

REPLY

What Does Psychopathology Research Have to Gain by Studying All Mental Disorders at the Same Time?

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The 16 constructive commentaries about our target article offer fresh ideas, new directions, and research questions. Here we amplify points raised about transdiagnostic approaches to etiology, treatment, and prevention; the role of cognitive ability in studying and treating mental disorders; the need to evaluate generational cohort changes in mental disorders; and the importance of assortative mating in life-course research and in treatment. We conclude by laying out a set of domains that would make sense for developing a p-informed measure of life-course vulnerability to psychopathology.

General Scientific Summary

In our target article, we made a case for why psychopathology research should avoid studying one mental disorder at a time. In responding to the thoughtful commentaries, here we try to show what psychopathology research has to gain by studying all mental disorders at the same time.

Keywords: structure of psychopathology, transdiagnostic approaches, developmental psychopathology

Discourse has become pugnacious in many spheres of life today, and debates in psychology—and among psychological scientists—likewise often turn quarrelsome. Against this background, we welcome the uniformly constructive commentaries about our article. As a set, the commentaries fuel an agenda for

research. They are brimming with fresh ideas, new directions, and research questions. We amplify several of the questions raised by flipping the title of our target article to ask: *What does psychopathology research have to gain by studying all mental disorders at the same time?*

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Implications of a Transdiagnostic Approach for Etiology, Treatment, and Prevention

Specificity is one of the nine criteria, or “viewpoints,” which epidemiologists have historically used to evaluate whether an association between a risk factor and an outcome is likely to be causal (Hill, 1965). The idea is that if a risk factor is associated with many dissimilar outcomes it is less likely to be causal, and more likely to be a product of confounding, than if it is associated with fewer outcomes. This criterion is problematic (as attested by historical debates about tobacco smoking, which is both associated with many outcomes and causes them; Parascandola, 2014). Nevertheless, psychopathologists continue to seek evidence of specificity (one-to-one relationships between causes and disorders), whether through research design (e.g., case-control studies) or statistical procedures (e.g., controlling for comorbidity). Even in those instances where transdiagnostic discoveries have been made, these came against the background of seeking disorder-specific discoveries. For example, a recent study identified five highly correlated genetic factors that account for the majority of variance across 14 different psychiatric disorders, suggesting that the same genetic factors may influence multiple different conditions (Grotzinger et al., 2026). This exemplar of cooperative science (Harden, 2026) demonstrates the value of looking across disorders rather than studying one disorder at a time. However, it bears noting that the author list of this article is made up of working groups of the Psychiatric Genomics Consortium, each of which was originally focused on a different disorder when the Psychiatric Genomics Consortium began in 2007: Schizophrenia Working Group, Bipolar, Eating, Depression, and so on. Would we have gotten to the 2026 genetic finding faster by studying multiple disorders at the same time? Looking forward, can new insights be achieved faster by studies that collect comprehensive mental health data on multiple dimensions of psychopathology rather than sampling one or a couple of disorders at a time?

Identifying transdiagnostic causal factors is an etiological goal, but it can also inform more effective mental-health treatments. Targeting vulnerability factors that cut across disorders (e.g., emotion dysregulation) could reduce both current and future mental-health problems, when balanced against components that are tailored to an individual’s current presentation. Determining how to best achieve this balance represents an important aim of the next, “Third Wave” of psychological treatments (Dagleish, 2026). Intervening on universal risks is also resource-efficient (Mansell, 2026), which is necessary given the gap between treatment need and availability and the siloing of mental health care (McGinty & Eisenberg, 2022).

