and 1.0 in MZ twins. Shared environmental factors are those aspects of the environment that have equal effect in both twins regardless of zygosity. Any remaining variance is attributed to environmental influences that are unique to each twin (E), including random measurement error. Structural equation modeling was used to fit the model for the relative contribution of A, C, and E to the data using maximum likelihood.

In the previous studies using this sample we have found unequal additive genetic effects for PLE in males and females,[16] but no sex differences in genetic effects for PDs.[33] Our models were therefore run with unequal parameters specified for PLE in males and females, equal parameters for PDs in males and females, unequal genetic and environmental covariance between PDs and PLE in males and females, but, according to the rationale of Neale et al.,[34] equal genetic and environmental correlations in males and females (i.e., Euclid's first axiom: things which are equal to the same thing are equal to each other). For all the variables we used ordinal variables with three categories. The level of PLE was categorized according to Nesvag et al.[16] where an ordinal was calculated by binning the latent distribution of the PLE factor into three levels. The PDs had three categories: 0 indicates no criteria present; 1, one criterion and 2, two or more criteria present.

# 3 | RESULTS

A total of 708 individuals (25.3%) were administered the full psychosis module in M-CIDI after endorsing one or more of the screening questions. Prevalence of individual with PLEs ranged from 0.1% (delusions about thought

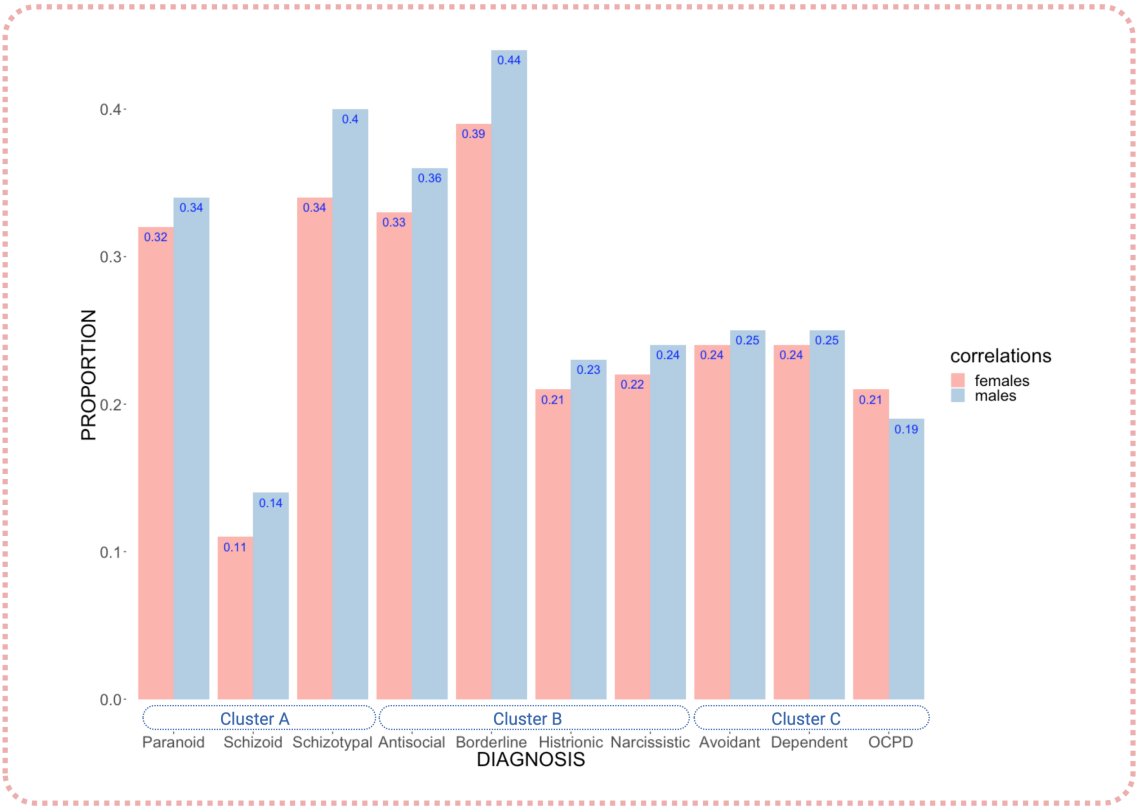


**FIGURE 1** Phenotypic correlations between DSM-5 personality disorder symptoms and psychotic like experiences in males and females. OCPD, Obsessive-compulsive Personality Disorder.

