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Why Psychopathology Research Should Avoid Studying One Mental Disorder at a Time: An Intergenerational and Developmental Evidence Base for Understanding "p"

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Richie Poulton is now deceased.

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Most etiological research on mental disorders tries to find specific causes of specific disorders. However, the search for causal specificity has been elusive. In fact, new evidence reveals that the major etiological factors are transdiagnostic. One possible reason for why the search for specificity has been elusive is that most disorders are more similar than they are distinct, an idea that prompted research on "p"—the tendency of a person to develop a wide range of different mental disorders. Here we bring together data from unique sources to provide the intergenerational and developmental empirical evidence base for understanding "p." Men and women with a history of mental disorders tend to mate with partners who are also prone to have mental disorders, but not necessarily the same disorders. This creates a situation whereby their offspring, whether through genetic and/or environmental transmission, are at heightened risk of developing a variety of different mental disorders, but which specific disorder offspring ultimately develop is not easy to predict. Given that offspring inherit these multiple liabilities, it may not surprise that these liabilities manifest as different disorders at different points throughout their lives, but which disorder emerges at a particular time is difficult to foretell. The intergenerational and developmental evidence about the familiality and course of mental disorders helps to deconstruct "p" and invites psychopathology research and clinical science to reconsider their common approach to studying one mental disorder at a time.

General Scientific Summary

This article brings together data about assortative mating, intergenerational transmission, and the longitudinal course of mental disorders to reveal how so many disorders become correlated. Together, the evidence yields a developmental understanding of why the search for specific causes, consequences and treatments of different mental disorders has been elusive. The data underscore the need to reshape measurement and design practices in psychopathology to advance etiological research and deliver more effective treatment.

Keywords: structure of psychopathology, assortative mating, intergenerational transmission, developmental psychopathology

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Most research on mental disorders tries to find specific causes of specific disorders. For example: What genetic factors cause schizophrenia? What altered brain morphology causes attention deficit hyperactivity disorder (ADHD)? What type of childhood adversities cause depression? However, the search for causal specificity has been elusive. In fact, new evidence reveals that major etiological factors are transdiagnostic. This is the conclusion that is emerging from genome-wide association studies, where the genetic risks for different disorders are highly correlated (Anttila et al., 2018; Grotzinger et al., 2025); from neuroimaging studies, where structural and functional alterations are shared by many forms of psychopathology (Opel et al., 2020); and from research in developmental psychopathology, which shows the same environmental and psychosocial risks for many different disorders (McMahon et al., 2003; Zhou et al., 2025).

Why has the search for specificity been elusive? We propose that one reason is that most disorders are more similar than they are distinct. However, in trying to test and refute this hypothesis, much psychopathology research has relied on data that may be limiting the field's ability to conduct and evaluate research on the tendency of a person to develop a wide range of different mental disorders, "p" (Caspi et al., 2014). Most of the data analyzed tend to be cross-sectional information about symptom-based categories or dimensions of mental disorders. Cross-sectional data yield snapshots of individuals at one point in time, in one generation. Here we present intergenerational and developmental data that help to deconstruct "p." Our goal is to document why it is important to carry out research on "p," rather than on one mental disorder at a time.

First, we present evidence about assortative mating for mental disorders. We document that it is ubiquitous, and that cross-disorder assortative mating is common. Cross-disorder assortative mating means that individuals with a parent with a particular mental disorder will carry risk genes and psychosocial risks for more than that one disorder. Second, we present evidence that specific mental disorders run in families, but that risk is also transdiagnostic. That is, parents with a particular mental disorder do not only have offspring with the same disorder; they also have offspring with other disorders. Third, we present evidence that, across the life course, individuals experience many different mental disorders and shift between internalizing, externalizing, and/or thought disorders. As shown in Table 1, we document these points with diverse data sources, including nationwide registries involving multiple generations and millions of individuals, and covering tens-of-million person-years, as well as longitudinal research tracking individuals and families over many decades. The findings converge to provide an intergenerational and developmental perspective on why most mental disorders share so much in common, why it is so difficult to find disorder-specific causes of mental disorders, and "p."

With a novel intergenerational and developmental empirical foundation for understanding "p," we will (a) spell out recommendations to reshape measurement and design practices in research settings to advance etiological research; and (b) identify implications for assessment practices in clinical settings to deliver more effective treatments that can benefit the entire population, and vulnerable groups in particular. The goal of this article is to articulate these ideas as a set of tractable new directions for psychopathology research and clinical psychology.

Assortative Mating Is Widespread and Transdiagnostic

Children's starting points in life depend on the genes and environments they receive from their parents. Men and women who form

Table 1

Sources of Data Used in This Article to Study Assortative Mating, Intergenerational Links, and the Longitudinal Course of Mental Disorders

Data source and measures	Assortative mating		Intergenerational links		Longitudinal course, longitudinal birth cohort study
	Nationwide hospital records	Nationwide primary-care records	Nationwide hospital records	Nationwide primary-care records	
Study population	We used population-level administrative data from hospital records in Denmark, from 1970 until 2018. Our focal individuals were born in Denmark between 1960 and 1970 and resided in Denmark between 1980 and 2018, as identified in the Danish population registers (age at baseline = 10–20 years, age at end of observation = 48–58 years). For each focal individual, we identified all opposite-sex partners (through cohabitation or marriage) between 1980 and 2018 and chose the person with whom the individual was partnered the longest as their primary partner. This resulted in a study population of 762,613 individuals, 383,175 males and 379,438 females representing 497,910 unique male/female pairs, male age: *M*(*SD*) = 15.3(4.9) years; female age: *M*(*SD*) = 13.0(4.8) years in 1980.	We used population-level administrative data from primary-care practices in Norway, available from January 2006 until December 2019. Our focal individuals were born in Norway between February 1955 and January 1986 and were full-time residents in Norway from January 2006 until December 2019 or until they died, as identified in the Norwegian Population Register (age at baseline = 20–50 years, age at end of observation = 34–65 years). For each focal individual, we identified all opposite-sex partners (through cohabitation or marriage) between 2006 and 2019 and chose the person with whom the individual was partnered the longest as their primary partner. To be included, partners had to be full-time residents in Norway from January 2006 until December 2019 or until they died, but did not have to be born in Norway. This resulted in a study population of 1,587,470 individuals, 793,155 males and 794,315 females representing 844,673 unique male/female pairs, male age: *M* (*SD*) = 37.6 (10.1) years; female age: *M* (*SD*) = 35.0 (9.8) years in January 2006.	For the parent–offspring analysis, the study population consisted of 713,090 children (365,466 males, 347,624 females) born in Denmark between 1985 and 1995, who resided in Denmark between 1986 and 2018. These children were aged <0 to 1 at baseline and 23 to 33 years at the end of the observation period. We linked each focal child to their legal parents, as identified through population registers which run from 1980, and contain yearly information on all residents in Denmark by January 1 each year. On average we observed mothers from age 7.21 (*SD* = 5.45) in 1970 to age 56.21 (*SD* = 5.45) at the end of the observation period, and on average from age 10.11 (*SD* = 6.39) to age 59.11 (6.39) for fathers. The number of children per family averaged 1.47 (*SD* = 0.64); all analyses accounted for nesting of children within 485,552 unique mother/father pairs.	For the parent–offspring analysis, the study population consisted of 818,221 children (419,818 males, 398,403 females) born in Norway between January 2000 and December 2014, who were full-time residents in Norway from January 2006 until January 2019 or until they died. These children were aged <0 to 6 years at baseline and 5 to <20 years at the end of the observation period. We linked each focal child to their legal parents, as identified in the Norwegian Population Register. To be included, children had to have at least one parent who was alive and fully resident in Norway from January 2006 until January 2019, or until they died. Parents were not required to be born in Norway. On average we observed mothers from age 29.00 (*SD* = 6.53) to age 42.98 (*SD* = 6.53) at the end of the observation period, and on average from age 31.93 (*SD* = 7.33) to age 45.87 (*SD* = 7.32) for fathers. The number of children per family averaged 1.52 (*SD* = 0.70); all analyses accounted for nesting of children within 508,493 unique mother/father pairs.	The Dunedin Study is a longitudinal investigation of health and behavior in a complete birth cohort. |The 1,037 (535 male) participants were all individuals born between April 1972 and March 1973 in Dunedin, New Zealand, who participated in the first assessment at age 3 years, representing 91% of participants who were eligible based on residence in the province (Poulton et al., 2022). The cohort represented the full range of socioeconomic status on New Zealand's South Island and in adulthood matches the NZ National Health and Nutrition Survey on key health indicators (e.g., BMI, smoking, GP visits) and matches the NZ Census of citizens the same age on educational attainment. The cohort is primarily New Zealand European; 8.6% self-identified as having Māori ethnicity at age 45. Assessments were carried out at birth and ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26, 32, 38, and most recently, 45 years, when 94% of the 997 study members still alive took part. At each assessment, each study member is brought to the research unit for an assessment day including interviews and examinations.
Mental-health assessments	We linked the focal individuals and their partners to (a) The Danish Psychiatric Central Register, which contains information on all inpatient treatment at psychiatric hospitals and wards in Denmark from 1970 to 1994, and (b) The Danish National Patient Register, which contains information on all contacts at psychiatric hospitals, wards, and emergency rooms including both	We linked the focal individuals and their partners to their primary-care records. All residents of Norway are assigned a PCP. Access to specialist healthcare typically requires a referral from the PCP. Service is free for juveniles and highly subsidized for adults. To receive reimbursements, PCPs bill the Norwegian Health Economics Administration, sending at least one primary diagnosis or reason	We linked both parents and offspring to (a) The Danish Psychiatric Central Register, which contains information on all inpatient treatment at psychiatric hospital and wards in Denmark from 1970 to 1994, and (b) The Danish National Patient Register, which contains information on all contacts at psychiatric hospitals, wards and	We linked both parents and offspring to their primary-care records coded according ICPC-2. Both parents' and children's primary-care visits took place during a 14-year observation window, between 2006 and 2019. Among parents, we focused on the same 14 mental-health conditions that we reported about in the assortative mating analysis and that had a	Beginning at age 11 years, participants were interviewed about past-year symptoms of mental disorders. Interviews were conducted by health professionals. At ages 11, 13, and 15 years, interviews were performed with the Diagnostic Interview Schedule for Children (A. Costello et al., 1982) assessing the following disorders: externalizing disorders (i.e., attention-deficit/hyperactivity disorder and conduct disorder) and internalizing

(*table continues*)

Table 1 (*continued*)

Data source and measures	Assortative mating		Intergenerational links		Longitudinal course, longitudinal birth cohort study
	Nationwide hospital records	Nationwide primary-care records	Nationwide hospital records	Nationwide primary-care records	
	inpatient and outpatient contacts from 1995 and onwards. Focal individuals and their partners were classified as having a mental disorder if at any point between 1970 and 2018 they were registered with a diagnosis (primary or secondary) within the following diagnosis categories, according to *ICD-8* and *ICD-10*: Externalizing disorders (substance abuse, externalizing behavior in childhood or adolescence); internalizing disorders (neurotic (i.e., anxiety), mood, and eating disorders); thought disorders (schizophrenia, bipolar disorder, and OCD); and other disorders (personality disorders, pervasive developmental disorders) (Table 2). We constructed binary indicators for each mental disorder diagnosis category, which took the value one if the individual had a record of that diagnosis category at any point between 1970 and 2018. Table S1 in the online supplemental materials provides descriptive information about the individuals and mental disorders included in the analysis. Figure 1A provides information about the co-occurrence of different mental disorders within the same individuals; that is, the familiar phenomenon of psychiatric comorbidity.	for the visit. Thus, it is unlikely that visits to PCPs go unreported. Information is coded according to the ICPC-2 which is divided into 17 chapters representing health conditions in different body systems (see Caspi, Houts, Moffitt, et al., 2024). Focal individuals and their partners were classified as having a mental-health condition if they were registered with a code from ICPC-2 Chapter P-Psychological. We studied 14 mental-health conditions that had at least a 1% prevalence rate in the current study population: Substance abuse, ADHD, depression, acute stress reaction, anxiety, phobia, PTSD, somatization, psychosis, mental disorders not otherwise specified, sleep disorder, sexual concerns, personality disorder, and suicide/suicide attempt (Table 3). We constructed binary indicators for each condition that took the value of one if the individual had a PCP visit with that code at any point between 2006 and 2019. Table S2 in the online supplemental materials provides descriptive information about the individuals and mental-health conditions included in the analysis. Figure 2A provides information about the co-occurrence of different mental-health conditions in the same individuals.	emergency rooms including both inpatient and outpatient contacts from 1995 and onwards. Since the psychiatric registers start in 1970, for offspring, all of whom were born after 1985, we observed their entire lifetime of diagnoses, ranging from ages 0 to 33 years. Offspring and their parents were classified as having a mental disorder if they were registered with a diagnosis (primary or secondary) according to *ICD-8* and *ICD-10* (see Table 2). Table S3 in the online supplemental materials provides descriptive information about the individuals and the mental disorders included in the analysis.	prevalence across the 14-year observation window greater than 1%. Among the offspring, we focused on these same 14 conditions plus three additional mental-health conditions that had a prevalence greater than 1% in the population of children (continence issues; developmental delay/learning problems; stammering/stuttering/tic; see Table 3). Table S4 in the online supplemental materials provides descriptive information about the individuals and the mental-health conditions included in the parent–offspring resemblance analysis.	disorders (i.e., depression, anxiety, and fears [including separation anxiety, overanxiety, social phobia, and simple phobia]). At ages 18, 21, 26, 32, 38, and 45 years, interviews were performed with the Diagnostic Interview Schedule (Robins et al., 1995; Robins et al., 1981) assessing the following disorders: externalizing disorders (i.e., attention-deficit/hyperactivity disorder [at ages 18, 38, 45], conduct disorder, alcohol dependence, cannabis dependence, other drug dependence [at ages 26, 32, 38, 45], and tobacco dependence), internalizing disorders (i.e., depression, generalized anxiety disorder, fears [including social phobia, simple phobia, agoraphobia, and panic disorder], posttraumatic stress disorder [at ages 26, 32, 38, 45], and eating disorders [including bulimia and anorexia at ages 18, 21, 26]), and thought disorders (i.e., obsessive–compulsive disorder, mania and schizophrenia [the latter two at ages 21, 26, 32, 38, 45]). Diagnoses were based on symptom algorithms and impairment ratings, but also incorporated information including standardized teacher, parent, and informant reports as developmentally appropriate; psychiatrists' review of interviewers' detailed case notes; pharmaceutical review; and staff ratings of symptoms observed (M. H. Meier et al., 2014). Up to age 15 years, diagnoses were made according to *DSM-III;* at ages 18 and 21 years, according to *DSM-III-R*; at ages 26, 32, and 38 years, according to *DSM-IV*; and at age 45 years, according to *DSM-5* with the exception of substance-dependence disorders, which were diagnosed according to *DSM-IV* to retain the dependence versus abuse distinction removed by *DSM-5*.

Note. NZ = New Zealand; BMI = body mass index; GP = general practitioner; *ICD* = *International Classification of Diseases*; OCD = obsessive compulsive disorder; PCP = primary-care physician; ICPC = International Classification of Primary Care; ADHD = attention deficit hyperactivity disorder; PTSD = posttraumatic stress disorder; DSM = *Diagnostic and Statistical Manual of Mental Disorders*.

pair bonds resemble each other on practically every anthropometric, social, medical, and psychological attribute (Horwitz et al., 2023). Such assortative mating—the tendency of people to mate with others who resemble them more than would be expected by chance—has also been observed for many different mental disorders, including internalizing, externalizing, and thought disorders, as well as neurodevelopmental disorders (Merikangas, 1982). Even more remarkable is evidence of nonrandom mating across the spectrum of mental disorders (Nordsletten et al., 2016). This makes children's starting points in life more unequal than they would be without assortative mating.

Assortative mating has been of interest to geneticists because it may lead to children inheriting from both the mother and the father genes associated with the traits underlying partner choice. This can create correlations between genes associated with different traits (Border et al., 2022; Peyrot et al., 2016; Torvik et al., 2022). Assortative mating has been of interest to social scientists because it reinforces and exacerbates wealth, social, and health inequalities between families (Milanovic, 2019). Assortative mating should be of interest to mental-health researchers because it can lead to offspring being exposed to larger variation in the genetic and environmental risks for different mental disorders.

Here we use nationwide data from Denmark and Norway to evaluate the scope of assortative mating within the population. In both nations, we identify all cohabiting and married opposite-sex couples in the population, and ask: How much do they resemble one another in terms of their mental health?

Assortative Mating for Mental Disorders: I. Evidence From Nationwide Hospital Records

We used population-level administrative data from Denmark (Table 1) to analyze hospital-treatment data and examine mental disorders that have come to the attention of inpatient and outpatient clinics (Table 2). To estimate the strength and extent of assortative mating for mental disorders, we examined associations between males' and females' diagnoses. The analysis does not distinguish whether the mental disorder was experienced before, during, or after cohabitation/marriage, and it does not establish whether the male's disorder was experienced before or after the female's disorder. The analysis simply establishes whether people who experience a hospital-treated mental disorder between childhood and their 40s/50s form unions with mates who are also more likely to experience a mental disorder during their own lifetime. We assessed assortative mating by calculating *OR*s between partners; averages across subsets of *OR*s were calculated via random effects meta-analysis.

Over a 38-year observation period of 762,613 people between the ages of 10–58, 10% were hospital-treated for a mental disorder as in- or outpatients. Both women and men diagnosed with a mental disorder were more likely than individuals without a hospital-treated diagnosis to partner with individuals treated for a mental disorder in hospital settings themselves (17% vs. 8% among women and 23% vs. 11% among men; $OR = 2.54$, 95% confidence interval [CI]: [2.39, 2.71]). Moreover, individuals who had been diagnosed with multiple mental disorders were also more likely to partner with mates who had been diagnosed with multiple disorders (incident rate ratio [IRR] = 1.45, 95% CI = [1.43, 1.46]). Critically, assortative mating for mental disorders was not confined to particular combinations of disorders but was evident across the vast majority of mental-disorder pairings: 93% (80 out of 86) of the elements in the heatmap in Figure 1B are statistically significant. Four findings stand out.