Although the question of specificity has traditionally focused on causal factors, it is equally relevant to downstream effects: Different mental disorders often have the same consequences. Consider unintentional physical injuries (i.e., injuries for which there is no evidence of predetermined intent) and also injuries due to assaults. Whereas injury researchers have focused on specific disorders, such as substance use and attention-deficit/hyperactivity disorder [ADHD], we recently generated a comprehensive evidence base about the link between multiple different mental disorders and physical injuries (Richmond-Rakerd et al., 2026). Multiple mental disorders were all associated with an elevated likelihood of being injured. Of special interest, multiple mental disorders were consistently associated with trauma to the head and face, a finding that offers a window to

identifying mechanisms by which multiple mental disorders in early life might shape brain health in later life. More generally, the findings suggest that injury psychoeducation could become a feature of transdiagnostic interventions.

Not only treatment but also prevention needs to become transdiagnostic. For this to happen, risk screening tools are needed that can capture transdiagnostic factors—a Framingham-style risk score for mental health (D’Agostino et al., 2013). Also needed are screening tools for overall psychopathology (p) that take a multiple mental-disorder approach (Neulinger et al., 2024). This message has found its way into clinical settings. For example, readers who have visited American primary-care and hospital clinics may have noticed that over the past decade the content of intake questionnaires has broadened considerably to inquire not only about depression but also about anxiety, substance use, concentration difficulties, and so forth. Along these lines, Tackett and Katz (2026) draw attention to the possibilities afforded by integrating personality assessments into models of psychopathology and possibly into risk assessment. As pointed out in the commentaries, tools that screen for overall mental illness or that capture p (Moore et al., 2019) are not a substitute for specific diagnoses, but they can inform prognosis and treatment planning (Pettersson, 2026; Pettersson et al., 2020).

Cognitive Ability

Our target article did not address cognitive abilities (Wilson et al., 2026), a significant omission given evidence that low IQ and mental disorders cluster in families (Weiser et al., 2023). There are at least three ways to incorporate information about cognitive ability into psychopathology and clinical science. First, in relation to nosology, information about cognitive dysfunction can be integrated into structural models of psychopathology. This kind of research is vital to better understand whether certain cognitive functions are more impaired in some disorders than in others and to identify cognitive functions that are impaired transdiagnostically (Ringwald et al., 2025).

Second, in relation to etiology, low cognitive ability has been proposed as a causal factor in the development of mental disorders (Barnett et al., 2006). Evidence from cognitive epidemiology (Deary & Batty, 2007) shows that low IQ is related to the risk of developing practically all mental disorders (Fries et al., 2025). For example, we recently examined the longitudinal associations between cognitive ability and mental disorders using military conscript test data from 18-year old Norwegian men ($N = 272,351$) and mental-disorder data from primary-care registers 20 years later (Nordmo et al., 2025). Lower cognitive ability was associated with a monotonically increasing risk of developing all the studied mental disorders (except bipolar disorder, a fascinating anomaly that has been observed in several studies [e.g., Gale et al., 2013; Koenen et al., 2009; Smith et al., 2015]). The associations were independent of educational attainment and held even when comparing the cognitive abilities of brothers raised in the same family, attesting that lower cognitive ability is not only associated with mental disorders because both arise from the same family-background circumstances. This aligns with evidence that cognitive difficulties characterize p. Individuals with higher levels of p fare less well on tests requiring attention, concentration, memory, as well as visual-perceptual processing speed and visual-motor

coordination (Caspi et al., 2014; Castellanos-Ryan et al., 2016; Martel et al., 2017). These cognitive difficulties are not simply a consequence of lifelong disorders; they are present already in early life, before the onset of most disorders (Caspi & Moffitt, 2018). Several factors, not mutually exclusive, could account for these transdiagnostic associations and warrant research. Lower IQ may be a marker of neuroanatomical and functional brain differences that affect executive function, attention, and processing speed and increase vulnerability to mental disorders. Individuals with lower IQs are also more likely to encounter stressful life events and are less equipped to cope with stressors, making them potentially more vulnerable after such events. Lower IQ may make activities of daily living more challenging and, therefore, stressful. Negotiating public transportation systems, comparison shopping, managing money, helping children with homework, and navigating the Internet are more demanding for individuals with lower IQs (Gottfredson, 1997). Finally, the association between IQ and mental disorders may be mediated by mental health knowledge, which facilitates early help-seeking, improving access to evidence-based care and promoting treatment compliance.