insertion) to 2.2% (delusions of reference). In Figure 1, we show the associations between PD symptoms and PLEs. All 10 PD symptoms were to some extent correlated with PLEs. However, there were broadly three patterns of associations. First, two cluster A PDs (i.e., schizotypal and paranoid) and two cluster B PDs (i.e., borderline and antisocial) were moderately correlated ($r = 0.32$ to 0.44) with PLEs. Second, the remaining two cluster B PDs (narcissistic and histrionic) and all three cluster C PDs (avoidant, dependent, and obsessive-compulsive personality disorders) were modestly correlated with PLEs ($r = 0.19$ to 0.25). Three, the remaining cluster A PD, schizoid, was weakly correlated with PLEs ($r = 0.11$ to 0.14). There were only small sex differences in association, and the pattern was similar across sex.

We found the PDs to be moderately heritable. Cluster A: Paranoid (19%), Schizoid (29%), and Schizotypal (25%); Cluster B: Antisocial (38%), Borderline (25%), Histrionic (32%), and Narcissistic (25%); and Cluster C: Avoidant (35%), Dependent (31%), and OCPD (27%). We estimated PLEs to be 56% heritable in males and 26% heritable in females.

In Figure 2, we present the genetic correlations between PDs and PLEs. Three broad patterns of genetic correlations occurred. First, four genetic factors for PDs from cluster A (paranoid and schizotypal) and cluster B (borderline and antisocial) were moderately to strongly correlated with genetic factors for PLEs ($r = 0.49$ to 0.72). Second, the genetic factors for one cluster C PD, OCPD, was weakly correlated with genetic factors for PLEs. Third, genetic factors for the remaining PDs (one cluster A, two cluster B, and two cluster C) were all modestly correlated with genetic factors for PLEs ($r = 0.26$ to 0.34).

Environmental correlations, which are attenuated by random measurement error, were all modest to weak ($r = 0.28$ to 0.06). The genetic and environmental covariances are presented in Supplementary Figure 1 (males) and 2 (females).

## 4 | DISCUSSION

In the current twin study, we have quantified genetic and environmental correlations between psychotic like experiences (PLE) and 10 personality disorders (PDs). In general, the genetic correlations were larger than the environmental, and the genetic correlations with PLE were stronger for PDs in cluster A (eccentric) and B (dramatic) than cluster C (anxious). We found the largest

