

## COMMENTARY

# Toward Integrative, Multisource, and Multimodal Approaches to Assessing Psychopathology Intergenerationally and Across the Lifespan: Commentary on Caspi et al. (2026)

Andres De Los Reyes

Comprehensive Assessment and Intervention Program, Department of Psychology, University of Maryland

Models of psychopathology contain constructs that cannot be directly observed. Assessing these constructs involves identifying specific behaviors that justifiably serve as “markers” of their presence, thus facilitating their precise and accurate measurement. Examples of these behavioral markers include the destruction of property for conduct disorder, the avoidance of unfamiliar people for social anxiety, and sleeping too much or too little for depression. These and other behavioral markers typify the measurement of both specific mental disorders as well as “p” as a transdiagnostic index of psychopathology. Importantly, researchers commonly implement measurement, methodological, and data-analytic practices that conflict with the data conditions that underlie psychopathology studies. For example, researchers often rely on one source to measure behavioral markers of psychopathology (e.g., self-report), even though 60 years of research indicate that the measurement source robustly dictates the conclusions of psychopathology studies. When researchers measure markers of psychopathology with multiple sources, they often integrate these multisource data using data-analytic strategies that misclassify valid data as measurement error; these misclassifications beget underpowered studies and, by logical extension, replication failures. These issues pertain to psychopathology research more broadly, but they also facilitate interpreting Caspi et al. (2026), and the foundation it sets for studying psychopathology both developmentally and intergenerationally. In this commentary, I highlight measurement, methodological, and data-analytic issues inspired by Caspi et al. and summarize work that supports their careful consideration. Caspi et al.’s findings call for developing integrative, multisource, and multimodal assessments that optimize the validity of psychopathology assessments intergenerationally and across the lifespan.

**General Scientific Summary**

This article focuses on steps researchers can take to improve how they assess mental health among youth, adults, and families, to improve the accuracy of mental health studies.

*Keywords:* converging operations, operations triad model, situational specificity, validity

- Do specific diagnostic patterns linked to assortative mating (e.g., both mates diagnosed with depression) pose increased risk for dementia among mates?
- Do disparities in mental health service access exacerbate the familial transmission of mental disorders?
- Are some patterns of sequential comorbidity more heritable than others?

Caspi et al. (2026) opened with questions about the causes of specific mental disorders (e.g., attention-deficit/hyperactivity disorder, depression). I opened this commentary with transdiagnostic questions. What transdiagnostic and disorder-specific questions have in common is that they focus on constructs that cannot be directly observed. Psychopathology researchers leverage measurement, methodological, and data-analytic practices that trace back to astronomy and physics (De Los Reyes, 2024). Physicists cannot “see” gravity, psychopathology researchers cannot see constructs like depression, and thus both rely on indirect markers to measure their constructs. Psychopathology researchers rely on behavioral markers to measure constructs. This is true of diagnostic criteria (American Psychiatric Association, 2013): destruction of property (conduct disorder), avoidance of unfamiliar people (social anxiety), and sleeping too much or too little (depression), for example. This is also true for

Aidan G. C. Wright served as action editor.

Andres De Los Reyes  <https://orcid.org/0000-0003-0214-983X>

Andres De Los Reyes served as lead for conceptualization, writing—original draft, and writing—review and editing.

Correspondence concerning this article should be addressed to Andres De Los Reyes, Comprehensive Assessment and Intervention Program, Department of Psychology, University of Maryland, Biology/Psychology Building, Room 3123H, College Park, MD 20742, United States. Email: [adlr@umd.edu](mailto:adlr@umd.edu)

transdiagnostic constructs: A recent study relied on parent-reported behavioral checklists to index “p,” for instance (Hill et al., 2025). Caspi et al.’s (2026) findings have broad implications for psychopathology research, signaling that psychopathology research “requires measurement strategies that explicitly incorporate familial and developmental information” (p. 486). This commentary focuses on reforms to measurement, methodological, and data-analytic practices that will facilitate researchers’ efforts to build from the work of Caspi et al.

### Detecting Valid Data in the Discrepant Results Produced by Psychopathology Assessments

Caspi et al.’s (2026) assortative mating findings reveal the interactive nature of psychopathology between adult partners. The modal study of adults relies on self-reports to assess psychopathology (De Los Reyes, 2024). Yet, in areas of psychopathology research where multisource data are common (e.g., parent, teacher, and youth reports to assess youth), the data source robustly dictates a study’s conclusions. For example, in a 50-year review of controlled trials for youth interventions and across a range of sampling, geographic, client, methodological, and therapy characteristics, the largest moderating factor of therapy effects, by far, was the source of data used to estimate effects (Weisz et al., 2017).

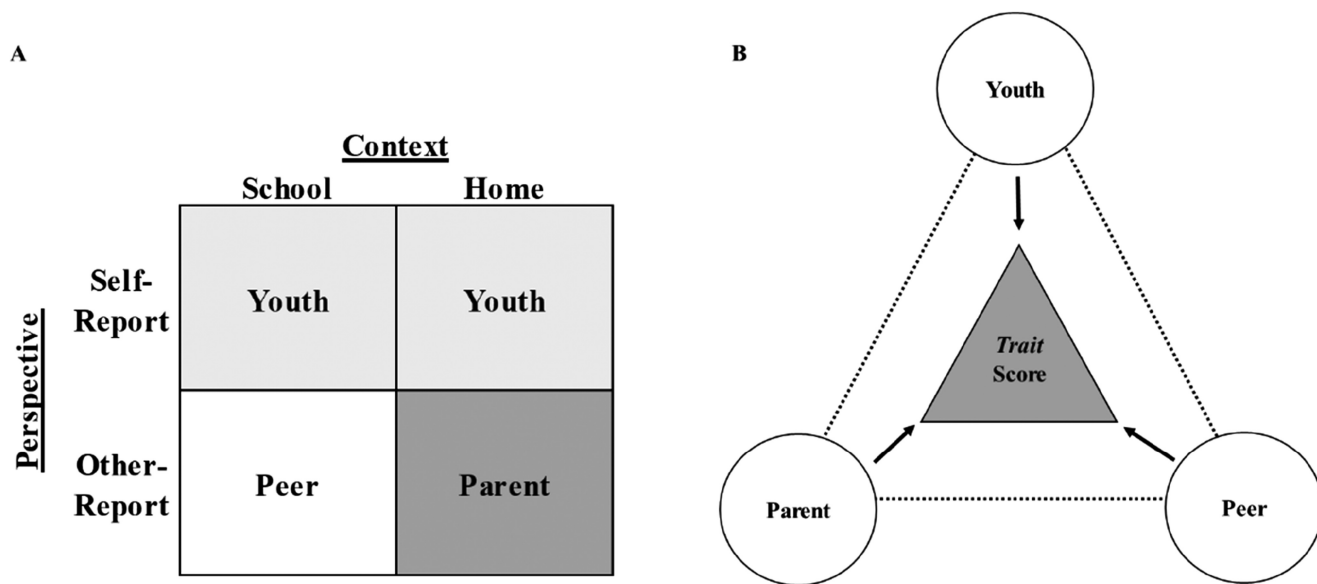
Now, if discrepancies among data sources reflected measurement error and nothing more, addressing these discrepancies might be as simple as collecting data from as many sources as possible. However, when assessing youth psychopathology, discrepancies among sources contain valid data about such domains as the contexts

in which youth experience psychopathology (e.g., school vs. home), therapy outcomes, risk for substance use, family functioning, and physical health (De Los Reyes, 2024). These discrepancies tell us more about those undergoing evaluation than just the additive contribution of sources involved in the evaluation. Beyond the sum of parents’ and teachers’ observations of youth, discrepancies between their observations reveal how youth behavior varies within and across social contexts (De Los Reyes et al., 2023). As psychological phenomena, discrepancies among data sources are likely as interactive and rich in information as other psychopathology-relevant processes, including assortative mating. Researchers who study psychopathology at any point in the lifespan should capitalize on valid data wherever it can be found, and this requires taking a multisource approach to assessing psychopathology (De Los Reyes et al., 2022).