First, the elements along the diagonals show that individuals were more likely to partner with others who had an experience with the same mental disorder as they did. This was true for all disorders assessed. The average same-disorder assortative mating coefficient was $OR = 4.33$, 95% CI = [2.50, 7.50]. On average, same-disorder assortative mating was more notable than cross-disorder assortative mating (Table S5 in the online supplemental materials), and this was most notable for schizophrenia and externalizing conditions. For example, women with a hospital-treated substance-use condition (1.9% of the study population) were more likely than women without a substance-use condition to be partnered to men who also had a substance-use condition (16% vs. 3%, $OR = 6.2$, 95% CI = [5.9, 6.6]).

Second, the elements in the diagonal boxes show that individuals who experienced a specific disorder within a particular disorder family (e.g., an externalizing, internalizing, or thought disorder)

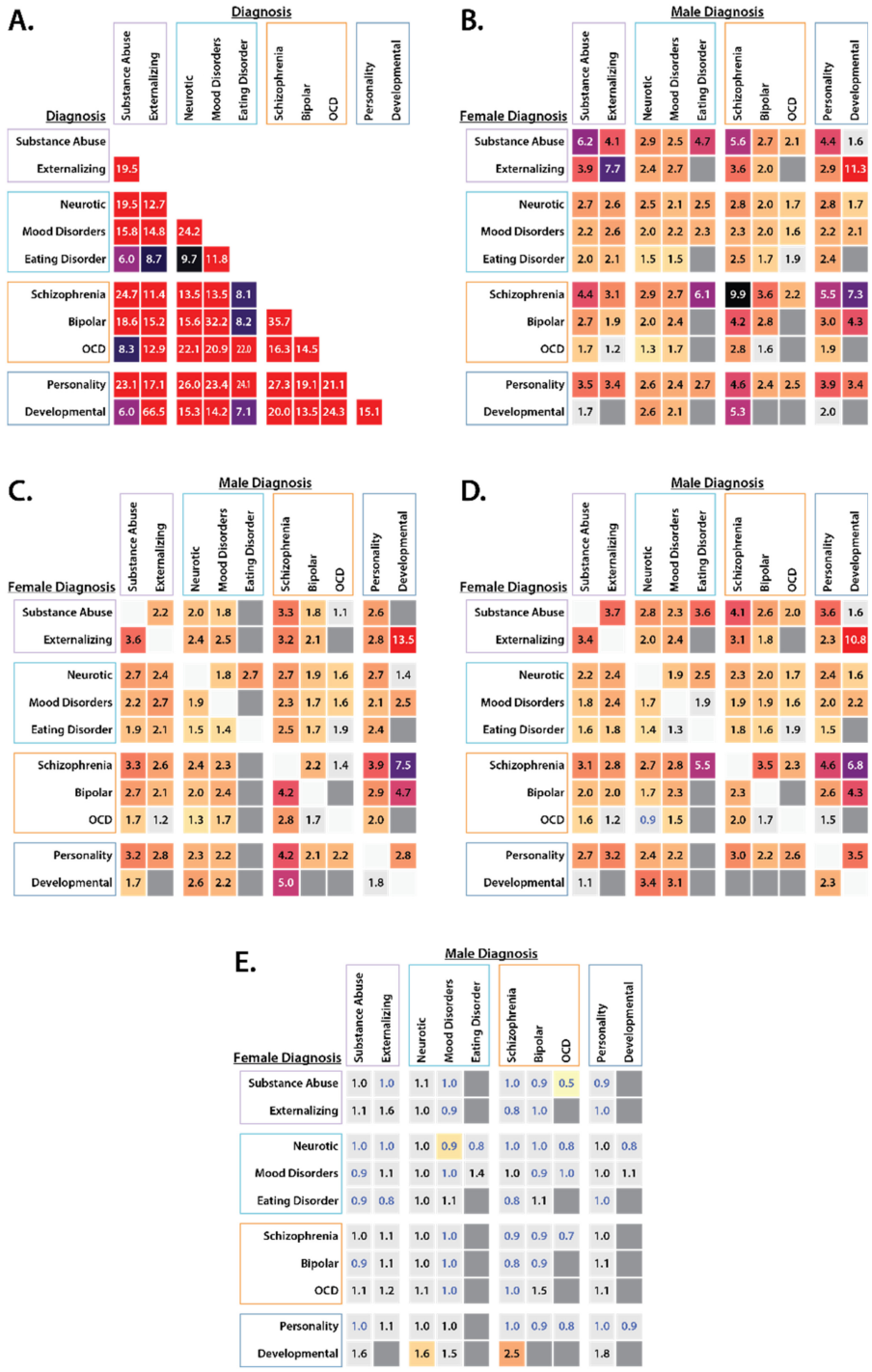
Table 2
Classification of Mental Disorders Between 1970 and 2018 in Nationwide Hospital Data in Denmark

Diagnosis category	*ICD-10*	*ICD-8*
Externalizing disorders		
Mental and behavioral disorders due to substance abuse	F10–F19	291.x9, 294.39, 303.x9, 303.20,303.28, 303.90, 304.x9
Externalizing behavior in childhood or adolescence	F90–F92	308.1, 308.2, 308.3
Internalizing disorders		
Neurotic disorders	F40–F41 + F43–F48	300.x9 (excl. 300.49 + 300.39), 305.x9, 305.68, 307.99
Mood disorders	F32–F39	296.x9 (excl. 296.89 + 296.19 & 296.39), 298.09, 300.49, 301.19
Eating disorders	F50	305.60, 305.50, 306.58, 306.59
Thought disorders		
Schizophrenia and related disorders	F20–F29	295.x9, 296.89, 297.x9, 298.29–298.99, 299.04, 299.05, 299.09, 301.83
Bipolar	F30–F31	296.19, 296.39, 298.19,
OCD	F42	300.39
Other disorders		
Personality disorders	F60	301.x9 (excl. 301.19) 301.80, 301.81, 301.82, 301.84
Pervasive developmental disorders	F84	299.00, 299.01, 299.02, 299.03

Note. For both the assortative mating analysis and the intergenerational transmission analysis, individuals were classified as having a mental disorder if at any point between 1970 and 2018 they were registered with a diagnosis (primary or secondary), coded according to *ICD-8* and *ICD-10*. *ICD = International Classification of Diseases*; OCD = obsessive compulsive disorder.

Figure 1

Assortative Mating for Mental Disorders Observed in Nationwide Hospital Data (N = 497,910 Couples)



Note. Panel A provides information about the co-occurrence of different mental disorders in the same individuals; that is, the phenomenon of psychiatric comorbidity. Panel B shows widespread assortative mating for mental disorders. Cross-disorder assortative mating was not simply a function of comorbidity within each partner; that is, men (Panel C) and women (Panel D) with a specific mental disorder were more likely to partner with mates who had a different mental disorder, even when they themselves did not have the co-occurring condition. Panel E shows concordance for mental disorders between two partners chosen at

(*figure continues*)

were more likely to partner with others who experienced a different disorder within that same disorder family (average externalizing: $OR = 4.00$, 95% CI = [3.16, 5.07]; average internalizing: $OR = 1.82$, 95% CI = [1.48, 2.24]; average thought disorder: $OR = 2.93$, 95% CI = [1.89, 4.55]). For example, men who experienced mood disorders were more likely to partner with women who experienced anxiety disorders (14% vs. 8%, $OR = 2.1$, 95% CI = [2.0, 2.2]) and women who experienced eating disorders were more likely to partner with men who experienced anxiety disorders (7% vs. 5%, $OR = 1.5$, 95% CI [1.3, 1.7]).

Third, the off-diagonal elements show extensive cross-disorder assortative mating between different families of disorders. Individuals who experienced any particular mental disorder were more likely to partner with others who experienced any number of different mental disorders. The average cross-disorder assortative mating coefficient was $OR = 2.64$, 95% CI = [2.41, 2.90]. On average, there was no evidence that cross-disorder assortative mating within a disorder family was greater than cross-disorder assortative mating across disorder families (Table S5 in the online supplemental materials). For example, women with a substance-use condition were more likely than women without a substance-use condition to be partnered to men who were hospital-treated for an externalizing (nonsubstance use) disorder (1.5% vs. 0.4%, $OR = 4.1$, 95% CI [3.4, 4.9]) and to men who were hospital-treated for schizophrenia (5% vs. 1%, $OR = 5.6$, 95% CI = [5.0, 6.1]).

Fourth, given pervasive comorbidity at the individual level (see Figure 1A), we tested whether cross-disorder assortative mating was simply a function of comorbidity within each partner. It was not. Men (Figure 1C; average $OR = 2.36$, 95% CI = [2.18, 2.56]) and women (Figure 1D; average $OR = 2.31$, 95% CI = [2.13, 2.51]) with a specific mental disorder were more likely to partner with mates who had a different mental disorder, even when they themselves did not have the co-occurring condition or when their partner did not have the specific mental disorder. If people mated randomly, the landscape would look very different. The heatmap in Figure 1E shows concordance for mental disorders between randomly selected opposite-sex partners from the population of partners between 1980 and 2018 (average $OR = 1.01$, 95% CI = [0.98, 1.03]) Actual couples who chose each other are clearly more concordant for mental disorders than two partners assigned to each other at random from the population.

It is possible that these assortative mating estimates are inflated because we relied on hospital-treatment data. Patients with multimorbid conditions are more likely to be hospital-treated than patients with only one condition, and hospital-treated patients also tend to present with more severe conditions. Studying men and women who have been hospital-treated for a mental health condition runs the risk of exaggerating the extent to which they partner with mates who experience other disorders. We thus broadened our analysis of assortative mating by turning to a different data source: all primary-care records in the health system of an entire nation where cost barriers do not generate bias in the subset of unwell individuals who seek care.

Assortative Mating for Mental-Health Conditions: II. Evidence From Nationwide Primary-Care Records

We used population-level administrative data from Norway (Table 1) to analyze mental-health conditions that have come to the attention of primary-care physicians (Table 3). While the rates of mental-health conditions in primary-care settings exceed the rates of hospital-treated mental disorders, the assortative mating findings in nationwide primary-care data corroborate those from nationwide hospital-treatment data. Over a 14-year observation period of 1,587,470 people between the ages of 20–64, 56% were seen in primary-care settings with a mental-health condition. Individuals with a mental-health condition were more likely than individuals without a mental-health condition to be partnered to an individual who had also been diagnosed with a mental-health condition (54% vs. 36% among women and 74% vs. 58% among men; $OR = 2.11$, 95% CI = [2.09, 2.13]). Moreover, individuals who experienced multiple different mental-health conditions were also more likely to partner with individuals who experienced multiple different mental-health conditions (IRR = 1.21, 95% CI = [1.16, 1.25]).

The heatmap in Figure 2B indicates that assortative mating occurred across the spectrum of mental-health conditions. First, the diagonals show that, on average, individuals were more likely to partner with others who had an experience with the same mental-health condition as they did (average $OR = 2.92$, 95% CI = [2.14, 3.99]). On average, same-disorder assortative mating was more notable than cross-disorder assortative mating (Table S5 in the online supplemental materials), and this was mostly the case for externalizing conditions. For example, women with a substance-use condition (4.8% of the study population) were more likely than women without a substance-use condition to be partnered to men who also had a substance-use condition (25% vs. 6%, $OR = 5.7$, 95% CI = [5.6, 5.8]). Second, the diagonal boxes show that, on average, individuals who experienced a specific mental-health condition within a particular disorder family were more likely to partner with others who experienced a different mental-health condition within that same disorder family (average externalizing: 3.82, 95% CI = [2.12, 6.91]; average internalizing: 1.69, 95% CI = [1.59, 1.79]). Third, the off-diagonal elements show significant assortative mating across families of different mental-health conditions. The average cross-family assortative mating coefficient was 2.06, 95% CI = [1.95, 2.17]. On average, there was no evidence that cross-disorder assortative mating within a disorder family was markedly greater than cross-disorder assortative mating across disorder families (Table S5 in the online supplemental materials). For example, women with a substance-use condition were more likely than women without a substance-use condition to be partnered to men who experienced ADHD (6% vs.

Figure 1 (*continued*)

random from the population; actual partners are clearly more concordant. Entries in the figure are odds ratios. Blue (light gray) text indicates that the odds ratio is <1.0. Light gray cells indicate that the confidence interval included 1. Dark gray cells without text indicate that there were insufficient observations to calculate associations. This is mostly due to sex differences in base rates of different conditions; for example, there are few men with a diagnosis of eating disorder and few women with a diagnosis of pervasive developmental disorder, resulting in few partnerships with these overlapping conditions. OCD = obsessive compulsive disorder. See the online article for the color version of this figure.

Table 3
Classification of Mental-Health Conditions Between 2006 and 2019 in Nationwide Primary-Care Data in Norway

Diagnosis category	ICPC-2
Externalizing disorders	
Substance abuse	P15–P19
ADHD	P81
Child/adolescent behavior symptom/complaint[a]	P22–P23
Internalizing disorders	
Depression	P03, P76
Acute stress reaction	P02
Anxiety disorder	P01, P74
Phobia/compulsive disorder	P79
Posttraumatic stress disorder	P82
Somatization disorder	P75
Thought disorders	
Psychosis	P71–P73, P98
Other disorders	
Psychological disorders NOS	P29, P99
Sleep disturbance	P06
Sexual concern	P07–P09
Personality disorder	P80
Suicide/suicide attempt	P77
Continence issues[a]	P12–P13
Developmental delay/learning problems[a]	P24, P85
Stammering/stuttering/Tic[a]	P10

Note. For the assortative mating analysis, we studied 14 mental-health conditions, coded according to the ICPC-2 that had at least a 1% prevalence rate among adults, aged 20–50 years. For the intergenerational analysis, we studied the same 14 mental-health conditions in both parents and offspring, plus four additional mental-health conditions that had a prevalence greater than 1% in the population of children. ICPC = International Classification of Primary Care; ADHD = attention deficit hyperactivity disorder; NOS = not otherwise specified.
[a] Only studied in children.

2%, $OR = 4.0$, 95% CI = [3.8, 4.2]) and to men who experienced psychosis (5% vs. 2%, $OR = 3.2$, 95% CI = [3.1, 3.6]). Fourth, although comorbidity was pervasive within individuals (see Figure 2A), cross-condition assortative mating was not simply a function of comorbidity within each partner (Figure 2C and 2D; average $OR_{\text{no male comorbidity}} = 1.87$, 95% CI = [1.79, 1.95]; average $OR_{\text{no female comorbidity}} = 1.88$, 95% CI = [1.80, 1.96]). Actual couples who chose each other are clearly more concordant for mental disorders than two partners assigned to each other at random from the population (average $OR = 1.00$, 95% CI = [1.00, 1.02]) (Figure 2E).

Summary

Nationwide evidence from two countries, relying on complementary data sources, reveals widespread assortative mating across pairwise combinations of practically every mental-health condition. It is not simply that men and women with a particular mental-health condition are more likely to mate with partners who experience the same specific condition; they also have partners who are at increased risk of experiencing practically every mental-health condition.

Assortative mating for mental disorders can come about for multiple reasons. First, assortative mating may reflect phenotypic assortment. Here, pair bonds are formed based on an observable phenotype. People may choose mates based on physical traits, cultural preferences, and personality traits, as well as mental health itself. Even if similarity between partners is not the direct result of assorting on a specific mental health condition, it may be an indirect result of assorting on phenotypes that are strongly linked to multiple, different mental health conditions (e.g., personality traits of high neuroticism, low agreeableness, and low conscientiousness). Second, assortative mating may reflect social homogamy. Here, partner similarity comes about as a result of assortment within subgroups of the population that have different probabilities of having the studied traits. There are strong incentives and constraints for people to choose mates from similar cultural, social, and geodemographic backgrounds, and this may result in assortment on correlated mental-health conditions. In fact, assortative mating may occur without any direct preferences on the part of individuals (Xie et al., 2015). Third, observed similarity between partners may reflect the convergence of phenotypes over time rather than initial assortment. Here, couples come to increasingly resemble each other with time, whether due to mutual influence or to shared circumstances. This phenomenon may occur for some disorders more than others. For example, there is evidence that phenotypic convergence is more pronounced for substance use and misuse than it is for depression (Torvik et al., 2024). Fourth, partner similarity may emerge as a result of behavioral contagion, in which partners copy each other's behavior. This phenomenon may occur for many risk-taking behaviors as well as suicide (Suzuki et al., 2016). Fifth, it is possible that cross-disorder assortment may emerge from a process by which partners initially mate assortatively on the same disorder, and that disorder then has causal effects on risk for other, different disorders (e.g., partners could mate assortatively for depression, and depression may then causally increase their mate's risk of other disorders; Sjaarda & Kutalik, 2023).