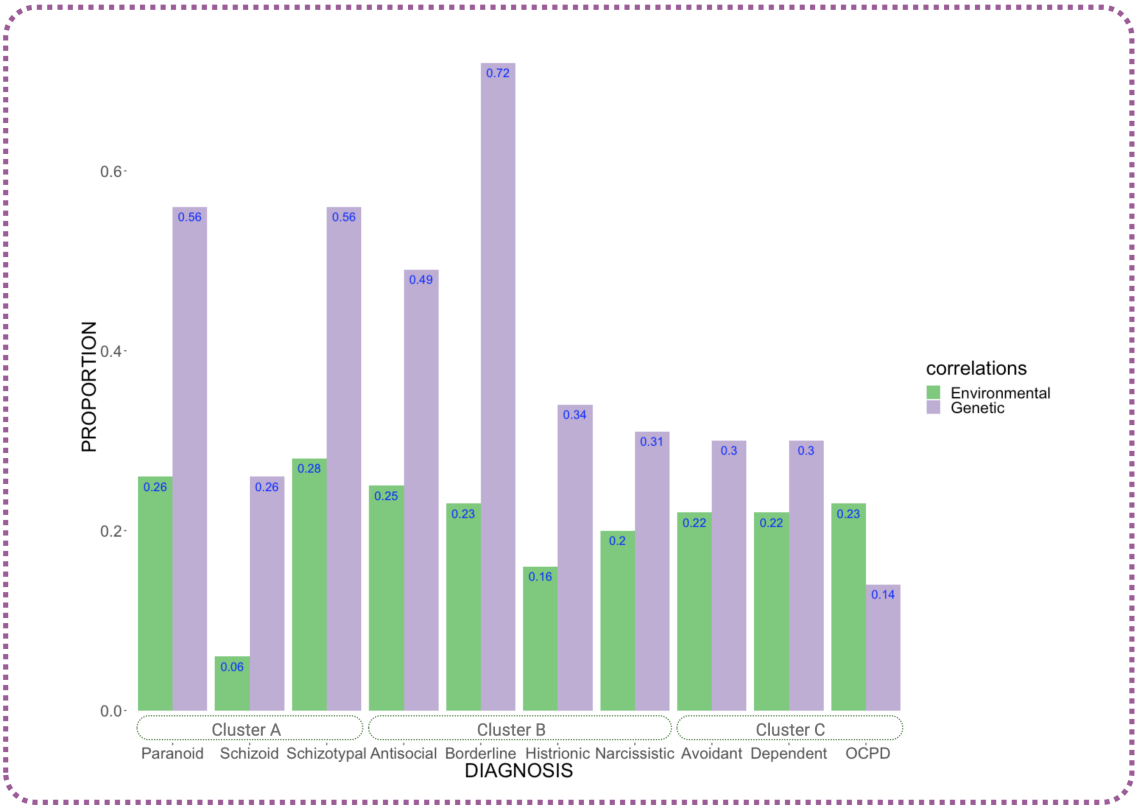


**FIGURE 2** Genetic and environmental correlations between DSM-5 personality disorders and psychotic-like experiences. OCPD, Obsessive-compulsive Personality Disorder

genetic correlation between PLE and borderline PD ($r = 0.72$). This pattern of correlations at genetic and environmental level (Figure 2) was to a large extent in accordance with the overlap at the phenotypic level (Figure 1). Our findings were also in line with observations at phenotypic level from previous studies, where familial co-aggregation of psychotic disorders and several PDs, including schizotypal, schizoid, paranoid and avoidant PDs[11] have been reported. One clinical study found that among patients diagnosed with borderline personality disorder, 67%–77% fulfilled criteria for a diagnosis within the broader "schizophrenia spectrum".[9] In fact, the term "borderline" was coined in the late 19th century denoting a condition characterized by a symptom profile crossing the borderline between neurotic and psychotic disorders. Nevertheless, borderline was included among the PDs in DSM-III in 1980 and is still considered as such in DSM-5 (2013). However, more recently borderline PD has been abolished as a diagnosis in the ICD-11 classification where it is now only possible to specify as an additional non-diagnostic specifier (WHO, 2023). Despite clinical guidelines advising against psychopharmacological treatment as a primary intervention, patients suffering from borderline PD often use a combination of psychotropic drugs including second generation antipsychotics.[35,36] In contrast to antidepressants, antipsychotics have shown some effect on borderline symptoms in randomized clinical trials, in particular impulsivity and aggression.[37] As for PDs in cluster A (eccentric), these conditions share clinical characteristics with psychotic disorders, including paranoid ideations, brief hallucinations and delusions, as well as social disinterest and withdrawal.[38] Schizotypal PD is often considered a premorbid condition of psychotic disorders, in that nearly 30% of adolescents with this condition later develop a psychotic disorder.[38] It is noteworthy that *schizotypal PD* is considered a PD in DSM-5, whereas *schizotypal disorder* is categorized under psychotic disorders in ICD-10. Moreover, the term *schizotypal* was introduced in the 1960s as an abbreviation of *schizophrenic phenotype*, whereas *schizoid PD* has been found to share clinical features with high functioning autism spectrum disorders/Asperger syndrome.[39] Thus, our findings of a high genetic correlation between PLE and cluster A, in particular schizotypal and paranoid PDs, but schizoid to a smaller degree, are in accordance with clinical evidence and nosologic conceptualizations. Furthermore, our findings of genetic correlations between PLE and several PDs across the proposed clusters, provide further support to the notion that these clusters are essentially theoretical constructs with limited empirical validity.