### Integrating Discrepant Results Produced by Psychopathology Assessments

If multisource approaches to assessment produce discrepant results about psychopathology, then these discrepancies necessitate integrating data. Figure 1 depicts one integration strategy—the Satellite Model—delineated decades ago by Kraemer et al. (2003). The Satellite Model is grounded in the notion that—contrary to what many researchers to this day believe—discrepancies among sources are the path to optimizing measurement validity. When researchers choose their sources strategically, the discrepant data produced by these sources complement one another, like the positioning of satellite arrays in geographic space. Studies about familial

**Figure 1**  
Graphical Depiction of the Satellite Model Approach to Integrating Multi-Informant Data



*Note.* (A) An example of the “mix-and-match” criterion to identify optimal informants to include in a multi-informant assessment. Informants systematically vary in the perspective and context from which they rate youth behavior, with the goal of effectively triangulating on a Trait score. (B) A graphical depiction of multi-informant reports triangulating, much like the global positioning system, to identify the Trait score. Both peer and parent reports provide information from an other-perspective, with peers providing information about the school context and parents providing information about the home context. Youth reports provide the self-perspective and information about both the school and home contexts. Reproduced from *Youth Development in Context: Integrating Multiple Informants to Assess Behavior* (Figure 3.1, p. 30), by B. A. Makol, J. Yang, M. Wang, and A. De Los Reyes, 2025, Springer (<https://doi.org/10.1007/978-3-031-80549-3>). Copyright 2025 by Springer Nature.

This document is copyrighted by the American Psychological Association or one of its allied publishers. This article is intended solely for the personal use of the individual user and is not to be disseminated broadly. All rights, including for text and data mining, AI training, and similar technologies, are reserved.

risk of psychopathology commonly rely on multisource approaches to assessment. Yet, recent studies in youth psychopathology have shed light on data-analytic practices that often reduce the explanatory power of these studies.

Kraemer et al.'s (2003) Satellite Model has been underappreciated as a strategy; it is not in the mainstream of integration strategies like more rudimentary strategies (e.g., composite scoring, combinational algorithms, picking a primary outcome) or even more sophisticated ones (e.g., latent models focused on estimating common variance; see De Los Reyes, 2024). Yet, when discrepancies among sources contain valid data, the Satellite Model explains more about psychopathology outcomes than these commonly used strategies (Makol et al., 2025). Taking a simple average of scores or constructing a latent model to attain a pristine estimate of common variance requires assuming that commonalities among sources contain all the valid data. Kraemer et al.'s model requires users to hold a different assumption: Valid data might reside in sources' commonalities, but also in what makes each source unique. Beyond the use of multiple sources, researchers must also consider whether their data-analytic practices allow them to capitalize on all the valid data produced by multisource assessments.

### Constructing Integrative, Multisource, and Multimodal Assessments Across the Lifespan

When used to integrate multisource survey data, the integrated scores produced by the Satellite Model do not just explain more variance in psychopathology outcomes than commonly used integration strategies; they also boost the explanatory power of survey data. For instance, when used to integrate multisource surveys about adolescent social anxiety, scores produced by the Satellite Model predict independent observers' ratings of anxiety behaviors at correlation magnitudes approaching .70 (Makol et al., 2025). Researchers have invested over 60 years of work seeking to understand the discrepant results produced when assessing youth psychopathology, and thus we know that survey data rarely predict observed behavior at correlation magnitudes beyond .10 or .20 (De Los Reyes, 2024). The kinds of multisource, multimodal, and integrative assessments used to assess youth are rare in adult psychopathology studies. Part of their rarity has to do with the social contexts that distinguish adults from youth. Youth in much of the world live in the kinds of social environments that are conducive to soliciting reports from psychometrically sound sources (e.g., parent at home and teacher at school). In contrast, adults vary in their social environments to such a degree that there does not exist a "universal" infrastructure for strategically soliciting data sources.

A disparity exists in the use of multisource, multimodal, and integrative approaches to assessing adults versus youth. This disparity translates to researchers' abilities to optimize the validity of assessments of youth psychopathology to a greater degree than adult psychopathology assessments. If assessments earlier in life achieve levels of measurement validity that outperform assessments later in life, then the measurement validity of assessments decreases from one generation to the next. This introduces methodological confounds in intergenerational work: Greater measurement validity means greater statistical power to understand intergenerational

effects as they manifest earlier in life relative to later in life. In terms of replicability, this should raise cause for concern: large-magnitude effects are more likely to replicate than low-magnitude effects (Open Science Collaboration, 2015). We must invest in the development of assessment approaches that can be adapted across multiple periods of development, types of families, data sources, and social contexts. Decades of work in youth psychopathology reveal an approach to building these assessments, and we are overdue for extending this approach across the lifespan.