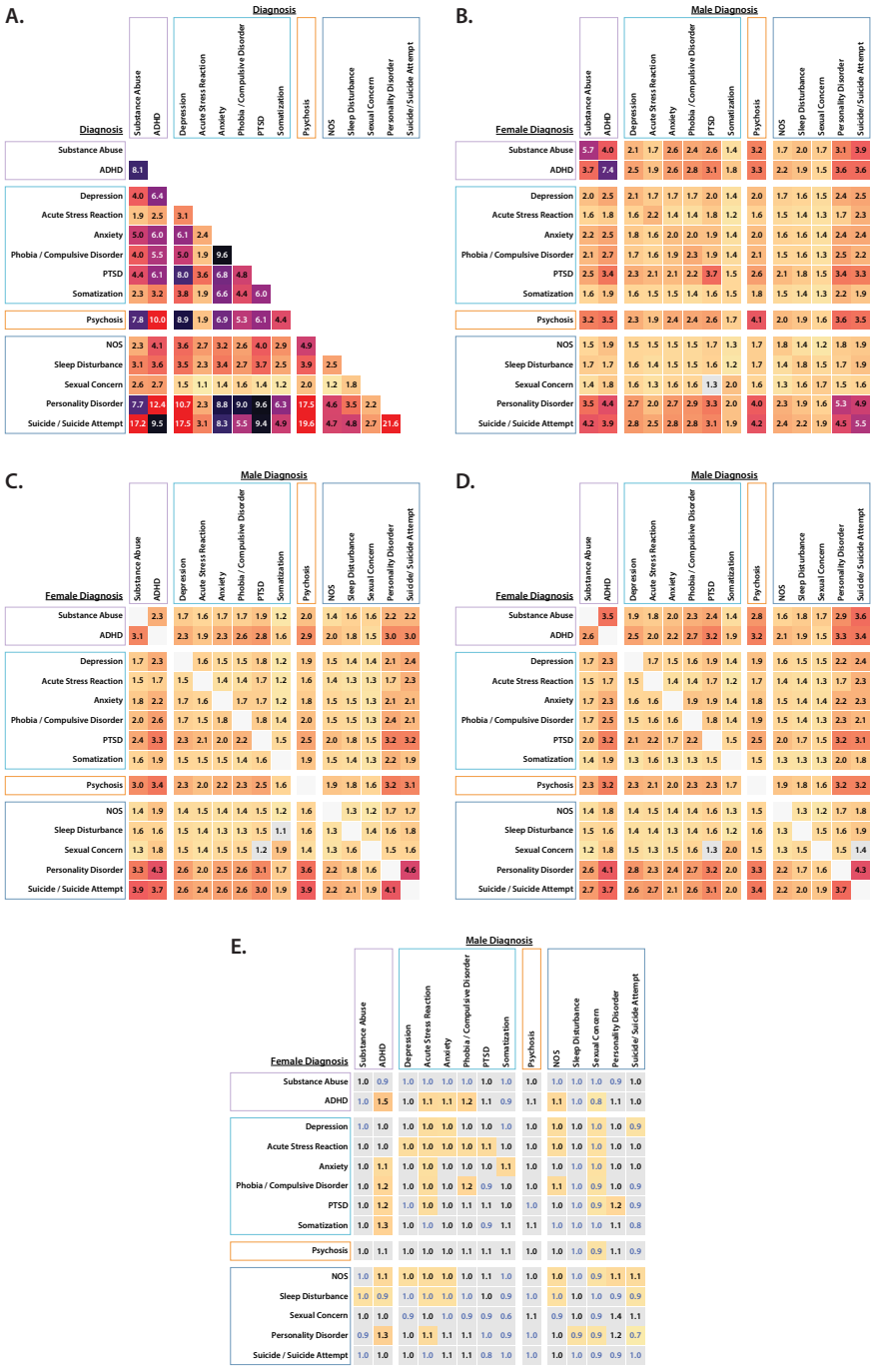
However it comes about, pervasive assortative mating across all mental-health conditions means that individuals experiencing a mental disorder are more likely to cohabit and procreate with individuals who also experience mental disorders. These individuals do not just experience the same condition, but also many other conditions. The intragenerational and intergenerational implications should not escape attention. Within a generation, pervasive assortative mating has implications for understanding the life histories of individuals with mental disorders. Cognate research on assortative mating and adult personality development suggests that assortative mating for mental disorders is likely to give rise to persistence of mental health problems during a person's life course (Caspi & Herbener, 1990). Across generations, there are implications for understanding the transmission of risk. Whether due to genetic transmission, environmental transmission, or most likely both, cross-trait assortative mating gives rise to a dynamic of transdiagnostic risk in which the offspring of parents who have different mental disorders have an increased propensity to experience a broad range of mental-health problems themselves.

The Familial Risk of Mental Disorders Is Transdiagnostic

What can I expect given that my mother has disorder X? What is the likelihood that my son will develop disorder Y if I had the disorder as a child? Researchers are keen to develop prognostic algorithms that can forecast such risk; clinicians are dedicated to providing guidance about these questions; and parents and children want answers (V. Costello, 2012). Much of the evidence bearing on this question comes from high-risk studies, which follow children in families with an affected parent, and from hospital registry studies.

Figure 2

Assortative Mating for Mental Disorders Observed in Nationwide Primary-Care Data (N = 809,822 Couples)



Note. Panel A provides information about the co-occurrence of different mental disorders in the same individuals; that is, the phenomenon of psychiatric comorbidity. Panel B shows widespread assortative mating for mental disorders. Cross-disorder assortative mating was not simply a function of comorbidity within each partner; that is, men (Panel C) and women (Panel D) with a specific mental disorder were more likely to partner with mates who had a different mental disorder, even when they themselves did not have the co-occurring condition. Panel E shows concordance for mental disorders between two partners chosen at random from the population; actual partners are clearly more concordant. Entries in the figure are odds ratios. Blue (light gray) text indicates that the odds ratio is <1.0. Light gray cells indicate that the confidence interval included 1. ADHD = attention deficit hyperactivity disorder; PTSD = posttraumatic stress disorder; NOS = not otherwise specified. See the online article for the color version of this figure.

Both designs suggest that mental disorders not only run in families, but also that the risk of mental disorders in offspring of parents with a mental disorder is transdiagnostic (Uher et al., 2023; Zhou et al., 2024). Here, we use nationwide hospital-treatment and primary-care data to evaluate the scope of parent–child resemblance for mental disorder in the population. We linked parents to their offspring, and ask: How much do they resemble one another across a broad range of disorders?

Intergenerational Links: I. Evidence From Nationwide Hospital Records

We used population-level administrative data from Denmark (Table 1) to analyze hospital-treatment data and examine mental disorders that have come to the attention of inpatient and outpatient clinics (Table 2). For our primary parent–offspring resemblance analysis, we examined associations between children's diagnoses and a joint measure of parental diagnoses, which took the value one if either of the parents had received the diagnosis. Our analysis does not distinguish whether parents and offspring are biologically related. Additionally, as we focus on legal parents, we do not include step-parents or foster parents in our analysis (unless they are registered as legal parents). The analysis also does not distinguish whether parental mental disorder was experienced before, during, or after the focal offspring lived with the parent. The analysis simply establishes whether parents who experience a mental disorder in their lifetime are more likely to have children who experience a mental disorder. We assessed parent–child resemblance by calculating *OR*s between parents and children; averages across subsets of *OR*s were calculated via random effects meta-analysis.

Over a 33-year observation period of 713,090 young people between birth to 33 years, 15.3% were hospital-treated for a mental disorder as in- or outpatients. Offspring who received hospital treatment for a mental disorder were more likely than those who did not to have at least one parent who received hospital treatment for a mental disorder themselves (33% vs. 17%, $OR = 2.43$, 95% CI = [2.39, 2.46]). If both parents had a disorder, the risk of offspring disorder was greater than if only one parent had a disorder (two parents vs. 0 parents: $OR = 3.91$, 95% CI = [3.40, 4.50]; two parents vs. one parent: $OR = 1.72$, 95% CI = [1.62, 1.84]; one parent vs. zero parents: $OR = 2.27$, 95% CI = [2.19, 2.35]). Moreover, the more disorders parents had, the more disorders their offspring were likely to have (IRR = 1.43, 95% CI = [1.42, 1.44]).

The heatmap in Figure 3A shows associations of parents and their offspring for different mental disorders. Associations were similar when we analyzed mothers and fathers separately (Figures S1 and S2 in the online supplemental materials), although mother–offspring associations tended to be stronger. Four findings stand out. First, the diagonal elements in Figure 3A show that parents and children were likely to be concordant for the same mental disorders. For example, offspring with a mood disorder were more likely than offspring without a mood disorder to have parents who were also hospital-treated for a mood disorder (16% vs. 8%, $OR = 2.2$, 95% CI = [2.2, 2.3]). Although same-disorder resemblance was true for all disorders assessed, it was especially pronounced for pervasive developmental disorders, externalizing behavior in childhood or adolescence, and bipolar disorder. The average same-disorder parent–offspring resemblance coefficient was 4.57 (95% CI = [2.89, 7.21]), and on average, same-disorder parent–offspring associations were stronger than cross-disorder parent–offspring associations (Table S5 in the online supplemental materials).

Second, parents who experienced a specific disorder within a particular disorder family (e.g., an externalizing, internalizing, or thought disorder) were more likely to have offspring who experienced a different disorder within that same disorder family (average externalizing: $OR = 3.35$, 95% CI = [2.11, 5.31]; average internalizing: $OR = 1.87$, 95% CI = [1.52, 2.30]; average thought disorder: $OR = 2.13$, 95% CI = [1.70, 2.66]). For example, parents with a history of mood disorders were more likely to have offspring who experienced neurotic disorders and eating disorders.

Third, the off-diagonal elements show extensive cross-disorder parent–offspring resemblance across families of different disorders. Parents who experienced any particular disorder were more likely to have offspring who experienced any number of different disorders. For example, parents with a history of mood disorders were more likely to have offspring who experienced externalizing disorders, thought disorders, pervasive developmental disorders, and personality disorders. The average cross-family parent–offspring resemblance coefficient was 2.22, 95% CI = [2.11, 2.34]. On average, there was no evidence that parent–offspring resemblance within a disorder family was greater than parent–offspring resemblance across disorder families (Table S5 in the online supplemental materials). For example, offspring with a mood disorder were more likely than offspring without a mood disorder to have a parent who was hospital-treated for a neurotic disorder (21% vs. 12%, $OR = 2.0$, 95% CI = [1.9, 2.0]) and to have a parent who was hospital-treated for a substance-use disorder (10% vs. 6%, $OR = 1.9$, 95% CI = [1.8, 2.0]).

Fourth, the risk for mental disorders among offspring of parents with a mental disorder extended to disorders not present among parents (average $OR = 1.99$, 95% CI = CI = [1.91, 2.09]) (Figure 3B). That is, parents with a specific mental disorder were more likely to have offspring who had different mental disorders, even when the parents themselves did not have the co-occurring condition. If parents and children did not share genes or rearing environments, the landscape would look very different. The heatmap in Figure 3C shows the resemblance between focal children and randomly matched parents (average $OR = 1.01$, 95% CI = [1.00, 1.02]). Actual parent–child pairs are clearly more concordant for mental disorders than randomly assigned sets of parents and children who did not share genes and/or family environments.

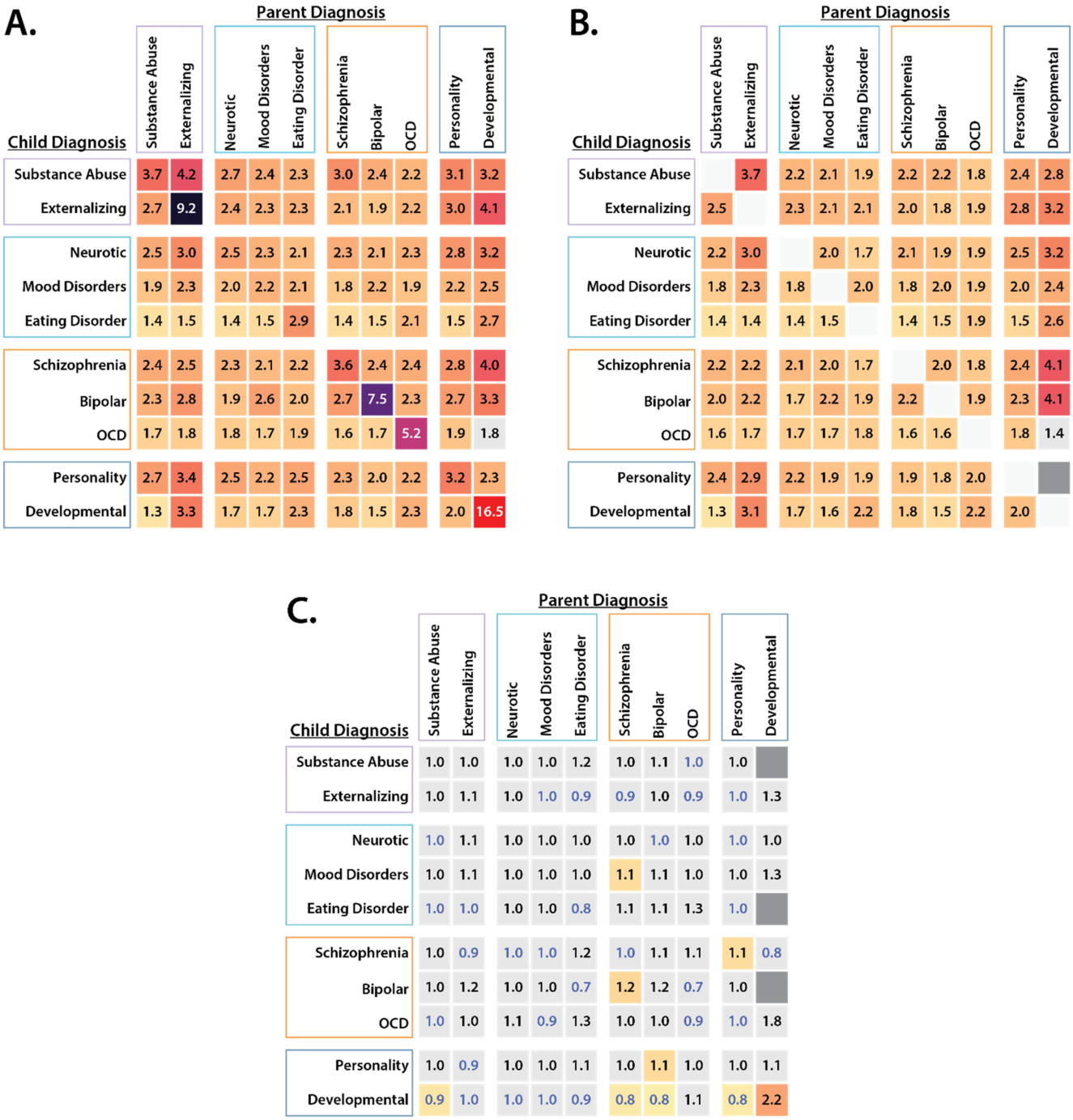
As studies that rely on hospital-treatment data as well as high-risk studies include individuals who present with more severe, comorbid conditions, studying the offspring of these individuals may inflate estimates of intergenerational transmission. We therefore broadened our analysis by turning to primary-care records.

Intergenerational Links: II. Evidence From Nationwide Primary-Care Records

We used population-level administrative data from Norway (Table 1) to analyze mental-health conditions that have come to the attention of primary-care physicians (Table 3). The findings corroborate those derived from nationwide hospital-treatment data. Over a 14-year observation period of 818,221 young people between birth and 20 years of age, 25.6% were seen in primary-care settings with a mental-health condition. These young people were more likely than young people without a mental-health condition to

Figure 3

Parent–Offspring Associations for Mental Disorders Observed in Nationwide Hospital Data (N = 713,090 Parent/Child Pairs)



Note. Panel A shows the odds ratios between parents' disorders and their offspring's disorders. We examined a joint measure of parental diagnoses, which took the value of 1 if either parent had received a diagnosis. Panel B shows that the risk of mental disorders to offspring of parents with a mental disorder is transdiagnostic and extends to disorders not present among parents (i.e., parents with a specific mental disorder were more likely to have offspring who had different mental disorders, even when the parents themselves did not have the offspring's co-occurring condition). Panel C shows odds ratios between parents' disorders and randomly matched offspring's disorders. Blue (light gray) text indicates that the odds ratio is <1.0. Light gray cells indicate that the confidence interval included 1. Dark gray cells without text indicate that there were insufficient observations to calculate associations. OCD = obsessive compulsive disorder. See the online article for the color version of this figure.

have at least one parent who was seen in primary-care settings for a mental-health condition themselves (86% vs. 73%, $OR = 2.31$, 95% CI = [2.28, 2.34]). If both parents had a disorder, the risk of offspring disorder was greater than if only one parent had a disorder (two parents vs. zero parents: $OR = 3.03$, 95% CI = [2.99, 3.08]; two parents vs. one parent: $OR = 1.71$, 95% CI = [1.69, 1.73]; one parent vs. zero parents: $OR = 1.77$, 95% CI = [1.75, 1.80]). Moreover, the more types of disorders parents had, the more types of disorders their offspring were likely to have (IRR = 1.25, 95% CI = [1.25, 1.25]).

The heatmap in Figure 4A indicates that parents diagnosed with a specific mental-health condition confer trans-disorder risk to their offspring, and the associations were similar when we analyzed mothers and fathers separately (Figures S3 and S4 in the online supplemental materials). First, the diagonals show that parents and children were likely to be concordant for the same mental-health condition across all mental-health conditions assessed (average $OR = 3.11$, 95% CI = [2.39, 4.04]). For example, offspring with depression were more likely than offspring without depression to have parents who also had depression (64% vs. 42%, $OR = 2.4$, 95% CI = [2.3, 2.4]). On average, same-disorder parent–offspring associations were stronger than cross-disorder parent–offspring associations (Table S5 in the online supplemental materials). Second, the diagonal boxes show that, on average, parents who experienced a mental-health condition within a particular disorder family (e.g., an externalizing or internalizing disorder) were more likely to have offspring who experienced a different mental-health condition within that same disorder family (average externalizing: 2.44, 95% CI = [1.85, 3.21]; average internalizing: 1.91, 95% CI = [1.75, 2.08]). Third, the off-diagonal elements show extensive parent–offspring resemblance across families of different disorders (average $OR = 1.87$, 95% CI = [1.80, 1.93]). On average, there was no evidence that cross-disorder parent–offspring resemblance within a disorder family was greater than cross-disorder parent–offspring resemblance across disorder families (Table S5 in the online supplemental materials). For example, offspring with depression were more likely than offspring without depression to have a parent who experienced anxiety (33% vs. 22%, $OR = 1.7$, 95% CI = [1.7, 1.7]) and to have a parent who experienced a substance-use condition (14% vs. 8%, $OR = 1.8$, 95% CI = [1.8, 1.9]). Fourth, the risk of mental disorders to offspring of parents with a mental disorder extended to disorders not present among parents (Figure 4B). That is, parents with a specific mental disorder were more likely to have offspring who had different mental disorders, even when the parents themselves did not have the co-occurring condition (average $OR = 1.91$, 95% CI = [1.84, 1.97]). The heatmap in Figure 4C shows resemblance between focal children and randomly matched parents. Actual parent–child pairs are clearly more concordant for mental-health problems than randomly assigned sets of parents and children who did not share genes and/or family environments (average $OR = 1.00$, 95% CI = [1.00, 1.00]).

Summary

Nationwide evidence from two countries, relying on complementary data sources, reveals that virtually every parental mental-health condition—whether on the part of mothers or fathers—is associated with increased odds that offspring will have any mental-health condition. The overriding message from our analysis of parent–offspring resemblance is not simply that parents and children resemble each other, but that the resemblance is transdiagnostic (Raballo et al., 2021).