Third, in relation to treatment, even if they are not transdiagnostic causal factors, cognitive difficulties are complicating features that are important to consider in prevention and treatment planning. Interventions attempting to support and improve cognition (Harvey, 2025; Harvey et al., 2014) can be a useful complement to transdiagnostic interventions for emotional and behavioral disturbances.

Generational Cohort Effects

The absence of history in our target article is also notable (Keyes, 2026). After all, developmental trajectories are shaped by the intersection of individual lives, families, and historical contexts (Elder et al., 1988). Cohort effects refer to differences in health among people born during different historical periods (Rohrer, 2025). There is solid evidence that mental-health problems are on the rise among young people (Keyes & Platt, 2024), leading the American Psychological Association to declare a crisis. Most efforts to address the causes of increasing mental-health problems focus on specific conditions, such as autism, ADHD, or depression. But do proffered explanations for increasing rates of one disorder apply to other disorders? Are there explanations for increases that may be common across different disorders? Consider evidence about youth mental health, as documented in primary-care settings. For this reply, we studied the mental health of nine 1-year birth cohorts of children born between 2001 and 2009, following each cohort for 10 years from ages 5 to 15 years (Table 1 in the target article describes the mental-health conditions assessed in primary-care settings). Even over this short span, there were increases in the rates of both internalizing conditions (e.g., anxiety, depression) and externalizing conditions (e.g., ADHD) among children born more recently. The data suggest that these increases may not simply be an artifact of more young people visiting primary-care physicians as we did not observe this increase for somatic conditions or injuries (Figure 1A). An alternative approach to tackling the question of increasing rates of mental-health conditions one disorder at a time is to focus on three developmental parameters that covary within individuals. Together these three parameters 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. These three parameters are core features of *p* (Caspi et al., 2020). We found that the age at first presenting to primary care for mental-health conditions has not become younger among children born more recently, but their mental-health conditions have become more persistent and more diverse (Figure 1B). This preliminary analysis is restricted to a brief historical period and a narrow developmental span, but it highlights how an emphasis on *p* rather than single disorders can reframe questions about causes and treatment.

Do explanations for the increasing rates of specific disorders (e.g., improved detection, greater awareness, changing diagnostic criteria, social media, eco-anxiety, and economic inequality) also explain the changing nature of mental-health trajectories among more recent birth cohorts? If these trajectories have really changed, we might also expect to see greater social, emotional, and occupational dysfunction (Atkinson et al., 2026). Indeed, such changing trajectories may align with increases in young people disengaged from education systems and the labor market (NEET: not in education, employment, or training) due to mental-health issues. Moreover, if the historical changes are not only disorder-specific changes but also changes in key developmental parameters that define *p*, it may be important to rethink service delivery. For example, if young people's mental-health conditions have become more persistent, greater emphasis would need to be placed in health care on moving young people from family-centered care to independent, patient-driven care as they grow older (Calabrese et al., 2022).