### References

- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). <https://doi.org/10.1176/appi.books.9780890425596>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding "p." *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- De Los Reyes, A. (2024). *Discrepant results in mental health research: What they mean, why they matter, and how they inform scientific practices*. Oxford. <https://doi.org/10.1093/oso/9780197686607.001.0001>
- De Los Reyes, A., Talbott, E., Power, T., Michel, J., Cook, C. R., Racz, S. J., & Fitzpatrick, O. (2022). The Needs-to-Goals Gap: How informant discrepancies in youth mental health assessments impact service delivery. *Clinical Psychology Review*, 92, Article 102114. <https://doi.org/10.1016/j.cpr.2021.102114>
- De Los Reyes, A., Wang, M., Lerner, M. D., Makol, B. A., Fitzpatrick, O., & Weisz, J. R. (2023). The Operations Triad Model and youth mental health assessments: Catalyzing a paradigm shift in measurement validation. *Journal of Clinical Child and Adolescent Psychology*, 52(1), 19–54. <https://doi.org/10.1080/15374416.2022.2111684>
- Hill, E. D., Kashyap, P., Raffanello, E., Wang, Y., Moffitt, T. E., Caspi, A., Engelhard, M., & Posner, J. (2025). Prediction of mental health risk in adolescents. *Nature Medicine*, 31(6), 1840–1846. <https://doi.org/10.1038/s41591-025-03560-7>
- Kraemer, H. C., Measelle, J. R., Ablow, J. C., Essex, M. J., Boyce, W. T., & Kupfer, D. J. (2003). A new approach to integrating data from multiple informants in psychiatric assessment and research: Mixing and matching contexts and perspectives. *American Journal of Psychiatry*, 160(9), 1566–1577. <https://doi.org/10.1176/appi.ajp.160.9.1566>
- Makol, B. A., Yang, J., Wang, M., & De Los Reyes, A. (2025). *Youth development in context: Integrating multiple informants to assess behavior*. Springer. <https://doi.org/10.1007/978-3-031-80549-3>
- Open Science Collaboration. (2015). Estimating the reproducibility of psychological science. *Science*, 349(6251), Article aac4716. <https://doi.org/10.1126/science.aac4716>
- Weisz, J. R., Kuppens, S., Ng, M. Y., Eckshtain, D., Ugueto, A. M., Vaughn-Coaxum, R., Jensen-Doss, A., Hawley, K. M., Krumholz Marchette, L. S., Chu, B. C., Weersing, V. R., & Fordwood, S. R. (2017). What five decades of research tells us about the effects of youth psychological therapy: A multilevel meta-analysis and implications for science and practice. *American Psychologist*, 72(2), 79–117. <https://doi.org/10.1037/a0040360>

Received October 15, 2025

Accepted October 16, 2025 ■

## COMMENTARY

Shared Transdiagnostic Risks Are Universal, Dynamic, and Connected:  
Commentary on Caspi et al. (2026)

Warren Mansell

School of Population Health, Curtin enAble Institute, Curtin University

The systematic, large-scale analysis of the development and intergenerational transmission of mental health disorders as transdiagnostic adds to 2 decades of evidence that continues to be out of sync with clinical practice, research, and mainstream culture. These findings need to be combined with evidence for the single continuum between well-being and mental health, and an accessible unified biopsychosocial framework, to inform codesigned, community-grounded interventions.

**General Scientific Summary**

The general population has a measurable vulnerability to a mental health disorder. Conversely, robust data on eventual recovery exists for a proportion of individuals with each of the major adult mental health disorders. The scientific objective should therefore be to identify, understand, harness and scale the universal catalysts for mental health disorder prevention and recovery that already exist.

*Keywords:* adidiagnostic, community mental health, ecological, psychiatric services, precision medicine

The target article (Caspi et al., 2026) supports at least 2 decades of earlier research with a detailed analysis of extensive longitudinal developmental and intergenerational transmission to confirm that the core components of chronic psychological distress are transdiagnostic. The authors describe recommendations that are long overdue, including research methods that would enable greater replicability, and interventions that would be more efficient and scalable. The findings particularly underline how children and families should be a focus of investment in mental health support.

My concern with regard to this research and its potential interpretation is the lack of a sufficiently wide scope such that the continuity between clinical, subclinical, at-risk, and general population instances of apparent mental health symptoms could be assessed. There is possibly a concern within the academic and clinical worlds that highlighting this continuity could either pathologize “normal” behavior (Kardefelt-Winther et al., 2017) or “normalize” mental health disorders (Chappell & Jeppsson, 2023); this

is seen as a particular concern if the institutions involved assume that interventions are necessarily pharmaceutical (Kinderman, 2014). Yet, either view would miss the dynamic, emergent interplay between everyday experiences, at-risk states, near misses, mental health crises, remission, relapse, recovery, and return to community life, that this research indicates occurs within individuals over time, and across generations.

Thus, the target article (Caspi et al., 2026) affirms my own team’s research and development of psychosocial “catalysts” that support—both self-driven and community—mental health recovery and everyday well-being (Mansell et al., 2025). Just as studying the same processes in multiple different mental health disorders is often a redundant, resource-inefficient exercise, so too is investing resources in studying the cross-sectional differences between “healthy controls” and diagnosed individuals; the target article points out that well over a third of individuals in their research switch groups from clinical to control, or vice versa, over the period of their study. These findings are consistent with evidence that well-being and mental health are not orthogonal constructs as commonly claimed; they are at least interrelated (Iasiello et al., 2020) and can be regarded as one continuum once methodological issues of measurement acuity, measurement range, and arbitrary differences in wording are accounted for (Johnson & Wood, 2017).

An approach that identifies universal factors provides a connection between the known systemic and transdiagnostic risk factors for mental health problems such as trauma, poverty, and

Aidan G. C. Wright served as an action editor.

Warren Mansell  <https://orcid.org/0000-0002-5697-1784>

Warren Mansell served as lead for conceptualization and writing—original draft.

Correspondence concerning this article should be addressed to Warren Mansell, Curtin enAble Institute, School of Population Health, Curtin University, Bentley Campus, Kent Street, South Perth, Western Australia 6845, Australia. Email: [warren.mansell@curtin.edu.au](mailto:warren.mansell@curtin.edu.au)

chronically competitive environments (Gilbert, 1995)—and the systemic protective and therapeutic factors such as autonomy support, needs provision, play, and supportive conversations that support reflection (Brenning et al., 2022). When combined with codesign and an accessible framework, these elements can inform the development of psychosocial interventions that are accessible, time- and resource-efficient, and adapted to the individual and their culture, without necessarily requiring triaging, greater resource allocation, and possible stigmatization of mental disorder diagnosis (Mansell et al., 2025).

## References

- Brenning, K., Soenens, B., Vansteenkiste, M., De Clercq, B., & Antrop, I. (2022). Emotion regulation as a transdiagnostic risk factor for (non) clinical adolescents' internalizing and externalizing psychopathology: Investigating the intervening role of psychological need experiences. *Child Psychiatry and Human Development*, 53(1), 124–136. <https://doi.org/10.1007/s10578-020-01107-0>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Chappell, Z., & Jeppsson, S. M. (2023). Recovery without normalisation: It's not necessary to be normal, not even in psychiatry. *Clinical Ethics*, 18(3), 298–305. <https://doi.org/10.1177/14777509231165880>
- Gilbert, P. (1995). Biopsychosocial approaches and evolutionary theory as aids to integration in clinical psychology and psychotherapy. *Clinical Psychology and Psychotherapy*, 2(3), 135–156. <https://doi.org/10.1002/cpp.5640020302>
- Iasiello, M., van Agteren, J., & Muir Cochrane, E. (2020). Mental health and/or mental illness: A scoping review of the evidence and implications of the dual-continua model of mental health. *Evidence Base: A Journal of Evidence Reviews in Key Policy Areas*, 2020, 1–45. <https://doi.org/10.21307/eb-2020-001>
- Johnson, J., & Wood, A. M. (2017). Integrating positive and clinical psychology: Viewing human functioning as continua from positive to negative can benefit clinical assessment, interventions and understandings of resilience. *Cognitive Therapy and Research*, 41(3), 335–349. <https://doi.org/10.1007/s10608-015-9728-y>
- Kardefelt-Winther, D., Heeren, A., Schimmenti, A., van Rooij, A., Maurage, P., Carras, M., Edman, J., Blaszczyński, A., Khazaal, Y., & Billieux, J. (2017). How can we conceptualize behavioural addiction without pathologizing common behaviours? *Addiction*, 112(10), 1709–1715. <https://doi.org/10.1111/add.13763>
- Kinderman, P. (2014). *A prescription for psychiatry: Why we need a whole new approach to mental health and wellbeing*. Springer.
- Mansell, W., Abid, M., Wrightson-Hester, A., Bullen, J., Sharbanee, J., Moullin, J. C., Newnham, E., Greenwood, K., & Rock, D. (2025). *Global psychosocial catalysts for mental health: A review of considerations for a contemporary framework for assessment and implementation* [Manuscript Submitted for publication]. School of Population Health, Curtin enAble Institute, Curtin University.