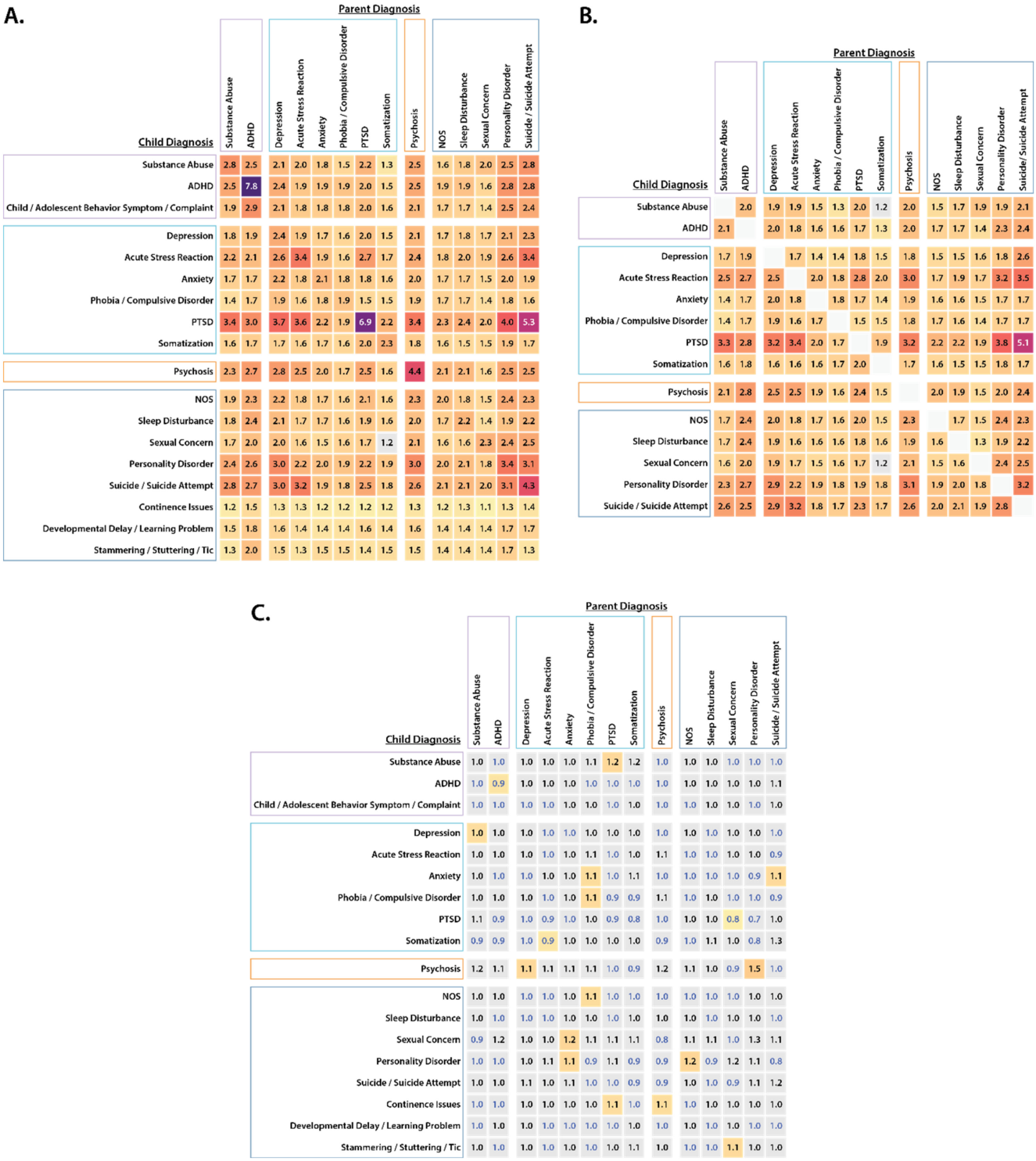
Associations between parental psychopathology and offspring psychopathology could emerge for several reasons. First, these may reflect genetic transmission of vulnerability to mental disorders. Second, and specific to maternal transmission, these associations may reflect sequelae of effects of maternal mental-health problems on fetal development (Wu et al., 2024). Third, associations could emerge as a result of parenting practices, ranging from compromised health knowledge and practices, to lack of warmth and hostility (expressed emotion), to maltreatment (Nevriana et al., 2024). Fourth, these associations may reflect stressors experienced by parents with mental disorders and their children, such as financial strain, housing instability, family disruption, and food insecurity (e.g., Keen et al., 2023; Melchior et al., 2009).

There are limitations to registry data. Affected parents may be more likely to seek services for their children. Offspring of affected parents may be more likely to make contact with health authorities because their parents have already had contact with said authorities (DuPont-Reyes et al., 2024). However, similar findings about parent–offspring resemblance have been reported in epidemiological surveys that have linked respondents' reports about their mental health with their family-history information (McLaughlin et al., 2012). We also cannot rule out the possibility of reverse causation, in which children's emotional and behavioral problems may lead to parental mental disorder. However, this is unlikely to fully account for intergenerational cross-disorder associations. Research about maternal depression is instructive. Adoption research that has obtained information about mental disorders directly from participants by assessing the presence or absence of disorders, rather than by relying on information obtained from health records, shows that maternal depression is a risk factor for both internalizing and externalizing disorders in offspring (e.g., conduct disorder, ADHD, substance-use disorders), and that this risk operates not only through genetic transmission but also through environmental influences (Tully et al., 2008). Moreover, longitudinal research suggests that the association between maternal depression and children's externalizing behavior problems cannot be accounted for by reverse causation (i.e., by children causing their mothers to become depressed; Kim-Cohen et al., 2005).

Thus far, we have assembled information from two data sets which offer an atlas of family-resemblance coefficients for a wide range of mental-health conditions. We used nationwide data from health registries to examine assortative mating and parent–offspring resemblance because registries offer large sample sizes with which to study multiple conditions, including low base-rate conditions, across extended periods of time. Whereas the hospital-treatment data provide an important window for observing same- and cross-disorder associations for severe mental illnesses, the primary-care data are illuminating because they provide a window for observing associations across a much wider range of mental-health problems, from mild to moderate to severe, and for conditions that do not ordinarily involve or require hospital treatment. Moreover, the primary-care data allowed us to see that the increased risk of a wide range of different psychological problems among offspring of parents with a mental disorder emerges years before the onset of disorders that require hospital treatment. For example, the children of parents who experienced different specific mental-health conditions (e.g., depression, posttraumatic

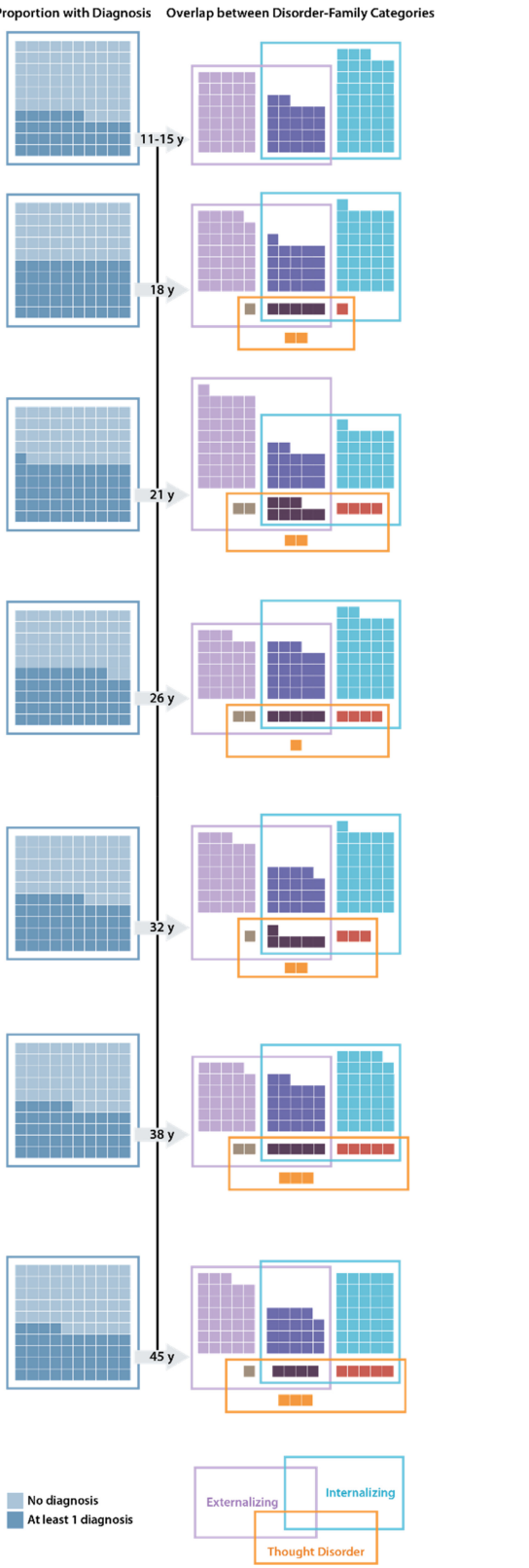
Figure 4

Parent–Offspring Associations for Mental Disorders Observed in Nationwide Primary-Care Data (N = 818,221 Parent/Child Pairs)



Note. Panel A shows the odds ratios between parents' disorders and their offspring's disorders. We examined a joint measure of parental diagnoses, which took the value of 1 if either parent had received a diagnosis. Panel B shows that the risk of mental disorders to offspring of parents with a mental disorder is transdiagnostic, and extends to disorders not present among parents (i.e., parents with a specific mental disorder were more likely to have offspring who had different mental disorders, even when the parents themselves did not have the co-occurring condition. Panel C shows odds ratios between randomly matched parents' disorders and offspring's disorders. Blue (light gray) text indicates that the odds ratio is <1.0. Light gray cells indicate that the confidence interval included 1. ADHD = attention deficit hyperactivity disorder; PTSD = posttraumatic stress disorder; NOS = not otherwise specified. See the online article for the color version of this figure.

Figure 5
Comorbidity Is Pervasive at Every Age



(*figure continues*)

stress disorder (PTSD), psychosis) were not only more likely to experience the same conditions, but they were also more likely to experience stuttering, enuresis/encopresis, or learning problems in the first decade of life. Each of the coefficients in the resulting atlas indexes links between any two conditions; each is interesting on its own and each invites speculation and can inspire research programs. However, stepping back from these trees—from the links between any two conditions—reveals a transdiagnostic forest. Both assortative mating and parent–offspring resemblance for mental disorders are highly transdiagnostic.

The Longitudinal Course of Mental Disorders Is Characterized by a Succession of Different and Changing Conditions

Given that cross-trait assortative mating is pervasive and that parents confer transdiagnostic risk to their offspring, what might we expect the natural history of mental disorders to look like? To answer this question, we turn to the Dunedin Longitudinal Study (Table 1). The Dunedin Study is unique in the annals of psychiatric epidemiology. In 1983 and 1984, when participants were aged 11 years, it was the first cohort to measure disorders in children using standardized diagnostic interviews (Anderson et al., 1987). Research diagnoses have now been made on nine occasions, until participants turned age 45 years. These data are especially useful because the Dunedin Study has repeatedly interviewed individuals about multiple mental disorders and the resulting time-series does not rely on treatment seeking or intake data in medical settings.

Mental Disorders Across the Life Course: Evidence From a Birth Cohort

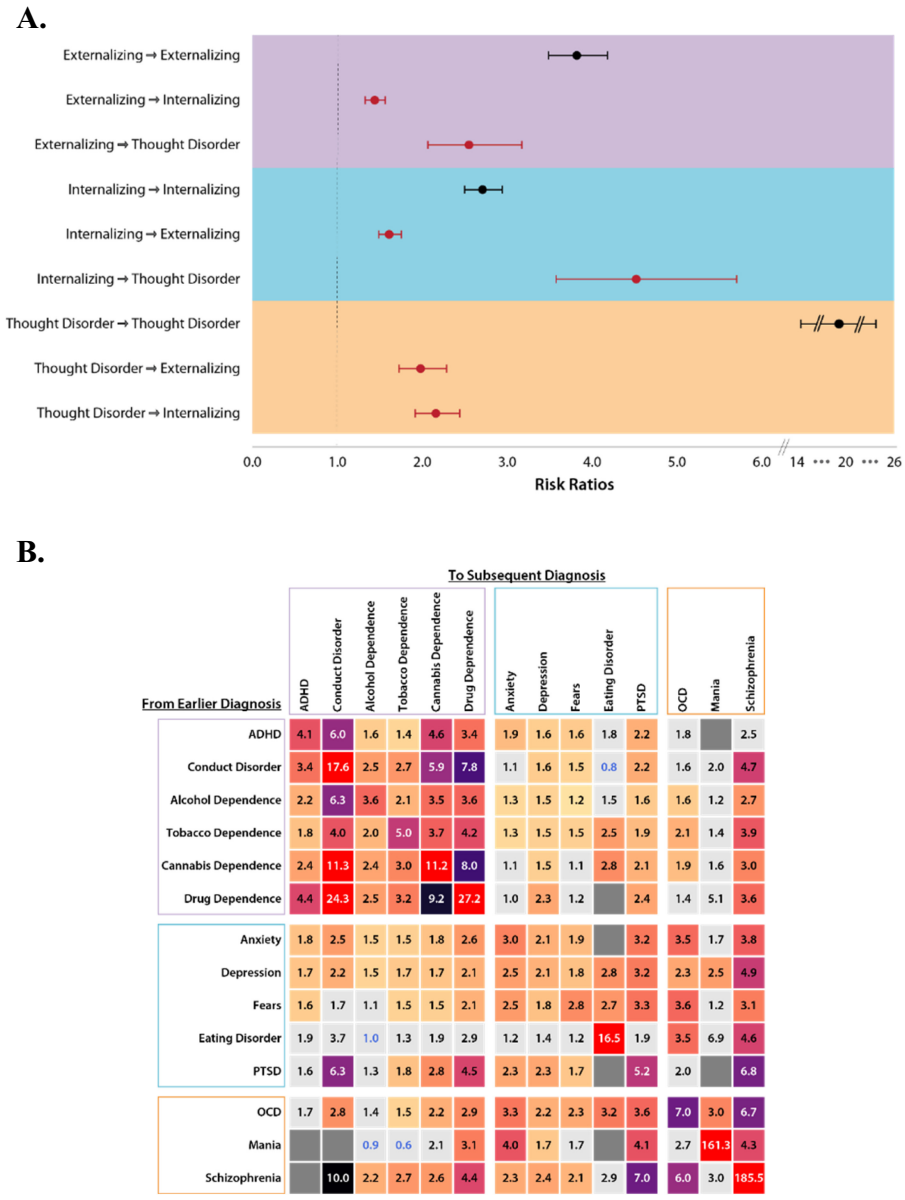
Concurrent Comorbidity Is Pervasive

Figure 5 shows the overlap, at each assessment phase of the Dunedin Study, between mental disorders grouped into three categories: Externalizing, internalizing, and thought disorders. At every age, people who met diagnostic criteria for one type of mental disorder were more likely to meet diagnostic criteria for other types of mental disorders (see Table S6 in the online supplemental materials). This is old news (de Jonge et al., 2018; Kessler et al., 1994; Newman et al., 1998). Over the past three decades, knowledge about widespread comorbidity has stimulated much of the research about the structure of psychopathology (Forbes et al., 2016; Krueger et al., 1998).

Figure 5 (*continued*)
Note. The figure shows the percentage of study members meeting diagnostic criteria for a mental disorder in the past 12 months at each assessment phase of the Dunedin study (12-month periods have been combined for the age 11, 13, and 15 assessments). The Venn diagrams show the overlap, at each assessment phase, between disorders grouped into three higher-order disorder-family categories: externalizing, internalizing, and thought disorders. Each square on the left-hand diagrams represents 1% of the study members at each assessment phase. Each square on the right-hand diagrams represents 1% of the diagnosed study members at each assessment phase. *OR*s for associations at each age are provided in Table S6 in the online supplemental materials. See the online article for the color version of this figure.

Figure 6

Sequential Comorbidity Is Widespread



Note. Panel A summarizes the sequential comorbidity of externalizing, internalizing, and thought disorders. Participants with a disorder in any of the three diagnostic families at one specific age were at significantly higher risk for both other diagnostic families at subsequent ages. The risk ratios in black depict the continuity of the same disorders (e.g., "What is the risk of people with an Internalizing disorder at age 15 or at age 18, or at age 21, etc., presenting with a subsequent Internalizing disorder at later phases?"). The risk ratios in red (gray) depict sequential comorbidity (e.g., "What is the risk of people with an Internalizing disorder at age 15, or at age 18, or at age 21, etc., presenting with a subsequent externalizing disorder at later phases?"). Panel B shows the risk of presenting with a specific disorder at subsequent assessment waves given a specific disorder at an earlier assessment wave. The risk ratios on the diagonal depict the continuity of the same disorder; the off-diagonal risk ratios depict sequential comorbidity from the row diagnoses to the column diagnoses. Dark gray cells indicate that risk ratios could not be estimated given that models would not converge. Light gray cells indicate that the confidence interval included 1. ADHD = attention deficit hyperactivity disorder; PTSD = posttraumatic stress disorder; OCD = obsessive compulsive disorder. See the online article for the color version of this figure.