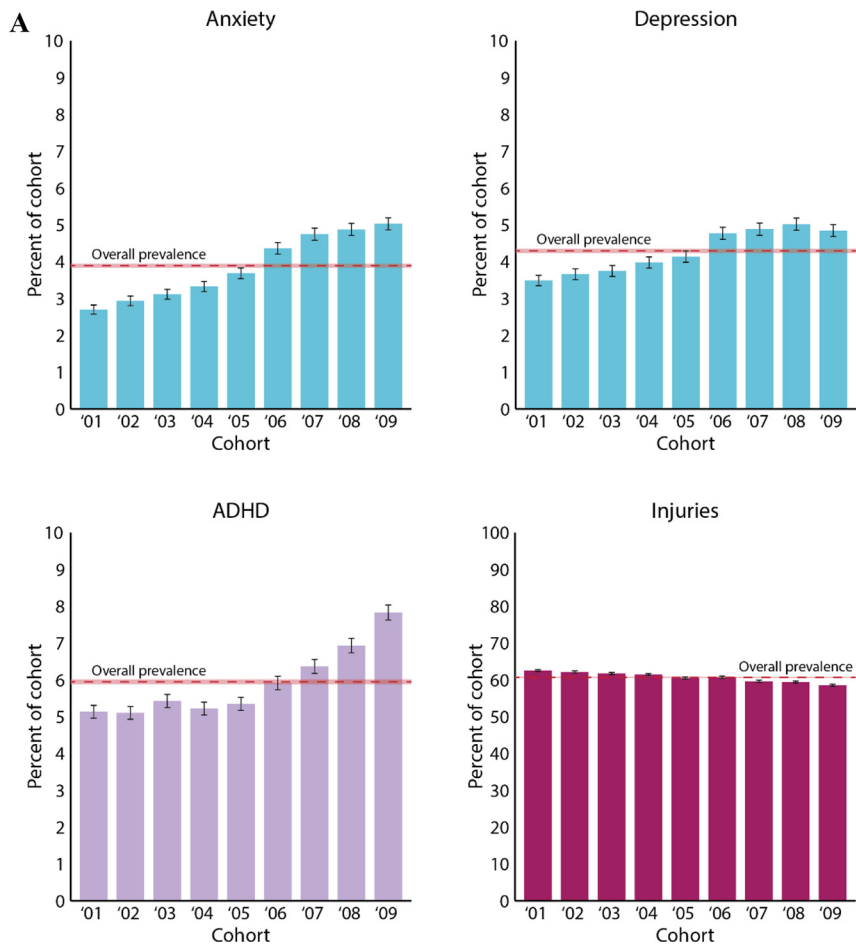
Assortative Mating

With notable exceptions (Border et al., 2022), studies of assortative mating typically focus on one disorder at a time. We documented that assortment was not specific to particular diagnostic categories, but we did not delve into transdiagnostic mechanisms (South, 2026).

It is unlikely that each mental disorder exists as a discrete etiological entity that only becomes correlated with other mental disorders after generations of assortative mating. Different disorders have too many shared risk factors for this to be the case. Indeed, assortment for mental disorders is often found to be indirect—based on related traits but not the observed diagnoses themselves (Torvik et al., 2024). This can lead to an entwining of all causal factors behind the traits that determine mate choice. In addition, gender differences in the expression of psychopathology and in partner preferences (Harper & Zietsch, 2025) suggest that partners may have different disorders even if they share similar risk factors. Moreover, diagnostic shifts within individuals imply that couples who have one combination of disorders at one point in time are at higher risk for all other combinations of disorders at later points in time. The partners in a couple remain the same, but the disorders they share shift. This also means that children rarely grow up with parents whose diagnostic statuses are stable throughout their entire childhood.

The implications of cross-disorder assortative mating for offspring are far-ranging, as assortative mating leads to a more unequal distribution of risk factors, passed on from both the mother and the father. This includes higher variance in *p* and greater covariance between different risk factors. Higher variance

Figure 1
Young People Born in More Recent Years Experience Distinct Mental-Health Trajectories



(Figure continues)

implies that there will be more offspring with very low or with very high values of p . An increase in variance most likely takes place when the degree of assortment is increasing, as has been shown for educational attainment and socioeconomic status (Sunde et al., 2024). However, it is uncertain how this weighs against reduced fertility among individuals who have mental disorders (Kravdal et al., 2025) and, presumably, higher p .

A particular case of correlated risk factors is covariance between the genetic and environmental components of multiple disorders (rGE). Such covariance means that the assumption of no gene-environment correlation, usually applied in genetic studies, does not hold. It also means that children who inherit a genetic risk for one disorder (e.g., depression) are more likely to live in environments created by parents with alcohol use disorder, OCD, and so forth. It is not straightforward to determine for which specific disorder these children are at risk. Instead, the risk constellation may lead to multiple, diverse, and unpredictable outcomes. Assortative mating therefore implies that p cuts not only across diagnostic boundaries but also across the nature-nurture divide.