Received September 22, 2025

Accepted September 25, 2025 ■

## COMMENTARY

On the Cross-Generational Expression of Psychiatric Disorders:  
Commentary on Caspi et al. (2026)

Katherine M. Keyes

Department of Epidemiology, Mailman School of Public Health, Columbia University

The set of interwoven analyses across rich data sources by Caspi et al. (2026) are a compelling combination of theory and empirical research that establish new insights on how psychiatric disorder risk is shaped within and across families, and within and across the life course. Caspi et al. underscore that individuals with psychiatric disorders tend to couple, and that disorder risk in their offspring is clustered alongside the phenotypes exhibited by parents, but it is also transdiagnostic across many different disorders. Further, Caspi et al. show that across the life course, the expression of the liability inherited from parents is not only transdiagnostic but abundantly dynamic.

The authors highlight that the utility of the “diagnostic” in “transdiagnostic” is in the eye of the beholder, and that psychiatric symptoms can be grouped together in many different ways, none of which are perfect representations of an underlying group truth. Debates about nosology are not new to the field, of course, and yet Caspi et al. (2026) add new curvature to the discussion, calling for a field realignment and reckoning to move beyond studies of single “disorders” and instead approaching psychopathology research informed by developmental staging, longitudinal information, and familial history. Caspi et al. make a convincing case that the reason that many risk factors (both genetic and environmental) are correlated with many different disorders is that given a specific underlying vulnerability, people will express many different psychopathological symptoms across the life course. In reading this work, I was reminded of the debates in the pages of *Archives of General Psychiatry* in 1980 regarding how psychiatric nosology should be operationalized in field studies of psychiatric epidemiology. The debates are multifold, but to boil down one among many issues, Weissman and Klerman (1980) argued that progress in the field would be advanced through clinical interview schedules that operationalized discrete disorders. Srole and Fischer (1980) argued that clinical diagnoses are appropriate in specialized settings, but had limited utility when assessing general population samples. In a particularly apt section, Srole and Fischer argued that psychiatric diagnoses, as defined by nosologies such as

*Diagnostic and Statistical Manual of Mental Disorders*, third edition, constituted core presentations of psychiatric patients, but that “these cores are ‘clinical pictures’ that are islands in a sea of fluid functional symptom combinations and permutations that overlap and intermingle” (Srole & Fischer, 1980, 1425). Srole and Fischer’s analogy of the symptom sea fits in nicely with Caspi et al.’s analysis of symptom progression and fluid reorganization; within the sea, there are currents, tides, waves, storms that sometimes converge into tempests and other times disperse into barely perceptible swells. While the debates about the validity and utility of psychiatric diagnoses will undoubtedly continue, it is refreshing that we as a field continue to inform these debates through increasingly sophisticated methodological approaches and clear through-lines of developmental theory.

In addition to providing the field with new ways to consider foundational debates, a hallmark of exceptional scholarship is that new puzzles emerge that we can tackle as a field, and Caspi et al. (2026) provide ample new puzzles to untangle. One of these is that while familial transmission of transdiagnostic risk is well documented, which specific symptoms and disorders individuals will express, and at what age, is difficult to predict. One key to understanding how and why particular disorders express themselves at particular ages may lie in analyzing cross-generational variance in disorder and symptom presentation that is beyond the family structure.

Cross-generational patterning of psychiatric symptom presentations has been documented in psychiatric epidemiology for decades. Studies of the Epidemiological Catchment Area data (Wickramaratne et al., 1989), the Midtown Manhattan and Sterling County (Murphy, 1986), among many others, documented that reported occurrence and onsets of mood and anxiety disorders seemed to change frequently between people born in different generation. Such cohort effects are also evident in many modern studies of psychiatric disorder risk, and demonstrate that prevalence of many disorders are reflections of the accumulations of risk factors and effects of societal environments that shift in prevalence over time and exert influence at key developmental ages. While retrospective recall bias is a threat to inference across many of these studies, scholars have generally converged in concluding that the variation across generations in the experience and expression of psychiatric disorder symptoms provides strong evidence for the broader role of the social environment into which each generation is born and develops as shaping population-level risk.

The evident cross-generational changes in disorder risk should force us to consider the entire experience of mental health and wellness as each generation progresses through the life course, as each generation confronts tensions between self and society. The fact

---

Aidan G. C. Wright served as action editor.

The author has no conflicts of interest.

Katherine M. Keyes served as lead for writing—original draft.

Correspondence concerning this article should be addressed to Katherine M. Keyes, Department of Epidemiology, Mailman School of Public Health, Columbia University, 722 West 168th Street, Room 724, New York, NY 10032, United States. Email: [kmk2104@columbia.edu](mailto:kmk2104@columbia.edu)

that most psychiatric disorders change and shift in prevalence across birth cohorts make clear that societal conditions create either fertile or dry ground for certain expressions of underlying transdiagnostic liabilities at certain ages.

Consider for example the evident cohort effects occurring right now among current generations of adolescents and young adults. In the past 15 years, reported symptoms of depression and anxiety have considerably increased among adolescents and adults, both in the United States and in other countries as well (Keyes & Platt, 2024). As adolescent cohorts have aged into young adulthood, prevalence has begun accelerating among young adults as well (Keyes et al., 2024). There are numerous hypotheses underlying these generational changes in mental health symptomatology with at least some empirical evidence, from the effects of social media and new digital technology, to drivers of the onset and timing of puberty, to age-specific manifestations of the effects of changing macroeconomic conditions (Keyes & Platt, 2024). What does not explain the accelerating prevalence among young people is familial risk; it is unlikely that the prevalence of assortative coupling or the virulence of familial transmission has changed. Instead, what is likely is that the social and developmental environment has changed, and that these changes exert their influence in age-specific ways that also interact with underlying liabilities passed down within families.

When we consider how to tackle questions of why this disorder or set of symptoms, in this person, at this age, factors relevant to broader social and developmental environments that manifest as cohort effects may be a relevant frame. Often in psychiatric epidemiology, when we consider the “environment” in gene by environment interaction or correlation, individual-level experiences are measured and analyzed, for example, stressful life events and adverse childhood environments. Operationalizing a research program that integrates interrogation of cohort effects into modern psychiatric research must move beyond conceptualizing “the environment” through individual-level variation, and consider broader societal shifts that influence generations. Bridging Caspi et al.’s (2026) insights on familial transmission with population-level cohort dynamics may be a critical lever to advance our science. Executing such a program of research requires data that spans generations, and within generations, that spans the life course.