Sequential Comorbidity Is Substantial

The newer news concerns sequential comorbidity (Caspi et al., 2020). Figure 6A shows that, in the Dunedin Study, people with a disorder in any of the three diagnostic groupings at one specific age were at higher risk for disorder in the other diagnostic groupings at subsequent ages. The risk ratios in black depict the continuity of the same disorders (e.g., "What is the risk of people with an internalizing disorder presenting with a subsequent internalizing disorder at later phases?"). The risk ratios in red (gray) depict sequential comorbidity (e.g., "What is the risk of people with an internalizing disorder presenting with a subsequent externalizing disorder at later phases?"). Moreover, all specific disorders were associated with an elevated risk for other disorders. Figure 6B shows the risk of presenting with a specific disorder at subsequent assessment waves given a specific disorder at an earlier assessment wave. The risk ratios on the diagonal depict the continuity of the same disorder; the off-diagonal risk ratios depict sequential comorbidity from the row diagnoses to the column diagnoses. Average risk ratios across assessment phases were calculated using generalized estimating equations that nested individuals within time. The overall impression in this figure is one of a positive manifold: Individuals who meet criteria for one disorder are significantly more likely to subsequently meet criteria for the same disorder (along the diagonal) but also different disorders. Of the 187 risk ratios estimated, 183 (98%) were positive and only four risk ratios were <=1.0. (Nine risk ratios could not be estimated as models would not converge; these nine mostly involved eating disorders and mania, which had the lowest prevalence rates in the Dunedin study.) The figure makes clear that longitudinal "cross-disorder" patterns are not confined to particular pairings but are ubiquitous. This finding is not unique to the Dunedin study. An analysis of all admissions to Danish psychiatric facilities since 1969 showed that, over years, every psychiatric disorder predicted every other subsequent psychiatric disorder (Plana-Ripoll et al., 2019). Analyses of first-time diagnoses in Danish psychiatric facilities revealed that almost half of all patients had a subsequent diagnosis that was different from their initial diagnosis (Høj Jørgensen et al., 2023).

Mental-Disorder Life Histories

Cross-sectional and sequential comorbidity give rise to two noteworthy observations about mental-disorder life histories. The first observation is that people who have a single disorder are rare. By age 45, 86% (869 of 1,013) of Dunedin study members met diagnostic criteria for a mental disorder, but only 21% (179 out of 869) had met criteria for only one disorder. An individual may experience a single disorder at a particular point in time, but over the life course it is rare for an individual to experience only one type of mental disorder. For example, among participants in the Dunedin study who were ever diagnosed with an externalizing disorder, most (478 of 625 [77%]) also experienced internalizing or thought disorders, another 11% (67 of 625) had multiple kinds of externalizing disorders, and only 13% (80 of 625) experienced only one "pure type" of externalizing disorder, such as attention-deficit/hyperactivity disorder or cannabis dependence. Among participants in the Dunedin study who were ever diagnosed with an internalizing disorder, most (503 of 712 [71%]) also experienced externalizing or thought disorders, another 16% (113 of 712) had multiple kinds of internalizing disorders, and only 13% (96 of 712) experienced only one pure type of internalizing disorder, such as depression or one type of anxiety disorder. Among participants ever diagnosed with a thought disorder, fewer than 2% (three of 177) experienced only one pure type of thought disorder, such as obsessive–compulsive disorder, mania, or schizophrenia. By midlife, participants characterized by only one pure disorder were atypical. Of interest, of the 179 participants with a pure disorder, 74% (132 of 179) met the diagnostic criteria at only one assessment age, 19% (34 of 179) met the diagnostic criteria at two assessment ages, and 7% (13 of 179) met the diagnostic criteria at three or more assessment ages.

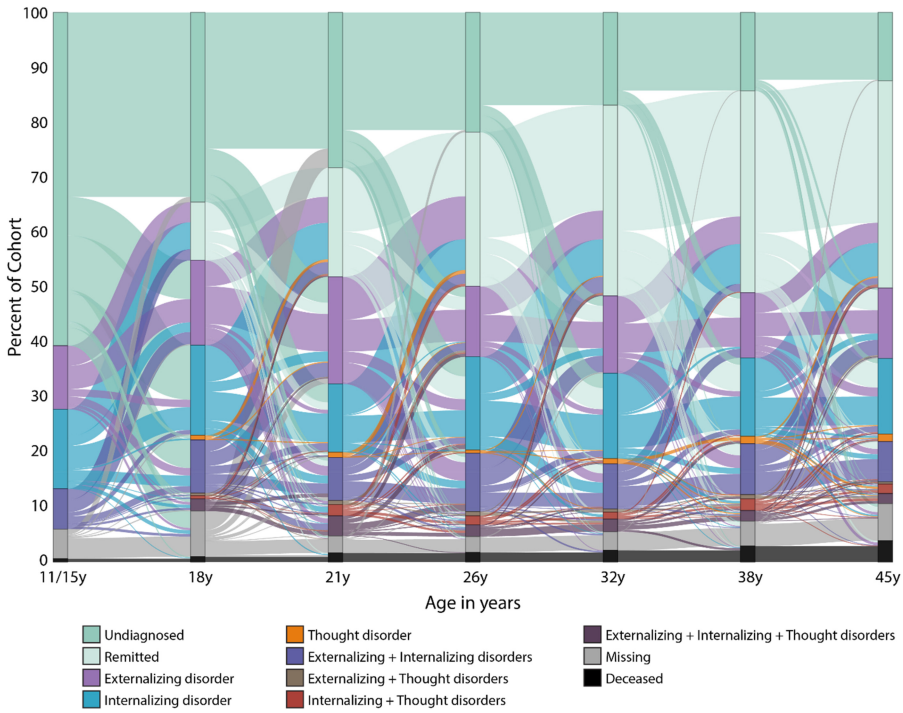
The second observation about mental-disorder life histories is that the longitudinal course of mental disorders is characterized by a succession of different and changing conditions. The Sankey chart in Figure 7 visualizes the flow of Dunedin study members from one adjacent assessment period to the next, beginning at ages 11 to 15 years and ending at age 45 years. The colors of the horizontal bands divide the diagram into different diagnostic statuses at each assessment age, as indicated in the key. The heights of the statuses in each horizontal bar show period-prevalence rates of different statuses at each assessment age. The figure shows that there was substantial movement between different diagnostic statuses in every direction throughout the first half of the life course. Tracing all 1,037 participants across time revealed 692 mental-disorder life history patterns, of which 605 (87.4%) were unique to one person. A cross-sectional view of mental disorders, with its focus on period prevalence and presenting diagnoses, is limiting. To appreciate how and why, we turn from a nomothetic approach to idiographic considerations.

Illustrating the Lived Experience of Mental Disorder as a Deck of Cards

Here we invoke the image of a deck of cards to illustrate the tension—and the mismatch—between how mental-disorder histories unfold across life and how mental disorders are typically studied. Imagine a longitudinal study as a deck of cards. Figure 8 depicts the mental-health histories of four individuals observed from t_1 to t_j. Their mental health at each age is shown on a card with symbols displaying different mental disorders. The symbols are organized in a three-color scheme corresponding to externalizing, internalizing, and thought disorders. Panel 8A depicts an individual who has never met diagnostic criteria for a mental disorder. Their life, their deck of cards, is devoid of any symbol; this person is a picture of enduring mental health. Panel 8B depicts an individual with episodic fears/phobias at different ages. As mental disorders reoccur and accumulate in this person's life, the color portion of successive cards expands to document how much their life is consumed by mental disorder. The watermarks that appear at ages when this person did not experience fears/phobias remind us that they experienced these in past years, and this remains a part of their life history. Panel 8C depicts an individual with a diverse externalizing history, shifting between conduct disorder and various substance dependencies over time. Finally, Panel 8D depicts an individual with a mental-health history characterized by multiple, shifting disorders within and across externalizing,

Figure 7

Flow of Dunedin Longitudinal Study Members From One Adjacent Assessment Period to the Next, Beginning at Ages 11–15 Years and Ending at Age 45 Years



Note. The figure traces all 1,037 study members across time. The horizontal bars depict 100% of the study sample, divided into different statuses at each assessment age. The statuses at adjacent assessment periods are linked to show paths through time. Adapted from "Longitudinal Assessment of Mental Health Disorders and Comorbidities Across 4 Decades Among Participants in the Dunedin Birth Cohort Study," by A. Caspi, R. M. Houts, A. Ambler, A. Danese, M. L. Elliott, A. Hariri, H. L. Harrington, S. Hogan, R. Poulton, S. Ramrakha, L. J. Hartmann Rasmussen, A. Reuben, L. Richmond-Rakerd, K. Sugden, J. Wertz, B. S. Williams, and T. E. Moffitt, 2020, *JAMA Network Open*, *3*(4), Article e203221, Figure 4A (https://doi.org/10.1001/jamanetworkopen.2020.3221). CC-BY. See the online article for the color version of this figure.

internalizing, and thought disorders (conduct disorder, substance dependence, anxiety, depression, obsessive compulsive disorder (OCD), and schizophrenia). By t_j, mental disorder has dominated their life.

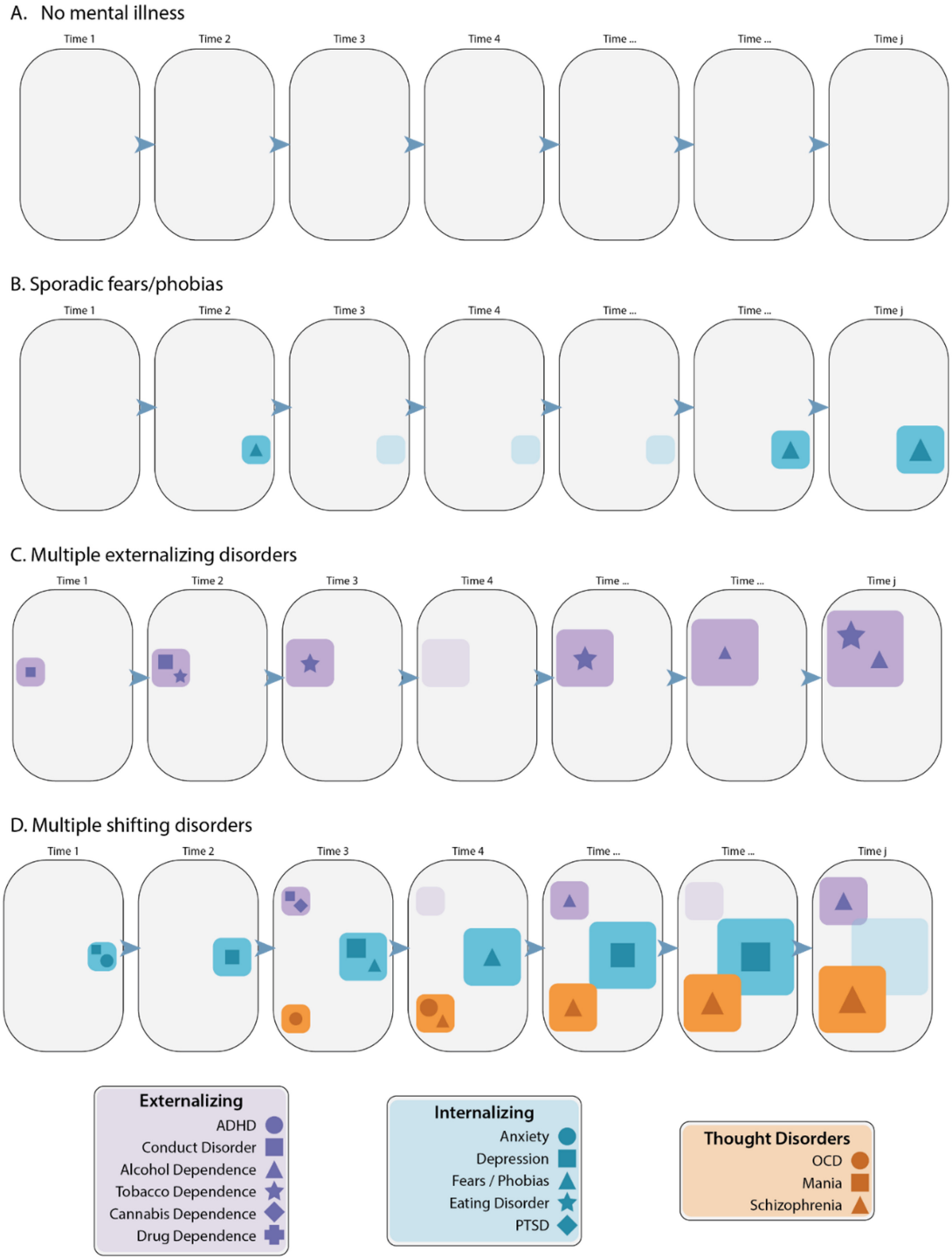
In Figure 9A, we have stacked the cards that make up this latter individual's life and compiled a "summary card" that shows their mental-disorder life history. A longitudinal cohort study is made up of many such "summary cards," or lives. The deck of cards in Panel 9B represents a longitudinal study of mental-disorder life histories, such as the Dunedin Study, where multiple mental disorders are monitored as they unfold for many individuals (i_1 to i_n) across time (t_1 to t_j). However, typical mental-health research ignores this developmental information. The limitations of typical approaches are visualized in Panels 9C and 9D. Cross-sectional research (Panel 9C) usually takes lives, slices them into cross-sections, and tries to learn about the nature of mental disorders by analyzing one cross-section at a time. This approach is informative about the nature of contemporaneous comorbidity, but it ignores developmental detail. Longitudinal research (Panel 9D) usually takes lives, slices them into disorder-specific time-series, and tries to learn about developmental changes one disorder at a time. This approach is informative about whether, how, and why a disorder of interest shows persistence, remission, or recovery, but it ignores cross-disorder changes over time. The deck of cards thus reveals the mismatch between the lived experience of mental disorder and the two most frequently used approaches to studying and treating these conditions.

Putting It All Together: A Three-Generation Analysis of Mental-Health Conditions

Most mental-disorder life-histories shift among different successive disorders, giving rise to high rates of comorbidity; the union of these lives creates cross-disorder assortative mating; and the resulting mating and rearing regenerate the phenomenon in the next generation. We document this by conducting a three-generation analysis of mental disorders in three different data sets, each of which offers complementary approaches to mental-disorder ascertainment (Table 4). In Denmark, we constructed family histories of mental disorders from nationwide hospital-treatment registries. In Norway we constructed family histories of mental-health conditions from nationwide primary-care registries. In the Dunedin study, we constructed family histories of mental disorders by using information gathered from study members and their parents.

Figure 8

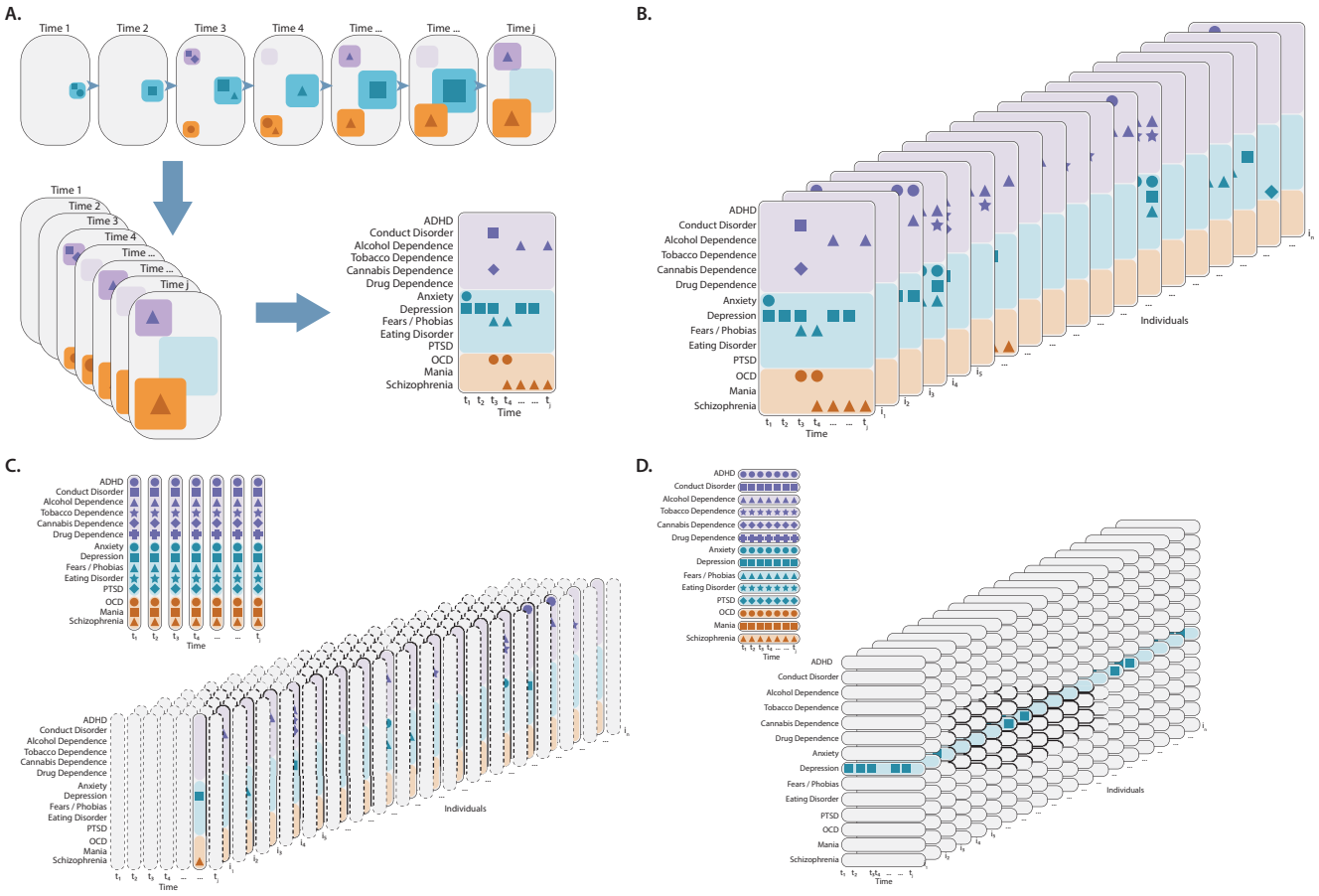
Mental-Disorder Life Histories



Note. The four panels depict the mental-health histories of four individuals observed from $Time_1$ to $Time_j$. Their mental health at each age is shown on a "card" with symbols representing different mental disorders. The symbols are organized in a three-color scheme corresponding to externalizing, internalizing, and thought disorders. As mental disorders reoccur and accumulate in a person's life, the color portion of successive cards expands to document how much of a life is spent with a mental disorder. The watermarks that appear at ages when a person did not experience specific conditions remind that they experienced these in past years, and this remains a part of their life history. ADHD = attention deficit hyperactivity disorder; PTSD = posttraumatic stress disorder; OCD = obsessive compulsive disorder. See the online article for the color version of this figure.