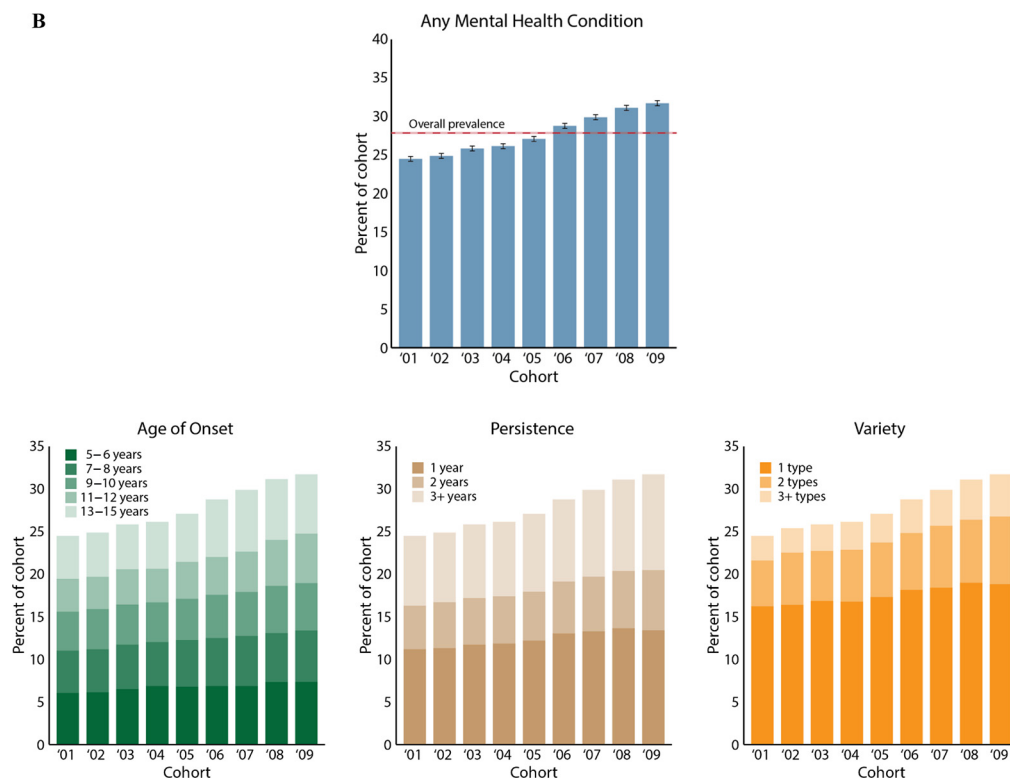
Cross-disorder assortative mating and the familial clustering of different mental disorders also draw attention to the need to bridge youth and adult treatments, with the potential to disrupt the familial

clustering of diverse types of psychopathology. For instance, there is encouraging evidence for transgenerational benefits of emotion regulation-focused interventions in parents (Zalewski et al., 2026), and both parents and their children may benefit from implementing similar skills to manage distressing symptoms (Ehrenreich-May & Milgram, 2026). However, many testable questions remain concerning which transdiagnostic approaches in youth and family therapy may work best and will be most acceptable to providers, as well as about potential limits to these approaches when specialized care may be needed (Ehrenreich-May & Milgram, 2026).

Should Our Article Have Provoked More Pushback?