Such data sources are increasingly becoming available, as many ongoing cohort studies now follow multiple generations within an across families. Integrating multicohort longitudinal designs with measurement instruments that attend to the underlying liability to experience psychiatric symptoms in ways that unfold in myriad manifestations of specific disorders or sets of symptoms has the highest likelihood of yielding truly new discoveries that we can leverage for prevention and interventions in the years to come.

## References

- Caspi, A., Houts, R. M., Tegner Anker, R. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Keyes, K. M., Kreski, N. T., & Patrick, M. E. (2024). Depressive symptoms in adolescence and young adulthood. *JAMA Network Open*, 7(8), Article e2427748. <https://doi.org/10.1001/jamanetworkopen.2024.27748>
- Keyes, K. M., & Platt, J. M. (2024). Annual Research Review: Sex, gender, and internalizing conditions among adolescents in the 21st century—Trends, causes, consequences. *Journal of Child Psychology and Psychiatry*, 65(4), 384–407. <https://doi.org/10.1111/jcpp.13864>
- Murphy, J. M. (1986). Trends in depression and anxiety: Men and women. *Acta Psychiatrica Scandinavica*, 73(2), 113–127. <https://doi.org/10.1111/j.1600-0447.1986.tb10576.x>
- Srole, L., & Fischer, A. K. (1980). Debate on psychiatric epidemiology. *Archives of General Psychiatry*, 37(12), 1424–1426. <https://doi.org/10.1001/archpsyc.1980.0178025010015>
- Weissman, M. M., & Klerman, G. L. (1980). Debate on psychiatric epidemiology-reply. *Archives of General Psychiatry*, 37(12), 1423–1424. <https://doi.org/10.1001/archpsyc.1980.01780250109014>
- Wickramaratne, P. J., Weissman, M. M., Leaf, P. J., & Holford, T. R. (1989). Age, period and cohort effects on the risk of major depression: Results from five United States communities. *Journal of Clinical Epidemiology*, 42(4), 333–343. [https://doi.org/10.1016/0895-4356\(89\)90038-3](https://doi.org/10.1016/0895-4356(89)90038-3)

Received October 24, 2025

Accepted October 27, 2025 ■

## COMMENTARY

## Multiple Disorders Across Individual Development: The Role of Heterotypic Continuity—Commentary on Caspi et al. (2026)

Stephen P. Hinshaw

Department of Psychology, University of California, Berkeley

Department of Psychiatry and Behavioral Sciences, University of California, San Francisco

The expansive and provocative proclamation of Caspi et al. (2026)—that mental disorders should not be examined in isolation—receives massive support from their thorough, population-based examinations of assortative mating, intergenerational transmission, and longitudinal “expansion” of conditions/disorders over time within individuals. In short, because transdiagnostic/cross-domain manifestations of mental disorders are normative, standard single-condition conceptions of psychopathology may well be myopic, limiting examinations of etiology, mechanisms, and intervention strategies. I focus on the third prong of their argument (development of different conditions over time), based on the ongoing Dunedin birth-cohort study, bringing into play the construct of heterotypic continuity. This term signifies that predictability and stability pertain not to surface behaviors/diagnoses but to deeper, transdiagnostic proclivities yielding different “phenotypes” over time—casting doubt over the contention that successive, sequential comorbidities of independent conditions are at play. Still, the nature of such proclivities (particularly underlying reasons for the displayed behaviors) remains elusive; theory must be invoked in arguments for heterotypic continuity. Heritable risk, a myriad of independent and dependent life experiences, their complex transactions, and individuals’ interpretations of trauma and protection are relevant to the search for heterotypic continuity. Urgent need exists for multidisciplinary work in clinical science and public health undergirded by advances in multilevel, longitudinal investigations, incorporating theory regarding underlying functions of different behavioral displays across development.

**General Scientific Summary**

Individuals with mental disorders often proceed to display different conditions over time. This commentary addresses the crucial question as to “why” by discussing the concept of heterotypic continuity, through which later conditions are related to earlier conditions via common functions or purposes. Without consideration of heterotypic continuity, progressions to subsequent disorders might simply be viewed as random. Theory is therefore needed to help understand progressions.

*Keywords:* multiple disorders across development, heterotypic continuity, theory

I laud the threefold synthesis of Caspi et al. (2026), positing that an exclusive focus on single manifestations of mental disorder is myopic and often misguided. Indeed, such a focus limits the search for heterogeneous outcomes that transcend usual conceptions of disorder and comorbidity—and belies the wide transdiagnostic heterogeneity of risk regarding individual developmental trajectories.

Herein, beyond assortative mating and intergenerational transmission, I focus on the third platform of their arguments, featuring data from the Dunedin birth cohort. Specifically, from childhood and adolescence through adulthood, single point-in-time mental conditions eventuate into variegated outcomes, often widely so. This phenomenon may signify the developmental psychopathology (DP) principle of heterotypic continuity, mentioned only in passing in the target article. Elaboration and clarification of this construct could facilitate understanding of “beyond single disorder” mechanisms.

Aidan G. C. Wright served as action editor.

Stephen P. Hinshaw  <https://orcid.org/0000-0001-6497-1082>

Stephen P. Hinshaw served as lead for conceptualization, writing—original draft, and writing—review and editing.

Correspondence concerning this article should be addressed to Stephen P. Hinshaw, Department of Psychology, University of California, Berkeley, 2121 Berkeley Way, 3rd Floor, Berkeley, CA 94720-1650, United States. Email: [hinshaw@berkeley.edu](mailto:hinshaw@berkeley.edu)

**Continuity in Development (but of What)?**

In a classic chapter on continuity in human development, both typical and atypical, Kagan (1969) posited a model distinguishing the form (morphology; specific type) of behavior from its function (i.e., underlying reasons). In short, at different developmental periods,

what appear to be identical behavioral manifestations may betray different functions, purposes, or reasons. For example, intense negative affect in infancy (e.g., extreme crying) may emanate from a need for comfort but by middle childhood be linked more strongly to coping with high anxiety. Conversely, apparently different behavioral displays across development may belie parallel functions/reasons, in what has become widely discussed as heterotypic continuity.

In a compelling synthesis, [Petersen \(2024\)](#) highlights a different example from [Kagan \(1969\)](#): High rates of midchildhood tantrums in certain girls predict, by adolescence, strong high school identification and low dependency needs. Why? These apparently different behavioral patterns may signal a broader and deeper propensity: Avoidance of traditional sex-role status expressed quite differently at different developmental stages. Considerations of heterotypic continuity have broadened the search for predictability of behavior and affect from, in my terms, identical manifestations to the discovery of meaningful stability and continuity driven by conceptual relatedness. As DP took hold (see [Cicchetti, 1984](#)), heterotypic continuity became a core tenet (see [Hinshaw, 2017](#), for an overview of DP principles, also including normal/atypical interchange, transactional models, multiple levels of analysis, and constructs of equifinality/multifinality).