Figure 9

A Longitudinal Approach to Studying Mental-Disorder Life Histories



Note. Panel A stacks the "cards" that make up the mental-disorder life history of a single individual (shown in Figure 8D) and summarizes this history into one "summary card." Panel B shows how multiple life histories make up a longitudinal study, with each individual (i) represented by a "summary card" capturing their mental-disorder history. Panel C shows how cross-sectional research slices life histories into cross-sections and ignores developmental information when studying mental health. Panel D shows how typical longitudinal research slices life histories into disorder-specific time-series and ignores information about cross-disorder changes over time when studying mental health. ADHD = attention deficit hyperactivity disorder; PTSD = posttraumatic stress disorder; OCD = obsessive compulsive disorder. See the online article for the color version of this figure.

Externalizing, Internalizing, and Thought Disorders Within and Across Three Generations

In each data set, we tested the model shown in Figure 10. The model included (a) within-person correlations within each generation, between externalizing, internalizing, and thought disorders; (b) assortative mating correlations between partners within each generation (i.e., grandmothers with grandfathers; mothers with fathers); and (c) intergenerational transmission correlations between parents and children (i.e., grandparents to parents; parents to children). Tables S7, S8, and S9 in the online supplemental materials provide the descriptive statistics and pairwise polychoric correlations used in testing the three-generation model in each data set. Models were run in R (V4.3.1 in Denmark; V4.2.3 in Norway; and V4.4.1 in Dunedin) using the lavaan package (V0.6-16 in Denmark; V0.6-15 in Norway; V0.6-19 in Dunedin). Modeling used pairwise deletion of missing data and the weighted least-square means-and-variance-adjusted chi-square test. Decisions about which nested models to keep/reject were made based on the chi-square difference tests and changes to root-mean-square error of approximation, comparative fit index, and Tucker-Lewis index. In each data set we tested a series of model constraints. First, we tested constraints on the prevalence rates within generations (e.g., mothers = fathers; maternal grandmothers = paternal grandmothers); second, we tested constraints on within-person correlations within generations (e.g., mothers = fathers; maternal grandfathers = paternal grandfathers); third, we tested constraints on the assortative mating correlations within generations (e.g., male-to-female = female-to-male); fourth, we tested constraints on the intergenerational transmission correlations within generation pairs (e.g., mother-to-child = father-to-child; grandmother-to-mother = grandfather-to-mother); finally, we tested constraints on the assortative mating and intergenerational transmission correlations across generations (e.g., assortative mating in parents = assortative mating in grandparents; grandparents to parents = parents to

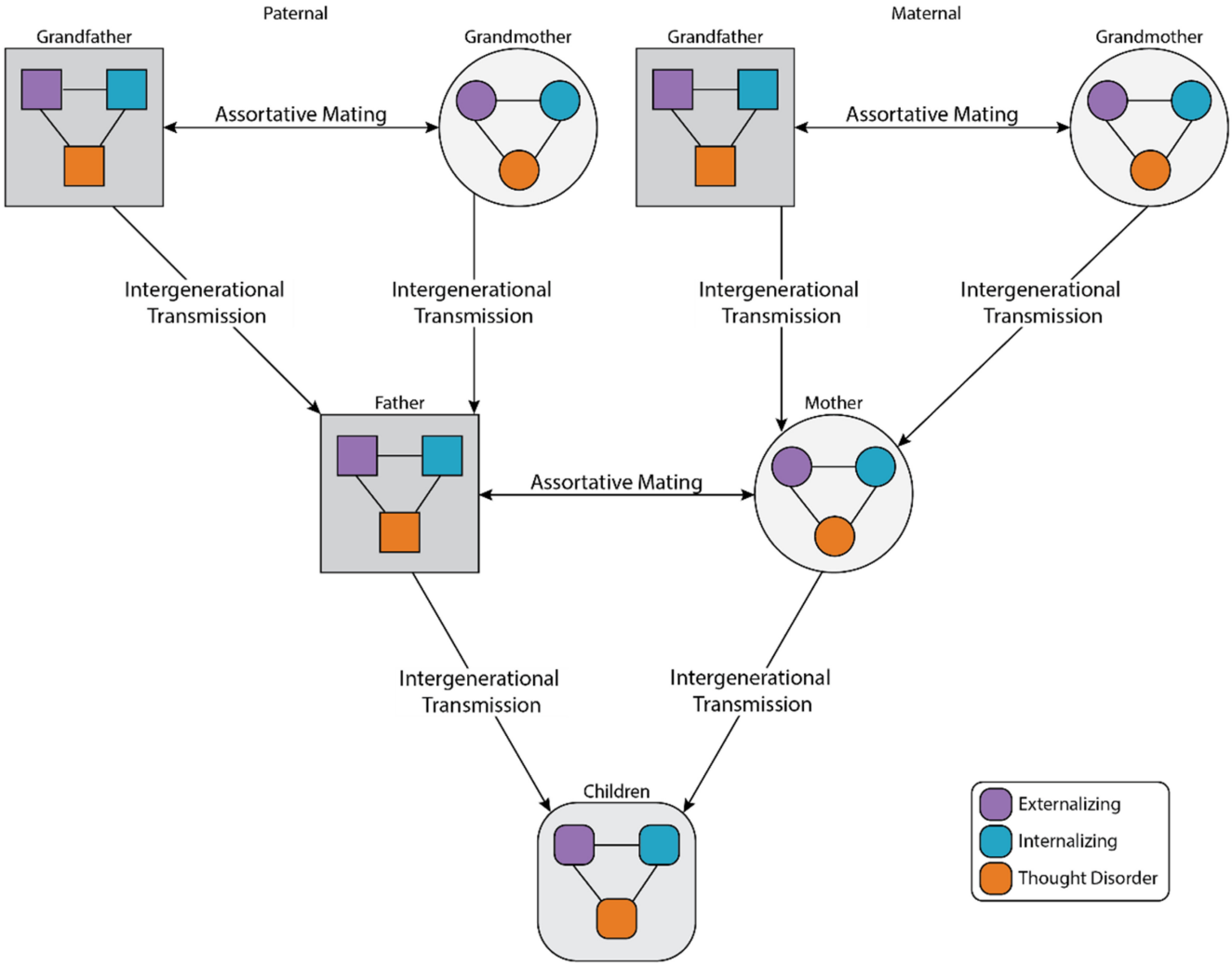
Table 4
Sources of Data Used in this Article to Conduct a Three-Generation Analysis of Mental Disorders

Nationwide hospital records	Nationwide primary-care records	Longitudinal birth cohort study
We constructed family histories of mental disorders using population-level administrative data in Denmark. We identified all children born in Denmark within 1985–1995, and we identified their parents as well as maternal and paternal grandparents in the population registers between 1986 and 2018. To be included, children needed to have both parents, and at least two grandparents who resided in Denmark between 1986 and 2018. Identified mothers and fathers were not required to be living with each other or their child. Families (for nesting purposes) were defined as unique mother/father pairs (i.e., if a mother had a child with more than one partner, each pairing was considered a new family). 625,238 children met these criteria and were nested in 415,029 families.	We constructed family histories of mental-health conditions using population-level administrative data in Norway. We identified all children born in Norway between January 2000 and December 2014, who were full-time residents in Norway from January 2006 until 2019 or until they died. These children were aged <0 to <6 at baseline and 5 to <20 years at the end of the observation period. From this base, we linked all mothers, fathers and grandparents who were likewise fully resident in Norway between 2006 and 2019. To be included, families needed a child, both parents, and at least two grandparents who met the residency requirement. Identified mothers and fathers were not required to be living with each other or their child. Families (for nesting purposes) were defined as unique mother/father pairs (i.e., if a mother had a child with more than one partner, each pairing was considered a new family). 704,960 children met these criteria and were nested in 432,083 families.	We constructed family histories of mental disorder in the Dunedin Study. Information about Dunedin Study members' grandparents and parents was obtained from the Dunedin Family Health History Study (Milne et al., 2008), which collected information about each study member's family from the study member and by interviewing both of the study member's parents. Data were collected between 2003 and 2006, when study members were 30–33 years old. Mothers and fathers provided information about the mental history of parents and grandparents and study members provided information about their parents. Study members did not report on their grandparents as we were not confident that they would have sufficient knowledge to report accurately. The mental health history of grandparents and parents was assessed using the FHS (Weissman et al., 2000), supplemented with items drawn from the DIS (Robins et al., 1995), the Short Michigan Alcoholism Screening Test (Selzer et al., 1975), and the Drug Abuse Screening Test (Skinner, 1982) to broaden the FHS's coverage. We also added a checklist of psychiatric conditions commonly understood by the public (e.g., "alcoholism," "depression"). In total, there were symptom-definition items pertaining to conduct disorder, alcohol dependence, drug dependence, major depressive episode, anxiety (generalized anxiety, panic, agoraphobia), and schizophreniform disorder. We obtained family history information for 981 families (out of 1,037 families). All three informants reported for members of 790 families, two informants reported for members of 154 families, and one informant reported for members of 37 families. A family member was considered to have a positive history of a disorder if one or more of the disorder's items were endorsed by at least 50% of informants (i.e., two of three informants, one of two informants, or one of one informant). We designated family members as having an externalizing disorder if they were reported as having any of the following: ADHD, conduct disorder/antisocial personality disorder, alcohol problems or drug problems; as having an internalizing disorder if they were reported to have had depression, generalized anxiety disorder, posttraumatic stress disorder, or any fear and as having thought disorder if they were reported to have had obsessive compulsive disorder, mania, or schizophreniform disorder.
Mental-health information about grandparents, parents, and children was obtained from hospital registers containing all inpatient contacts at psychiatric hospitals, wards and emergency rooms available from January 1970 until December 2018, and outpatient contact from January 1995 to December 2018, as described previously. We designated grandparents, parents, and children as having an Externalizing disorder if they had hospital-diagnosed externalizing behavior in childhood or adolescence (*ICD-10* codes: F90-F92 or equivalent *ICD-8* codes), or mental and behavioral disorders due to substance abuse (*ICD-10* codes: F10-F19 or equivalent *ICD-8* codes); as having an internalizing disorder if they had a hospital-level diagnosis of mood disorders (*ICD-10* codes: F32-F39 or equivalent *ICD-8* codes), neurotic disorders (*ICD-10* codes: F40–41 + F43–48 or equivalent *ICD-8* codes), or eating disorders (*ICD-10* codes: F50 or equivalent *ICD-8* codes); and as having a thought disorder if they had a hospital-level diagnosis of schizophrenia and related disorders (*ICD-10* codes: F20–29 or equivalent *ICD-8* codes), bipolar (*ICD-10* codes: F30–31 or equivalent *ICD-8* codes), or OCD (*ICD-10* codes: F60 or equivalent *ICD-8* codes).	Mental-health information about grandparents, parents, and children was obtained from primary-care records available from January 2006 until December 2019, as described previously. We designated grandparents, parents, and children as having an externalizing disorder if they had child/adolescent behavior symptom/complaint (ICPC-2 codes: P22 or P23), ADHD (ICPC-2 code: P81), or substance abuse (ICPC-2 codes: P15, P16, P17, P18, or P19); as having an internalizing disorder if they had a primary-care record of depression (ICPC-2 codes: P03 or P76), acute stress reaction (ICPC-2 code: P02), anxiety (ICPC-2 code: P74), phobia/compulsive disorder (ICPC-2 code: P79), posttraumatic stress disorder (ICPC-2 code: P82), or somatization (ICPC-2 code: P75); and as having a thought disorder if they had a primary-care record of psychosis (ICPC-2 codes: P71, P72, P73, P98).	Information about Dunedin Study members' mental disorder was obtained from interviews that have been conducted with them repeatedly throughout their lives, from ages 11 to 45 years, as described earlier in this article. We designated study members as having an externalizing disorder if they ever had any of the following: ADHD, conduct disorder, alcohol dependence, cannabis dependence, other drug dependence, or tobacco dependence; as having an internalizing disorder if they ever had any of the following: depression, generalized anxiety disorder, fears (social phobia, simple phobia, agoraphobia, panic disorder), PTSD, or eating disorders; and as having thought disorders if they ever had obsessive–compulsive disorder, mania, or schizophrenia.

Note. *ICD = International Classification of Diseases*; ICPC = International Classification of Primary Care; ADHD = attention deficit hyperactivity disorder; FHS = family history screen; DIS = Diagnostic Interview Schedule; PTSD = posttraumatic stress disorder.

Figure 10

Modeling Mental Disorders Across Three Generations



Note. The figure shows within-person correlations for each individual, assortative mating correlations in each generation, and parent–child resemblance across generations. See the online article for the color version of this figure.

children). Model comparisons, as well as final model fit statistics for each data set are shown in Table S10 in the online supplemental materials. Grandparent-to-grandchild and "in-law" correlations were set to 0 for simplicity, as they are not central to our study (but see sensitivity analyses in Tables S11 in the online supplemental materials where correlations across two generations and between in-laws are retained).[1]

Results from the final model in each data set are presented in Table 5. The results are striking in their consistency, especially considering that they rely on very different sources of information about mental disorders. Three patterns stand out. First, co-occurring disorders are widespread in each generation. For all family members and in each generation, having one type of disorder (externalizing, internalizing, and thought disorder) was positively associated with the risk of having another type of disorder. These within-person correlations varied a bit between generations within each data set, in

[1] We also retested the models specified in Table S10 in the online supplemental materials in which we allowed the "in-law" and grandparent-to-grandchild correlations to be freely estimated rather than constraining them to be zero; for example, this allows for the possibility that there is grandparent-to-grandchild transmission that does not operate solely through parents. The model-fit statistics and the results for each dataset are presented in Table S11 in the online supplemental materials. The results show that the within-person correlations, assortative mating correlations and intergenerational transmission correlations were practically unchanged across the two specifications, although the model-fit statistics were improved by freely estimating the in-law and grandparent-to-grandchild coefficients rather than constraining them to zero. Our three-generation models did not account for nesting of children within families which could impact the standard errors. Further sensitivity analyses restricted our datasets to one random child per family. Results are shown in Tables S12 and S13 in the online supplemental materials and document that results are essentially unchanged from those reported in Tables S10 and S11 in the online supplemental materials.

Table 5
Associations Between Mental Disorders Across Three Generations

	Denmark			Norway			Dunedin		
	Children DOB: 1985–1995			Children DOB: 2000–2014			Children DOB: 1972–1973		
Person/disorder family	Ext	Int	ThD	Ext	Int	ThD	Ext	Int	ThD
Comorbidity correlations									
Grandfather									
Ext	—			—			—		
Int	.70[a]	—		.40[a]	—		.14	—	
ThD	.52[b]	.63[c]	—	.32[b]	.37[c]	—	.17	.52	—
Grandmother									
Ext	—			—			—		
Int	.70[a]	—		.40[a]	—		.37	—	
ThD	.52[b]	.63[c]	—	.32[b]	.37[c]	—	.29	.53	—
Parents									
Ext	—			—			—		
Int	.74	—		.47	—		.46	—	
ThD	.65	.68	—	.50	.53	—	.56	.60	—
Children									
Ext	—			—			—		
Int	.59	—		.35	—		.21	—	
ThD	.59	.65	—	.38	.47	—	.33	.57	—
	Grandfather			Grandfather			Grandfather		
Person/disorder family	Ext	Int	ThD	Ext	Int	ThD	Ext	Int	ThD
Assortative mating correlations									
Grandmother									
Ext	.23	.15[d]	.09[e]	.26	.16[d]	.08[e]	.40[a]	.17[b]	.17[c]
Int	.15[d]	.14	.08[f]	.16[d]	.23	.11[f]	.17[b]	.30[d]	.18[e]
ThD	.09[e]	.08[f]	.07	.08[e]	.11[f]	.09	.17[c]	.18[e]	.20[f]
	Father			Father			Father		
Person/disorder family	Ext	Int	ThD	Ext	Int	ThD	Ext	Int	ThD
Mother									
Ext	.30	.19[g]	.18[h]	.43	.27[g]	.24[h]	.40[a]	.17[b]	.17[c]
Int	.19[g]	.21	.15[i]	.27[g]	.33	.23[i]	.17[b]	.30[d]	.18[e]
ThD	.18[h]	.15[i]	.19	.24[h]	.23[i]	.18	.17[c]	.18[e]	.20[f]
	Grandparent/parent			Grandparent/parent			Grandparent/parent		
Person/disorder family	Ext	Int	ThD	Ext	Int	ThD	Ext	Int	ThD
Intergenerational transmission correlations									
Parents/children									
Ext	.25	.19	.14	.27	.20	.13	.30	.11	.10
Int	.19	.19	.15	.16	.21	.11	.19	.19	.09
ThD	.16	.16	.21	.15	.16	.18	.18	.16	.12

Note. The table shows correlations between externalizing, internalizing, and thought disorders estimated in the final, best-fitting structural equation model in each of three data sets (nationwide hospital data in Denmark; nationwide primary care data in Norway; interview data in the Dunedin Study, New Zealand). Descriptive statistics and observed correlations are shown in Tables S7–S9 in the online supplemental materials; model fit statistics are shown in Table S10 in the online supplemental materials. Like superscript letters within each data set indicate correlations that were constrained to be equal. DOB = date of birth; Ext = externalizing; Int = internalizing; ThD = thought disorder.

ways that might be expected. For example, co-occurrence of mental disorders was highest in the Danish data which included mental disorders that were recorded in hospital registries, a pattern expected given that hospitalization usually occurs for more severe conditions and severity is associated with comorbidity. Still, the associations across the three data sets were uniformly positive and significant, and consistent with what is known about ubiquitous comorbidity. Second, assortative mating is widespread and transdiagnostic. In each generation, men and women who partnered with members of the opposite sex partnered with people who were more likely to share the same mental disorder with them and also to have different mental disorders from them. These assortative mating correlations varied a bit between generations in some data sets, but they were uniformly positive and significant. Third, the familial risk of mental disorders is transdiagnostic. From one generation to the next, parents and children were concordant for the same mental disorders, but parents

were also more likely to have offspring who experienced different disorders. These familial correlations varied a bit between generations in some data sets, but they were uniformly positive and significant. These three observations highlight the significant positive manifold of mental disorders within and across lives and families. All disorders go together.