We also found high genetic correlation between PLE and antisocial PD ($r = 0.49$), which is of interest for several reasons. Disentangling the relationship between antisocial behavior such as violent crime and the presence of psychosis lies at the heart of criminal insanity evaluations in many countries. Although there is, to the best of our knowledge, a lack of representative, epidemiological studies on the phenotypic overlap between antisocial PD and psychotic disorders, one clinical study found a prevalence of antisocial PD in 6.7% of patients with schizophrenia spectrum disorders, which is higher than in the general population.[40,41] Further, it has been shown that patients with psychotic disorders are at increased risk of violent crime, which is a core defining feature of antisocial PD.[42] Our findings suggest that the phenotypic co-occurrence between psychotic symptoms and antisocial PD is partially explained by shared genetic underpinnings ($r = 0.49$). When further explored, this finding might provide important knowledge to researchers and clinicians in the field of forensic psychiatry.

## 4.1 | Strengths and limitations

One of the strengths of this study is the use of structured diagnostic interviews in a large population-based sample of twins. Members of each twin pair were assessed by independent interviewers to reduce bias, and the use of IRT allowed for investigation of the construct validity of the latent PS and PLE.

However, the results must be interpreted with some potential limitations in mind. Firstly, PLEs have been associated not only with psychotic disorders per se, but also with a range of other symptom domains, and might thus lack specificity. Nevertheless, psychotic disorders are also associated with several other symptom domains and mental disorders, and might in e.g., manic and depressive episodes represent the degree of *severity* as well as the degree of *overlap* with another symptom dimension. This discussion is however outside the scope of the current study. Secondly, the validity of psychotic symptoms as rated by lay interviewers has been questioned in previous studies using the CIDI. The IRT modeling results indicate that the psychosis items in M-CIDI are reliable and demonstrate good construct validity in a non-clinical population setting, yet it is not clear if the same pattern of results would be found in a clinical psychosis sample. Thirdly, questions regarding personality and PLEs refer to lifetime occurrence without information about temporality. Fourthly, only 43% of invited twins completed the diagnostic interview. Attrition was predicted by male sex, dizygosity, and lower level of education, and not by mental health indicators.[31] Fifthly, the current study employs DSM-IV criteria for PDs, which are similar to ICD-10 categorization, despite the fact that the recently developed ICD-11 has made major changes to the classification of PDs. These modifications imply a main emphasis on severity with a secondary specifier or combinations of specifiers. For example, borderline has been abandoned as a separate category, and included as an additional specifier. Moreover, the recent evidence suggests that the borderline category might be conceptualized as a global PD severity index. Indeed, if the "borderline" category truly reflects the severity of the PD to a greater extent than identifying a separate entity, this could fit well with our finding that borderline PD has the highest genetic correlation with PLE among the PDs, as PLE and psychotic symptoms generally may also serve as a manifestation of the severity of mental disorders. Future studies should investigate how the ICD-11 classifications correlate genetically with psychotic disorders and PLE.

In conclusion, the present study demonstrates an association between symptoms of PDs and higher risk of PLEs in the general population. The twin modeling results indicate that the relationship may be explained by common genetic and environmental factors, the former to a larger extent than the latter. The genetic correlations with PLE are strongest for borderline, paranoid, schizotypal and antisocial PDs.

## CONFLICT OF INTEREST STATEMENT

The authors declare that they have no competing interests.

## PEER REVIEW

The peer review history for this article is available at https://www.webofscience.com/api/gateway/wos/peer-review/10.1111/acps.13596.

## DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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## SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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