An often-cited example of heterotypic continuity exists in the domain of externalizing behavior, whereby early childhood oppositionality can transform into fighting, delinquency, and partner violence by adolescence and adulthood (i.e., different and more severe forms of an externalizing “trait”). In terms of outcomes of single mental disorders at later timepoints (see [Speranza et al., 2023](#), for a narrative review), considerable evidence has emerged for a wide range of diverging conditions—sometimes moderated by participant sex and, in their view, partially explained by attachment patterns. Examining the Dunedin participants, [Caspi et al. \(2026\)](#) found a strong likelihood of divergent outcomes across development. Indeed, cross-sectional examination of a current condition can lead to hugely misguided predictions of future disorder(s).

### Closer Examination

Yet such careful description begs the question as to why. In his masterful exposition, [Petersen \(2024\)](#) sets a high bar for deciding whether and when heterotypic continuity should be invoked. In a 2 (form) × 2 (reason/function) model, (a) similar/identical forms of behavior across time undergirded by similar reasons/functions signify homotypic continuity; (b) similar forms (e.g., crying, in the example above) linked with different reasons/functions (e.g., expressing hunger vs. fending off anxiety) exemplify phenotypic continuity; (c) different behavioral forms emanating from the same core reasons/functions indicate heterotypic continuity; and (d) different forms relating to different reasons/functions signal discontinuity. Without comprehending the underlying reason or function, what may simply be a predictive association (i.e., early depression precedes later aggressive behavior) could be branded as heterotypic continuity. In other words, theory must be invoked regarding what constitutes a heterotypically continuous process ([Kent et al., 2023](#); [Watts et al., 2024](#)).

### Girls With Attention-Deficit/Hyperactivity Disorder

Our research team has prospectively followed a cohort of preadolescent girls with attention-deficit/hyperactivity disorder

(ADHD), along with a group-matched neurotypical comparison sample, for a quarter century. In brief, by adulthood, a substantial subset of the ADHD sample exhibited unplanned pregnancy, intimate partner violence, depression, and self-inflicted injury (non-suicidal self-injury; attempted/completed suicide). This pattern diverges from that of most boys with ADHD, who often display antisocial behavior and substance abuse. The question remains: Did our female participants traverse separable, comorbid outcomes/impairments across development? Or did early ADHD—poorly regulated attention, difficulties in response inhibition and other executive functions, plus associated family strife and peer conflict—foreshadow a path emanating in the devastating blend of internalizing and externalizing characteristics epitomized by self-harm, especially attempted suicide?<sup>1</sup> Pertinent here is the concept of ontogenic, cascading processes involving heritable propensity transacting with poorly fitting or even toxic environments ([Beauchaine & McNulty, 2013](#)). Still, what might be the reason or function underlying such different cross-time presentations?

For a girl with the neurodevelopmental, strongly heritable dimensions of extreme inattention and impulsivity, perhaps the complex underlying reason might be as follows, in her words: “I’m the girl who never really fit but didn’t know why: My family never ‘got’ me, teachers thought I was lazy, and other girls pushed me away because I interrupted so much and couldn’t follow their meanings. I was different, now showing that I’m truly different by hanging with losers, letting guys take advantage of me, and cutting myself to relieve the pain I sense. Feeling more and more hopeless, I ask: Why go on?”

In more academic, third-person language: For girls, overall sex-role expectations to be nurturing, competitive, and effortlessly sexual (encountering a “triple bind”) elicit reactions such as learned helplessness and pursuit of false selves, presaging ever-escalating rates of internalizing and externalizing behaviors this century ([Hinshaw & Kranz, 2009](#)). When (often inexplicable) ADHD compromises self-regulation, self-blame and self-loathing mount, fueling high despair and rage at perceived failures. In short, fear of nonadherence to idealized female norms predicts internalization of blame, transmogrifying ADHD symptoms (whether they remit) into self-destruction by adolescence and emerging adulthood.

### Underlying Propensity?

Is there a core underlying propensity or trait propelling differentiated outcomes from any single condition? [Caspi et al. \(2026\)](#) posit the ubiquitous, pluripotent “p-factor.” [Watts et al. \(2024\)](#), however, provide a major critique on both psychometric and conceptual grounds, arguing that an often statistically inflated general factor/principal component of myriad behaviors is more descriptive than causal. The key question remains: How are heritable vulnerabilities shaped by (largely) nonshared micro- and macrocontexts, including meanings ascribed to multiple life events, to produce impairment and suffering that differ in form (but perhaps with similar underlying reasons/functions) from earlier manifestations?

<sup>1</sup> Crucially, experiences of maltreatment (physical abuse, sexual abuse, and/or neglect) greatly intensified such risk for the ADHD sample ([Hinshaw, Porter, & Ahmad, 2024](#)), even with stringent covariation of socioeconomic and prenatal/perinatal variables, early out-of-home placement, and childhood depression/anxiety and aggression (adjusted *OR* = 3.1).

## Conclusion

The massive demonstration of multiple, extremely diverse behavioral and emotional conditions across development (Caspi et al., 2026) provides an essential counterpoint to single-disorder conceptions. Beyond description, research must contend with processes that portend phenotypically different manifestations of underlying functions. Heterotypic continuity clearly involves gene–environment interplay, cultural scaffolding of healthy versus unhealthy outcomes, and individual interpretations of life experiences. Rigorous theory (see Kent et al., 2023) must continue to undergird the search for what constitutes true heterotypic continuity, all too often linked with impairment and suffering.

## References

- Beauchaine, T. P., & McNulty, T. (2013). Comorbidities and continuities as ontogenic processes: Toward a developmental spectrum model of externalizing psychopathology. *Development and Psychopathology*, 25(4 Pt 2), 1505–1528. <https://doi.org/10.1017/S0954579413000746>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Cicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development*, 55(1), 1–7. <https://doi.org/10.2307/1129830>
- Hinshaw, S. P. (2017). Developmental psychopathology as a scientific discipline: A twenty-first century perspective. In T. P. Beauchaine & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (3rd ed., pp. 3–32). Wiley. <https://doi.org/10.1002/9781394258932.ch1>
- Hinshaw, S. P., & Kranz, R. (2009). *The triple bind: Saving our teenage girls from today's pressures*. Ballantine.
- Hinshaw, S. P., Porter, P. A., & Ahmad, S. I. (2024). Developmental psychopathology turns 50: Applying core principles to longitudinal investigation of ADHD in girls and efforts to reduce stigma and discrimination. *Development and Psychopathology*, 36(5), 2570–2584. <https://doi.org/10.1017/S0954579424000981>
- Kagan, J. (1969). The three faces of continuity in human development. In D. A. Goslin (Ed.), *Handbook of socialization theory and research* (pp. 983–1002). Rand McNally.
- Kent, J. S., Markon, K., & MacDonald, A. W., III. (2023). Theories of psychopathology: Introduction to a special section. *Journal of Psychopathology and Clinical Science*, 132(3), 223–227. <https://doi.org/10.1037/abn0000824>
- Petersen, I. T. (2024). Reexamining developmental continuity and discontinuity in the 21st century: Better aligning behaviors, functions, and mechanisms. *Developmental Psychology*, 60(11), 1992–2007. <https://doi.org/10.1037/dev0001657>
- Speranza, A. M., Liotti, M., Spoletini, I., & Fortunato, A. (2023). Heterotypic and homotypic continuity in psychopathology: A narrative review. *Frontiers in Psychology*, 14, Article 1194249. <https://doi.org/10.3389/fpsyg.2023.1194249>
- Watts, A. L., Greene, A. L., Bonifay, W., & Fried, E. I. (2024). A critical evaluation of the p-factor literature. *Nature Reviews Psychology*, 3(2), 108–122. <https://doi.org/10.1038/s44159-023-00260-2>