Discussion

We have compiled evidence that assortative mating occurs across multiple mental disorders, that parent-to-offspring transmission crosses multiple mental disorders, and that the longitudinal course of mental disorders is characterized by a succession of multiple changing conditions. Men and women with a history of mental disorders tend to mate with partners who are also prone to have mental disorders, but not necessarily the same disorders. This creates a situation whereby their offspring, whether through genetic and/or environmental transmission, are at heightened risk of developing multiple different mental disorders. However, which specific disorder offspring ultimately develop is not easy to predict. Given that offspring inherit these multiple liabilities, it may not surprise that these liabilities manifest as different disorders at different points throughout their lives. However, which disorder emerges when is difficult to predict. The course of mental disorders within individuals and their families has been difficult to predict because mental-health research often looks at psychopathology one disorder at a time. This single-disorder approach has been expedient, because dealing with one disorder at one point in time is manageable. However, prioritizing manageable research projects means that we have oversimplified the complexity of mental disorders in the lives of patients. The challenge is not unique to psychopathology research; all fields need to try to adopt new forms of inquiry when progress slows down (Weinfurt, 2020). The intergenerational and developmental evidence about the familiality and course of mental disorders invites a reckoning for psychopathology research and clinical science, which tend to focus on one disorder at a time.

Are the Results Attributable to the Limitations of Diagnostic Systems?

Before turning to the implications of the findings, an obvious problem must be acknowledged: The diagnostic edifice may be flawed. One possibility is that contemporary systems for diagnosing mental disorders are unreliable. Moreover, even though diagnostic systems contain explicit diagnostic criteria, in practice the criteria are not always uniformly applied. Even when the criteria are applied, they may encourage simplistic application of checklists to complex conditions. However, noisy measurement alone is unlikely to account for the findings we have reported. First, we observed systematic patterns of association that defy unreliable measurement. For example, same-disorder assortative mating was higher than cross-disorder assortative mating; same-disorder parent–child resemblance was higher than cross-disorder parent–child resemblance; and homotypic continuity was greater than heterotypic continuity. While we have drawn attention to remarkable transdiagnostic associations, we should not lose sight of these expected same-disorder associations. Second, we drew on data from very different sources, each with its own strengths and limitations. We used hospital-treatment data, which selected for severe, persistent and comorbid cases diagnosed by specialists; primary-care data, which captured less-severe cases and provided a view of mental disorders diagnosed by a nation's frontline healthcare providers; and diagnostic interviews with members of a birth-cohort study, which offered a view of experiences with mental-health difficulties provided by individuals themselves irrespective of health-care utilization. Third, we relied on different classification systems, including *International Classification of Diseases, International Classification of Primary Care,* and *Diagnostic and Statistical Manual of Mental Disorders*. The findings converge in showing how mental disorders are patterned across and within generations: there is widespread cross-disorder assortative mating, widespread cross-disorder parent–child resemblance, and widespread shifting of mental disorders across the life course.

A related possibility is that mental disorders fail to separate neatly both within and across generations because mental disorders lack validity as discrete nosological entities. Mental functioning is multidimensional and continuous, whereas diagnoses are categorical and binary. However, this does not mean that they do not have utility; that is, they may continue to offer testable propositions about etiology and prognosis, and prove useful to practicing clinicians (Jablensky, 2016; Kendell & Jablensky, 2003). Whatever flaws exist in contemporary diagnostic systems for classifying mental disorders, these are the systems that researchers and clinicians work with today. Our hope is that accurate facts about the developmental epidemiology of mental disorders—both across and within generations—will enhance their phenomenological accuracy and increase their usefulness in guiding etiological, prognostic, and treatment research.

Why Is Specificity so Hard to Find?

The search for specificity, guided by the classification of different mental disorders as distinct kinds, has dominated psychopathology and clinical science. Specificity is generally regarded as an etiological goal. Consider the link between inflammation and depression (Miller & Raison, 2016). Patients with depression have elevated levels of immune molecules indicative of inflammation (Osimo et al., 2019). Observational studies suggest that exposure to maternal inflammation during fetal development is associated with depression risk in offspring (Lipner et al., 2024). Experimental studies show that administering inflammatory stimuli induces symptoms of depression (Yirmiya, 2024). Some Mendelian Randomization studies that have used genetic variants as instruments for elevated inflammation levels suggest a possible causal role of proinflammatory activity in depression (Kappelmann et al., 2021; Zeng et al., 2024). It may even be that inflammation is associated with a specific subset of depression symptoms (Frank et al., 2021; Jokela et al., 2016). However, against this background of a specific connection between inflammation and depression, inflammation also appears to cut across diagnostic categories, from neurodevelopmental disorders in childhood to anxiety, PTSD, OCD, and schizophrenia in adulthood (N. Yuan et al., 2019). Such transdiagnostic results suggest that, if associations are causal, therapies targeting inflammation might ameliorate many different disorders, or at least those inflammation-associated symptom clusters that cut across different mental disorders. Of course, it is possible that different mechanisms may connect inflammation with different disorders. Or, it could be that inflammation is linked to different mental disorders through

common immune-to-brain pathways affecting circuitries involved in motivation and executive control (Nusslock et al., 2024).

Specificity is also a prognostic goal. Consider depression, which forecasts increased risk of developing dementia and doing so at younger ages (Elser et al., 2023). This robust association has led to research aimed at testing whether depression is an early symptom of cognitive decline or a causal factor leading to dementia; it has led to recommendations for clinicians to monitor depression symptoms in their older patients; and it has raised the possibility that treating depression could modify dementia risk. Depression is the only mental disorder that features in the Lancet Commission's list of dementia risk factors (Livingston et al., 2024), and it has been incorporated into algorithms that are intended to identify adults' risk for dementia, to inform prevention (Schiepers et al., 2018). In part, depression appears on this risk-factor list because the evidence about depression is so convincing. However, focusing on depression perpetuates the lamppost effect where researchers only detect associations they seek and masks broader links between mental disorders and dementia risk. In fact, mental disorders of many types are associated with risk of developing dementia, including psychotic, substance use, neurotic, personality, developmental, and behavioral disorders (Richmond-Rakerd et al., 2022). These results would suggest that, if associations are causal, ameliorating any mental disorder in early life, not just depression, might mitigate neurodegenerative disease in later life. Of course, it is possible that different mechanisms may connect different disorders with the same dementia outcome. For example, depression may prompt neuroinflammation (Beurel et al., 2020), excessive alcohol use can lead to brain damage (Rehm et al., 2019), and psychosis may precipitate accelerated cognitive and functional decline (P. D. Harvey & Rosenthal, 2018). Or, it could be that all mental disorders are linked to the same dementia outcome via a common pathway, such as undermined social connectedness (Samtani et al., 2022).

It behooves to remember that transdiagnostic approaches have only recently been championed over disorder-specific approaches to studying mental disorders (Dalgleish et al., 2020; but see Garber & Hollon, 1991; A. Harvey et al., 2004). Transdiagnostic research has been spurred by the recent availability of massive data—both samples (e.g., electronic medical records, nationwide biobanks) and variables (e.g., high throughput genomics)—that point to features—both causes and consequences—that are shared by putatively different disorders. In addition, new within-person methodologies are complementing traditional between-person approaches to identify transdiagnostic processes in daily life (Wright et al., 2025). However, even now, there continues to be surprise when research reveals that different mental disorders share so much in common, whether these shared features involve genetic risk variants, vulnerability networks in the brain, core cognitive behavioral processes, or outcomes. This surprise is reflected in science communication. For example, august bodies have announced transdiagnostic genomic findings with headlines such as "Major mental illnesses unexpectedly share brain gene activity" (Dengler, 2018) and "Genes reveal surprising overlaps in brain diseases and disorders" (Tampa, 2023).

The message that emerges from the data we have presented is that it should not surprise that there is so much nonspecificity across different mental disorders. In fact, this is what we should anticipate. The epidemiology of mental disorders as revealed by intergenerational and developmental data suggests that the reason people with different disorders have the same risk factors and similar outcomes is that the same people will have different disorders, if followed long enough. Moreover, the developmental epidemiology of mental disorders suggests that if single-disorder loyalty in causes, intermediate phenotypes, or consequences—that is, specificity—is expected, research designs need to explicitly test for it, not presume it. Characterizing shared risks across different mental disorders should be a research priority, not an activity that is constituted on an ad hoc basis by integrating data on different disorders studied in different people. This requires not only transdiagnostic but also developmental approaches to sampling and to psychopathology measurement. We summarize recommendations in Table 6 and elaborate on these points in the next sections.

Table 6
Implications of Intergenerational and Developmental Evidence About "p"

Area	Recommendation
Study design	Adopt healthy skepticism about case-control designs
	When studying dimensions, examine multidisorder dimensions instead of a single-disorder dimension
	Avoid point-in-time cross-sectional research designs
Measurement	Assess a wide array of symptoms beyond the presenting complaint or focal condition
	Measure transdiagnostic constructs and processes
	Ascertain key developmental parameters of psychopathology: age-of-onset, life-course duration, and diversity of disorders or symptoms
	Ascertain clinical staging, from asymptomatic risk to severe illness
	Gather family mental-health histories using reliable and valid tools
	Ascertain participants' lifetime mental-health histories using reliable and valid tools
Treatment	Treat stress generation in patients, regardless of diagnosis
	Develop support strategies and toolkits for carers in families with mental-health problems
	Develop couples-oriented strategies for managing mental-health problems
	If parent has a diagnosis, consider child safety irrespective of parental diagnosis type
	Prepare patients for a lifetime of mental-health hygiene; do not limit treatment to presenting symptoms
	Focus treatment on pluripotent symptoms that may differentiate into many different disorders
Funding	Encourage projects that go beyond a single focal mental-health condition and that consider a patient's complete mental-health picture over time
Publication	In articles, include a paragraph discussing whether findings are limited by a focus on a single mental-health condition or data collected at a single point in time
Training	Teach students about the intergenerational and developmental context of all mental disorders

What the Intergenerational and Developmental Evidence About Mental Disorders Tells Us About Psychopathology Research Designs

Mental-health researchers typically encounter the person they study at a single point in their life, when patients come in for treatment or when participants sign up to take part in a study. At that time, efforts are made to diagnose each person's presenting condition correctly. This is done because it is thought that a diagnosis adequately represents a patient's psychopathology. As a result, most research designs tend to be tailored to one presenting diagnosis (e.g., depression), or

perhaps comorbid ones (e.g., depression and co-occurring substance use). Diagnoses are relied upon to set up case-control comparisons to gather information about etiology, to identify mechanisms of intergenerational transmission, to detect sequelae, and to choose treatments to ensure the best response and prognosis.

The case-control design is efficient and has led to important discoveries (Paneth et al., 2004). However, imperfect sensitivity and specificity of diagnostic practices in selecting cases, as well as reliance on well-controls, can lead to biases that threaten validity (Schwartz & Susser, 2011). These problems have been discussed at length, but the use of case-control designs persists. In fact, case-control designs are gaining traction as researchers increasingly use advanced machine learning tools to train algorithms on large databases to identify features that can distinguish who will become a case of disorder X and which cases will benefit most from treatments (Lucasius et al., 2025).

It is incorrectly thought that the problem with case-control designs can be resolved by using dimensional data from quantitative classifications of psychopathology instead of categorical data from qualitative classifications of mental disorders. Even when research uses quantitative dimensions, the focus of research tends to remain on one dimension at a time. Rather than comparing people with a disorder (e.g., ADHD, depression, or OCD) to controls, tests now simply compare people who are located at different points along a single quantitative dimension of mental-disorder classification systems (e.g., internalizing; Kotov et al., 2011).

A fundamental problem with the case-control design, and with its dimensional extensions, is not only that it evaluates one disorder/dimension at a time, but also that it relies on point-in-time data to assess psychopathology. To appreciate the problem, we revisit the Sankey chart which showed mental-health trajectories of individuals studied across four decades (Figure 11A). Assume now that we did not have developmental information about mental-health histories of these individuals. Assume that, instead, we sampled them at a single point-in-time, assessed their mental health, and identified all those who share the same diagnosis at a particular point to constitute "cases" in a case-control design (Figure 11B). However, it turns out that each of these individuals has had a unique history of prior diagnoses (Figure 11C). Moreover, each individual will have a unique progression through later diagnoses (Figure 11D). If we had sampled these individuals at an earlier point, or at a later point, they would be classified differently. A related problem, called temporal bias, has been discussed in epidemiology where cases, who progress along a trajectory that differentiates them from controls at different rates, are sampled unevenly across the course of the healthy-to-ill trajectory (W. Yuan et al., 2021). Research that uses point-in-time assessments does not take into account the exchangeability of cases, as well as of controls, over time. It is not simply an issue of misclassification. Rather, it is a matter of the same person appearing differently at different points in the life course. As a concrete illustration, consider what would happen if we set out to study depression among young adults, and sampled individuals between ages 18–32 years to learn about brain function in depression or to learn about mechanisms involved in the intergenerational transmission of maternal depression. To obtain the answer, we used four repeated measurements gathered from Dunedin Study participants at ages 18, 21, 26, and 32 years to estimate the case-control status of individuals diagnosed with depression if they were resampled at a different point even within this rather narrow 14-year sampling window. We found that, on average, 31% of individuals who would have been selected as depressed "cases" at a point in time would be so selected again if sampled in the future, but 33% would have been selected as "controls" and 36% would have been classified as having a disorder(s) that did not include depression (Figure 12A). This is not limited to depression. We found the same pattern when we studied alcohol dependence (Figure 12B). Cross-sectional assessments capture a biased segment of the course of the developmental trajectory of a mental disorder. We need longitudinal–developmental information to fully understand mental health.

What the Intergenerational and Developmental Evidence About Mental Disorders Tells Us About Measuring Psychopathology

The caution against studying one disorder (or dimension) at a time begs the question: what are the alternative approaches? We list three common alternatives, but note that each is wanting. One alternative that has been tried with limited success is to redouble efforts to study one disorder at a time by reducing disorder heterogeneity. This can be achieved by identifying subtypes of specific disorders (e.g., ADHD, depression) on the basis of symptom profiles, brain circuits, or biomarkers. The hope is that phenotypic refinement will address the specificity conundrum and advance etiopathogenesis, identify targets for drug development, and improve the accuracy of treatment decision making (Zhang et al., 2023).

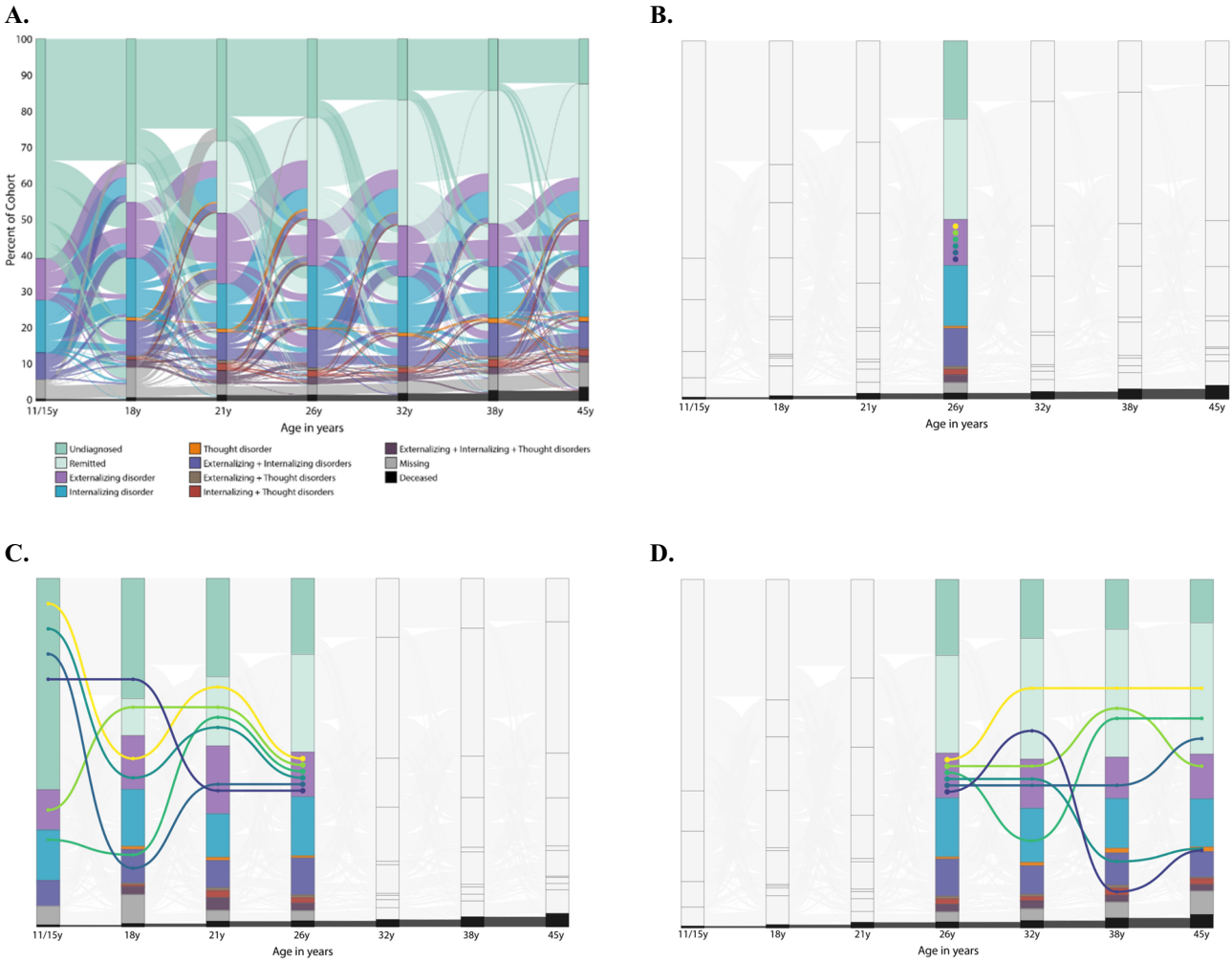
A second alternative is to measure a wide array of disorders/dimensions. Breadth of psychopathology phenotyping is crucial for research that seeks to evaluate what is common versus what is unique about different mental disorders. In clinical settings, the recommendation is to assess a wide array of symptoms of psychopathology beyond the presenting complaint (Lahey et al., 2017). In research settings, the recommendation is to assess a wide array of symptom dimensions that would cover disorders or multiple psychopathology spectra beyond the focal condition (Stanton et al., 2020). For example, the hierarchical taxonomy of psychopathology (HiTOP) is an empirical classification system that assembles symptom components and maladaptive traits identified in quantitative analyses of psychopathology data. Rather than relying on diagnostic categories, HiTOP identifies where individuals are located on continua of both general (common to many disorders) and narrow (specific to some disorders) features of mental illness (Conway et al., 2023; Kotov et al., 2017). HiTOP's hierarchical system is attractive because it allows scientists and clinicians to choose which level they want to focus on, depending on their goals (https://www.hitop-system.org/hitop-self-report-measures). Some commentators have suggested that the clinical utility of HiTOP has yet to be documented, and thus favor developing multidimensional symptom-based clinical characterizations of psychopathology (Leucht et al., 2024).