Given that the idea of p has raised hackles (see Plutynski, 2026; for a philosophical perspective on the debate), it may surprise that the commentaries were not more critical. Why? One possibility relates to the division between the two audiences of this journal: psychopathologists and clinical scientists. psychopathologists, who focus on the measurement and causes of symptoms of mental disorder, have been debating whether p is a substantive construct, an index of overall mental illness, or a measurement or statistical artifact. In contrast, clinical scientists, who seek to develop effective

Figure 1 (continued)



Note. We observed the mental health of nine 1-year birth cohorts of children born between 2001 and 2009, following them for 10 years from ages 5 to 15 years. The sample sizes in each cohort range from 52,734 to 58,772. Panel A shows that both internalizing (e.g., anxiety, depression) and externalizing (e.g., ADHD) conditions have increased among youth. Injuries, shown for comparison, have not increased. Panel B shows that the age at first presenting to primary care for mental-health conditions has not become younger among children born more recently; if anything it has increased slightly (e.g., in the earliest cohort the average age was 9.23 years; in the most recent cohort the average age was 9.42 years). But their mental-health conditions have become more persistent, $F(8, 497,646) = 111.99, p < .001$, and more diverse, $F(8, 497,646) = 251.38, p < .001$. See the online article for the color version of this figure.

treatments for mental disorders, have left the skepticism behind. They seem to recognize *p* in their patients and their families.

Our article sought to document the three reasons that clinical scientists see *p* during their work (cross-disorder assortative mating, transdiagnostic intergenerational transmission, shifting disorders over the life course). We aimed to articulate what this means for how psychopathologists carry out their research, but our article did not take a strong stance about what *p* means.

Take athleticism as an example. Most of us would agree that there is such a thing as athleticism. But that does not mean there is a unitary mechanism that explains athleticism. Rather, it is made up of multiple different causes: jumping ability, running speed, oxygen capacity, fine motor control, gross motor control, response time, and balance. Different combinations of these “causes” lead some people to be better at some specific sports (basketball, track and field, gymnastics). And because many of these causes are correlated, people who are excellent in some sports are generally better than other people in all sports. But equally, just because athleticism is made up of different (albeit correlated) causes does not mean there is no such thing as athleticism. *p* does not explain co-occurring mental disorders any more than athleticism explains athletic ability. But it does suggest what might be common across

many mental disorders, just like athleticism focuses our attention on what makes for athletic ability. It can also point to interventions with broad mental-health effects, just like improved exercise, rather than any specific physical activity, brings about widespread health benefits.

Raballo et al. (2026) summarize our thesis better than we have: “The *p*-factor, as statistically identified, is not a causal entity but rather the observable trace of a deeper vulnerability architecture, a developmental and social process that unfolds interactively across time and generations” (p. 536). By definition, *p* is transdiagnostic. A unified measurement tool is needed that can assess an individual’s life-course vulnerability to psychopathology (De Los Reyes, 2026). Such a tool would draw attention to what all disorders share and also to what all people share, to a degree. Its elements would likely include at least these components: a *diffuse unpleasant affective state*, often termed neuroticism or negative emotionality; *poor impulse control* over thoughts (e.g., impulsive overgeneralization from negative events) and emotions (e.g., impulsive speech and action in response to experienced emotions); *thought distortion* characterized by reality-distorted and reality-distorting cognitions; *interpersonal impairments*, which are nonspecifically evident across many disorders; and *low cognitive ability* characterized by difficulties with

attention, concentration, memory, processing speed, visual-perceptual processing, and visual-motor coordination. These are not competing explanations or accounts but reinforcing dimensions of psychological dys/function. Also, p is a life-course phenomenon and an intergenerational phenomenon. As such, it draws attention away from efforts to predict which symptoms will emerge and for whom. Instead, it draws attention toward efforts to study symptoms as maladaptive deviations from typical developmental trajectories (Moriarty & Hanson, 2026). In research and treatment, such a functional perspective calls for adopting a developmental framework that focuses on how individuals contend with biosocial imperatives; changing age-graded roles; and unexpected, nonnormative events (Del Giudice, 2026; Hinshaw, 2026). This perspective is also most compatible with transdiagnostic staging models (Buchweitz et al., 2026) that are focused on the evolving nature of mental illness rather than cross-sectional diagnostics.

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