Received October 30, 2025  
Accepted November 1, 2025 ■

## COMMENTARY

In Praise of Scientific Open Relationships:  
Commentary on Caspi et al. (2026)

K. Paige Harden

Department of Psychology, University of Texas at Austin

This commentary responds to arguments presented in Caspi et al.'s (2026) viewpoint article. I endorse the authors' overall argument but note that exactly how many mental disorders one needs to study at a time will likely depend on the specific research question. I also describe how transdiagnostic research requires strong norms of data and credit sharing.

**General Scientific Summary**

I discuss the benefits of transdiagnostic psychopathology research and the scientific and professional norms necessary to produce such research.

*Keywords:* p factor, genetics, GWAS, externalizing, open science

Caspi et al. (2026) marshal impressive evidence to demonstrate that mental disorders so commonly co-occur—across couples, generations, and lifespans—that the usual practice of conducting research on a single mental disorder should no longer be the norm in clinical psychological science. They recommend that every aspect of psychopathology research, from study design and measurement to publication and clinical translation, be revised accordingly, such that “single-disorder loyalty” is no longer the starting point of the research enterprise. I agree. Research focused on a single diagnosis, or its symptoms, is poorly suited to understanding psychological phenomena that are rarely contained within nosological boundaries.

But, probably like many readers, I am also genuinely interested in some clinical symptoms in some populations and not that interested in others. (Callous and aggressive children? Endlessly fascinating to me. Adults who can't stop worrying? Yawn.) The causes and consequences of mental disorders are rarely specific to individual disorders, but the intrinsic interest necessary to sustain a research program often is quite specific. Is the alternative to an entirely monogamous

intellectual relationship with one's favorite clinical disorder a dilettantish flirtation with every disorder? Or is there a research equivalent of a happy open relationship, in which widening one's attentions beyond the bounds of a single disorder also deepens and enriches one's relationship with the primary intellectual love object? Happily, my experience in psychiatric genetics suggests scientific open relationships can thrive—but they require a commitment to collaboration that is still too rare in clinical psychology.

Psychiatric genetics, as a field, has had some time to grapple with Caspi et al.'s (2026) central point, because it provided foundational evidence, in the form of genetic correlations, that the causes of mental disorders cannot be presumed to be specific to any individual disorder (The Brainstorm Consortium et al., 2018; Bulik-Sullivan et al., 2015). Genetic correlations, unlike ordinary phenotypic correlations, can be estimated for any two phenotypes that have been the focus of a genome-wide association study (GWAS), even if they have never been measured in the same individuals or manifest at opposite ends of the lifespan. Moderate to high genetic correlations among mental disorders have now proven to be ubiquitous: Much of the genetic architecture of any one disorder overlaps, sometimes nearly entirely, with other disorders (Lee et al., 2019; Mallard et al., 2022). Genes are almost never “for” just one thing.

Crucially, research studies that leverage the genetic sharing among mental disorders, rather than ignoring it, can make discoveries that would not have been possible if genes had as much single-disorder loyalty as scientists do. Our work on externalizing spectrum disorders is an instructive example. GWASs of externalizing disorders, such as alcohol use disorder, opioid use disorder, conduct disorder, and attention deficit hyperactivity disorder, have steadily increased in sample size (Deak & Johnson, 2021; Demontis et al., 2023), but the available data for any one disorder are still too small to identify more than a tiny fraction of the specific genes that confer risk for that disorder. In contrast, a multivariate genetic model, which jointly analyzes multiple externalizing-related disorders, behaviors, and personality traits,

Aidan G. C. Wright served as action editor.

K. Paige Harden  <https://orcid.org/0000-0002-1557-6737>

K. Paige Harden is a Faculty Research Associate of the Population Research Center at the University of Texas at Austin, which is supported by Grant P2CHD042849 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD). This work was also supported by Grant R01HD092548 from NICHD and by Grant R01DA050721 from the National Institute on Drug Abuse.

K. Paige Harden served as lead for conceptualization and writing—original draft.

Correspondence concerning this article should be addressed to K. Paige Harden, Department of Psychology, University of Texas at Austin, 108 East Dean Keeton Stop A8000, Austin, TX 78712, United States. Email: [harden@utexas.edu](mailto:harden@utexas.edu)

has substantially more statistical power than any single-disorder study (Dick et al., 2024; Karlsson Linnér et al., 2021). As a result, we can discover many more genes important for all externalizing disorders than were previously discoverable for any one disorder individually—in some cases, up to a hundred times more. Put differently, I would never have learned so much about the genes affecting child conduct problems, the form of psychopathology that I am most interested in, if I had not learned to sometimes ignore conduct problems and play with other data instead (Tanksley et al., 2024).

The astute reader will note that the line of research I just described aims to identify genetic associations multiple externalizing disorders, not with a general factor of all psychopathology (“p”). This decision is grounded in previous research: Although internalizing, externalizing, and thought disorders are indeed genetically correlated with each other, existing evidence suggests that a general factor of psychopathology does not cohere at the level of individual genetic variants (Clapp Sullivan et al., 2024). Thus, I also appreciate the authors’ caveat that “our claim that psychopathology research should stop studying one mental disorder at a time is not tantamount to claiming that mental disorders should only be studied at the broadest level” (Caspi et al., 2026, p. 489). How broad or narrow one’s scientific aperture needs to be is an empirical question, and the answer to it might differ depending on whether one is examining, for example, genetic causes, neurobiological correlates, early attachment experiences, or economic contexts.

Psychiatric genetics been able to produce transdiagnostic research because it has developed strong norms around collaboration, communication, and credit-sharing. Data, results, methods, and code are all shared as widely and openly as possible, and authorship lists can be hundreds of people long, with multiple shared senior authors. These practices, unfortunately, are not the norm in much of clinical psychology. Caspi et al. (2026) recommend that training programs “teach students about the intergenerational and developmental context of all mental disorders” (p. 484), but if we really are to abandon single-disorder loyalty, then more sweeping changes to how we train students are necessary. Too much of the field is dominated by faculty who have defined their entire professional identity around a single diagnosis, who have trained their students primarily to collect new data but not to share it (and certainly not to analyze other people’s data), and who seem as if they would rather crawl across broken glass than share credit with other senior investigators (partly because they would be penalized for tenure and promotion if they did). If the causes and consequences of mental disorders were specific to individual disorders, then perhaps labs that function as cottage industries for small-*N*, single-principal investigator, case-control studies could make substantial scientific progress. As the target article so powerfully demonstrates, however, that is not the reality of the disorders we claim to care about. No longer being in a scientifically monogamous relationship with a single disorder does not mean you need to divorce it entirely, but it does mean you will need to learn to share.