A third alternative is to turn to novel transdiagnostic constructs which can range from heuristic to data-driven to clinically informed. For example, the research domain criteria initiative identifies key brain-behavior constructs (e.g., negative valence systems, sensorimotor systems) that can be studied using different measurement strategies (e.g., genes, physiology, and behavior; Insel et al., 2010). Another approach, which has the added benefit of clinical utility, focuses on cognitive–behavioral processes (e.g., selective attention, expectancy bias, recurrent negative thoughts) that cut across different psychological disorders (A. Harvey et al., 2004).

The aforementioned approaches seek to address the dual challenge posed by (a) the heterogeneity that is apparent among people

Figure 11

A Visual Guide to Understanding Why Longitudinal Data Are Needed for Studying the Structure of Mental Disorders



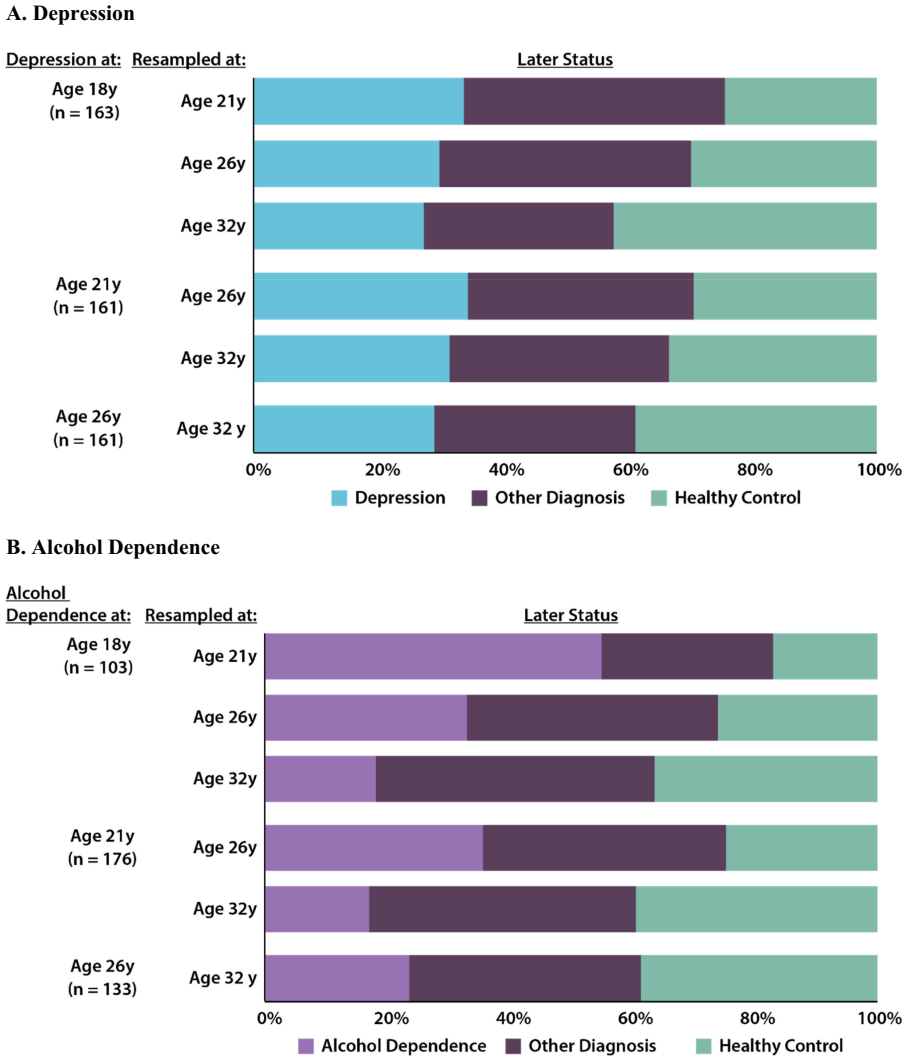
Note. Panel A shows the natural history of mental disorders, as taken from the Sankey chart in Figure 7. The snapshot in Panel B depicts point-in-time data collection intended to assess psychopathology in a sample of individuals. However, it turns out that people who had the same disorder at a point-in-time had a unique history of prior diagnoses (Panel C) and will have a unique progression through later diagnoses (Panel D). If these individuals had been sampled at an earlier point, or at a later point, they would be classified differently. See the online article for the color version of this figure.

diagnosed with the same disorder and (b) the similarity that is apparent among people diagnosed with different disorders. However, all of these measurement approaches are contemporaneous, focused on the here and now. For example, efforts to identify narrower and more homogenous phenotypes by using biological discriminators to subtype a disorder assume that more precise psychopathology phenotypes will emerge, but there is little evidence to suggest that biomarkers (apart from genetic variants) are more stable. HiTOP, which yields reliable dimensions, provides useful phenotypes for longitudinal research, but it does not include phenotypic features that describe the natural history of psychopathology. In contrast, the intergenerational and developmental evidence that we have presented suggests that to understand causes (where people have come from) and prognosis (where people are going) requires measurement strategies that explicitly incorporate familial and developmental information.

One approach could focus on three key developmental parameters that tend to covary within individuals and together signal a continuum of severity that differentiates between each person's mental-disorder life history: younger age-of-onset of disorder, longer life-course duration of disorder, and more diversity of disorders (across groupings of internalizing, externalizing, and thought disorders; Caspi, Houts, Fisher, et al., 2024). This developmental approach to defining mental-disorder life histories borrows insights from a cognate field of research, criminology, which documented that crime careers are defined by three developmental parameters: age-of-onset of offending, life-course duration of offending, and diversity of offense types committed (across groupings such as fraud, theft, and violence; Blumstein et al., 1986a). These three parameters tend to covary within individuals. For example, a "crime career" can be early-onset, chronic, and diverse or late-onset, brief, and specialized, or any pattern in between.

Figure 12

Cross-Sectional Assessments Capture a Biased Segment of the Course of a Mental Disorder's Developmental Trajectory



Note. We used 4 repeated measurements gathered from Dunedin study participants at ages 18, 21, 26, and 32 years to estimate the case-control status of individuals diagnosed with depression (Panel A) or with alcohol dependence (Panel B) if they were resampled at a different point even within this rather narrow 14-year sampling window. The figures show that many "cases" would have been selected as controls or would have been classified as having a different disorder if resampled at a different time point. See the online article for the color version of this figure.

Early-onset, long duration, and diversity found together define a phenotype that can be interrogated in etiological research and that signals a more significant liability to a serious crime career (Moffitt, 1993, 2018; Piquero et al., 2003). We draw on criminology not because we equate mental disorder with crime. Rather, just as the U.S. National Academy of Sciences report on the criminal-careers approach revolutionized crime research and justice policy (Blumstein et al., 1986b), it is possible that focusing on and measuring the developmental features of mental-disorder life histories may revolutionize psychopathology research and clinical practice.

Another approach could focus on clinical staging, a transdiagnostic framework which places individuals on a continuum of stages from a presymptomatic at-risk stage (Stage 0, defined, e.g., by a family history) to a severe illness stage (Stage 4; Scott et al., 2024). This approach borrows from staging models in general medicine which focus on illness progression (the course of the syndrome or the pathophysiology) and illness extension (symptoms and syndromes beyond the primary disease; Scott & Henry, 2017). Rather than concentrate on traditional disorders, transdiagnostic staging models make room for the mixed symptom profile that typifies clinical presentations and they reflect the dynamic

progression of developing psychopathology (P. McGorry & Nelson, 2016; P. D. McGorry & Mei, 2021). Clinical staging can inform indicated prevention as well as early intervention strategies. The hope is that, as in general medicine, the discovery of biomarkers and other modifiers may ultimately help to differentiate boundaries between different stages (P. McGorry et al., 2014).

The intergenerational and developmental evidence that we have presented also suggests that researchers and clinicians need better history-taking tools that will allow them to measure familial and developmental information. Intergenerational psychiatry (Duarte et al., 2020) underscores that a person's family history is not only a predictor of their risk for developing a disorder, but also predicts the course and prognosis of disorder (Milne et al., 2009). And yet, family history is not routinely gathered. There are barriers to collecting family-history information, ranging from the disinclination of participants/patients to the lack of confidence among researchers/clinicians. The absence of a gold-standard measurement approach has held back the routine gathering of such data. Recognition of the clinical and public health importance of family-history information has invigorated effective strategies for gathering and scoring family histories in an efficient and reliable manner. For example, the U.S. Centers for Disease Control and Prevention has developed tools and resources for gathering such information (https://www.cdc.gov/family-health-history/index.html).

Gathering accurate lifetime retrospective reports of mental disorders is an additional priority for research and practice. The manner in which most researchers collect lifetime reports of mental disorders yields unreliable retrospective data (Simon & VonKorff, 1995). However, methods are being refined. An example is the use of life-history calendars that use visual aids, inquire about streams of events, record event sequences, and contextualize questions about various life events to improve the quality of retrospective reports (Caspi et al., 1996; Freedman et al., 1988). Life-history calendars have been shown to yield more reliable information than standardized questionnaires about various vulnerabilities, including illnesses, crime victimization, and absenteeism (Belli et al., 2001; Morselli et al., 2016; Yoshihama et al., 2005). Importantly, calendars have been shown to improve measurement of lifetime experience with mental disorders (Axinn et al., 2020). A developmental view prioritizes valid expert history-taking both to enhance accurate measurement for research purposes and to support strategic treatment planning in patients' lives. There exists an opportunity for collaboration between cognitive scientists, psychopathologists, and clinicians to develop accurate data-collection tools for gathering lifetime retrospective reports of mental disorders.

What the Intergenerational and Developmental Evidence About Mental Disorders Tells Us About Treatment

The evidence presented here cautions against overreliance on diagnosis-specific research and clinical protocols. It also supports advancing and evaluating transdiagnostic therapeutic approaches (Caspi & Moffitt, 2018). However, this is not news. Transdiagnostic treatments are increasing in popularity (Hyde et al., 2022; Mansell et al., 2009; M. A. Meier & Meier, 2018; Sloan et al., 2017) and they are proving effective, especially in treating emotional disorders (Cuijpers et al., 2023), although there are skeptics (Fusar-Poli et al., 2019; but see Mansell, 2019). However, more than that, a life-course approach to mental-health problems orients practice away from looking at a single disorder at a single point in time toward considering the dynamics of an individual's mental-disorder life history. In this regard, the intergenerational and developmental data presented here suggest some additional implications for treatment.

The transdiagnostic familial data (both the assortative mating and parent–offspring data) suggest that the challenges posed by stress-generation processes are greater than they might initially appear. Individuals with mental disorders are known to generate stress for themselves, which exacerbates symptoms and contributes to the maintenance of mental health problems (Liu et al., 2024; Rnic et al., 2023; Santee et al., 2023). Stress generation was initially studied in relation to depression, but it is now recognized to be a transdiagnostic phenomenon. While stress generation is thought to contribute to a cycle of psychopathology and stress in an individual's life, the co-occurrence of multiple disorders among multiple people in the same family means that the cycle of stress and psychopathology is broader and deeper: People with any given disorder are likely to be contending with self-generated stress, as well as stress generated by family members experiencing lots of different disorders. Taming and reducing stress effectively is a treatment priority.

The transdiagnostic familial data also highlight an underappreciated circumstance surrounding caregiving. While providing care for family members with mental disorders is known to be challenging, these challenges are often shouldered by individuals who are contending with their own diverse mental-health problems. Effective support strategies and toolkits for family carers need to be devised with recognition that those who are called upon to advocate for, prevent crises, support recovery, and maintain a healthy relationship with a family member experiencing mental-health problems are managing their own, and often different, mental disorders.

The transdiagnostic assortative mating data suggest that couple-oriented interventions for managing mental disorders need to be considered alongside patient-based interventions, not only because aspects of partnerships affect physiology and behavior (Martire et al., 2010), but also because most individual mental disorders exist as part of co-occurring mental-disorder bundles in couples. Dyadic illness management, which calls on couples to manage diseases together, has attracted attention in clinical medicine (Manne & Soriano, 2025), especially in light of evidence about spousal concordance for both risk factors and diseases (Varghese et al., 2023). The data presented here suggest that practitioners and health-insurance providers need to be aware of the empirical evidence that may support couple-oriented strategies for managing mental health (Gil et al., 2023). The transdiagnostic intergenerational data suggest that interventions that are intended to prevent specific offspring disorders (e.g., depression) need to be conceived as interventions that seek to mitigate offspring's risk for a wide range of disorders also experienced in parents, rather than any specific parental disorder (McLaughlin et al., 2012).

The developmental data suggest that therapy cannot just mitigate presenting symptoms. Clinicians must treat the disorder that appears before them, offering relief for the patient's current complaint. However, because most patients' mental disorders appear to morph into different disorders, the long view suggests that therapy must also build fundamental skills for maintaining general and enduring mental health. Improving mental health literacy is a large part of this endeavor (Jorm, 2000). Therapy may also require light-touch monitoring and top-up support if new disorders emerge in the future, a health-

care model that resembles the way general practitioners provide repeated checkups and preventive medicine. In this regard, the data presented here provide a scaffold for learning how disorders are distributed in the population, within families, and across the life course.

Finally, screening tools are needed to assess an individual's life-course vulnerability to psychopathology to facilitate early preventive interventions. One approach is to identify pluripotent symptoms. These symptoms may be initially diffuse, but with time they may differentiate into many different disorders. Such pluripotent symptoms may represent a transdiagnostic prodrome for multiple mental disorders (Spooner et al., 2020). Another approach is to identify transdiagnostic risk factors. Initial work using artificial intelligence models trained on readily available psychosocial data documents that it may be possible to accurately predict future mental-disorder risk (p) and conversion to its highest levels while uncovering potential intervention targets, such as sleep disturbances (Hill et al., 2025).

Deconstructing "p"

It may be helpful to state what this article does not claim. First, it does not claim that clinicians should ignore patients' presenting conditions of specific disorders and focus only on general, transdiagnostic features of psychopathology. The clinician's immediate concern is to reduce risk of harm and alleviate pain and discomfort. However, at the same time, we must seek effective transdiagnostic treatments for mental disorders because these may offer a way to prevent subsequent, other mental-health disorders, and possibly physical-health disorders too (Moffitt & Caspi, 2019).

Second, this article does not claim that researchers should forsake efforts to find specific causes of specific disorders in favor of general, transdiagnostic features. Both goals can be pursued. This is explicitly addressed in the work of researchers who suggest that statistical models, such as bifactor models, can help orient the search for orthogonal causes of specific (sets of) mental-health conditions by quantifying the variance that is shared by specific symptoms after variance common to all symptoms has been partitioned (Lahey et al., 2017). However, at the same time, we are suggesting that most current research designs and point-in-time measurement strategies are not up to the task of finding true specific causes.

Finally, our claim that psychopathology research should stop studying one mental disorder at time is not tantamount to claiming that mental disorders should only be studied at the broadest level. The phenomenon of comorbidity, originally meant to convey coexistence of two or more conditions or disorders in the same person, suggested there is a more parsimonious structure to psychopathology than implied by nosologies that identify many distinct disorders (Clark et al., 1995). This has led to better understanding of spectra of closely related disorders. However, the spectra are also closely related, suggesting that clinical and research efforts need to be targeted at a more global level, not just at spectra.

The life-course epidemiological data that we have brought together suggest that it makes little sense to study mental disorders (or spectra) one at a time, treating each mental disorder (or spectrum) as a disembodied condition. Studying mental disorders one at a time does not accurately represent most patients' and families' lived experiences; it misleads about specificity; and it hides transdiagnostic discoveries from view. This message, supported by complementary findings across diverse data sources, suggests rethinking design, measurement, and treatment—as well as funding, publication, and training—all of which tend to be disorder-specific.

What does this have to do with "p"? A decade ago, the idea of p was advanced to summarize the ubiquitous overlap between different mental disorders and to suggest that there may be a propensity to develop any and all forms of mental disorders (Caspi et al., 2014). The idea of p captured the imagination (Adam, 2023; Jones, 2020; Wickelgren, 2024). Multivariate techniques such as exploratory factor analysis and confirmatory factor analysis have been used to study this idea (Caspi, Houts, Fisher, et al., 2024; Caspi, Houts, Belsky, et al., 2014; Lahey et al., 2012), just as these techniques had been used earlier to study empirical patterns of co-occurring psychological symptoms and identify spectra of mental disorders such as internalizing conditions or externalizing conditions (Krueger et al., 1998). Whereas modeling and studying such spectra is regarded as a promising way forward in mental-health research (Conway et al., 2019), there have been debates about p on psychometric grounds (Pettersson, 2025; Watts et al., 2024). However, p is not simply a higher-order factor-analysis superspectrum (a p-factor). p is about how mental disorders are experienced within families and across lives. Almost all research uses cross-sectional data to model the p-factor. Such research can provide a reasonable, but only imperfect representation of p. It can do this because comorbidity at any given time captures the severity that is associated with earlier age-of-onset, persistence, and diversity of mental-health problems. In this sense, the p-factor that emerges in factor-analysis research is a surrogate for the life-course phenomenon that is documented here by the assortative mating, intergenerational, and developmental data. We hope that deconstructing p into its intergenerational and developmental components demystifies the idea to researchers, clinicians, and the lay public and invigorates research both into what unites all mental disorders and how to most efficiently improve population mental health.

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