## References

- The Brainstorm Consortium, Anttila, V., Bulik-Sullivan, B., Finucane, H. K., Walters, R. K., Bras, J., Duncan, L., Escott-Price, V., Falcone, G. J., Gornley, P., Malik, R., Patsopoulos, N. A., Ripke, S., Wei, Z., Yu, D., Lee, P. H., Turley, P., Grenier-Boley, B., Chouraki, V., ... Neale, B. M. (2018). Analysis of shared heritability in common disorders of the brain. *Science*, 360(6395), Article eaap8757. <https://doi.org/10.1126/science.aap8757>
- Bulik-Sullivan, B., Finucane, H. K., Anttila, V., Gusev, A., Day, F. R., Loh, P.-R., Duncan, L., Perry, J. R. B., Patterson, N., Robinson, E. B., Daly, M. J., Price, A. L., & Neale, B. M. (2015). An atlas of genetic correlations across human diseases and traits. *Nature Genetics*, 47(11), 1236–1241. <https://doi.org/10.1038/ng.3406>
- Caspi, A., Houts, R. M., Tegner Anker, A.S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Clapp Sullivan, M. L., Schwaba, T., Harden, K. P., Grotzinger, A. D., Nivard, M. G., & Tucker-Drob, E. M. (2024). Beyond the factor indeterminacy problem using genome-wide association data. *Nature Human Behaviour*, 8(2), 205–218. <https://doi.org/10.1038/s41562-023-01789-1>
- Deak, J. D., & Johnson, E. C. (2021). Genetics of substance use disorders: A review. *Psychological Medicine*, 51(13), 2189–2200. <https://doi.org/10.1017/S0033291721000969>
- Demontis, D., Walters, G. B., Athanasiadis, G., Walters, R., Therrien, K., Nielsen, T. T., Farajzadeh, L., Voloudakis, G., Bendl, J., Zeng, B., Zhang, W., Grove, J., Als, T. D., Duan, J., Satterstrom, F. K., Bybjerg-Grauholm, J., Bækved-Hansen, M., Gudmundsson, O. O., Magnusson, S. H., ... Børglum, A. D. (2023). Genome-wide analyses of ADHD identify 27 risk loci, refine the genetic architecture and implicate several cognitive domains. *Nature Genetics*, 55(2), 198–208. <https://doi.org/10.1038/s41588-022-01285-8>
- Dick, D., Poore, H., Barr, P., Williams, C., Tanksley, P., Londono-Correa, D., Courchesne-Krak, N., Ning, Y., Koellinger, P., Waldman, I., Sanchez-Roige, S., Linnér, R. K., Mallard, T., Palmer, A., & Harden, K. P. (2024). New results from the Externalizing Consortium: Multivariate analyses of nearly 4 million individuals identify >1,400 loci underlying psychiatric and substance use disorders characterized by behavioral under-control. *European Neuropsychopharmacology*, 87(Suppl. 1), 95–96. <https://doi.org/10.1016/j.euroneuro.2024.08.198>
- Karlsson Linnér, R., Mallard, T. T., Barr, P. B., Sanchez-Roige, S., Madole, J. W., Driver, M. N., Poore, H. E., de Vlaming, R., Grotzinger, A. D., Tielbeek, J. J., Johnson, E. C., Liu, M., Rosenthal, S. B., Ideker, T., Zhou, H., Kember, R. L., Pasman, J. A., Verweij, K. J. H., Liu, D. J., ... Dick, D. M. (2021). Multivariate analysis of 1.5 million people identifies genetic associations with traits related to self-regulation and addiction. *Nature Neuroscience*, 24(10), 1367–1376. <https://doi.org/10.1038/s41593-021-00908-3>
- Lee, P. H., Anttila, V., Won, H., Feng, Y.-C. A., Rosenthal, J., Zhu, Z., Tucker-Drob, E. M., Nivard, M. G., Grotzinger, A. D., & Posthuma, D. (2019). Genomic relationships, novel loci, and pleiotropic mechanisms across eight psychiatric disorders. *Cell*, 179(7), 1469–1482.e11. <https://doi.org/10.1016/j.cell.2019.11.020>
- Mallard, T. T., Linnér, R. K., Grotzinger, A. D., Sanchez-Roige, S., Seidlitz, J., Okbay, A., de Vlaming, R., Meddens, S. F. W., Bipolar Disorder Working Group of the Psychiatric Genomics Consortium, Palmer, A. A., Davis, L. K., Tucker-Drob, E. M., Kendler, K. S., Keller, M. C., Koellinger, P. D., & Harden, K. P. (2022). Multivariate GWAS of psychiatric disorders and their cardinal symptoms reveal two dimensions of cross-cutting genetic liabilities. *Cell Genomics*, 2(6), Article 100140. <https://doi.org/10.1016/j.xgen.2022.100140>
- Tanksley, P. T., Brislin, S. J., Wertz, J., de Vlaming, R., Courchesne-Krak, N. S., Mallard, T. T., Raffington, L. L., Karlsson Linnér, R., Koellinger, P., Palmer, A. A., Sanchez-Roige, S., Waldman, I. D., Dick, D., Moffitt, T. E., Caspi, A., & Harden, K. P. (2024). Do polygenic indices capture “direct” effects on child externalizing behavior problems? Within-family analyses in two longitudinal birth cohorts. *Clinical Psychological Science*, 13(2), 316–331. <https://doi.org/10.1177/21677026241260260>

Received October 3, 2025  
Accepted October 6, 2025 ■

## COMMENTARY

## Understanding and Transcending “p” Requires a Functional Model of Psychopathology: Commentary on Caspi et al. (2026)

Marco Del Giudice

Department of Life Sciences, University of Trieste

Caspi et al. (2026) masterfully document the extensive overlap between mental disorders, summarized at the broadest level by the general construct of “p.” This overlap seems to leave little room for specificity; moreover, mainstream bottom-up approaches are ill-equipped to address the dual challenge of heterogeneity within diagnostic categories coupled with similarity across the same categories. Here I argue that we can make progress by taking an explicitly functional perspective, and remapping psychopathology onto broad, evolutionarily relevant dimensions of individual variation. I briefly present the fast–slow–defense (FSD) model, a taxonomy of psychopathology based on the evolutionary concept of life history strategies. The FSD model identifies meaningful subtypes within diagnostic categories and reassembles them into broad, functionally distinct spectra with different risk factors and developmental patterns (“lumping via splitting”). In doing so, it simultaneously explains, deconstructs, and goes beyond the descriptive notion of “p,” to provide an explanatory framework rooted in biological function.

**General Scientific Summary**

To overcome current limitations, models of psychopathology must meet the dual challenge of heterogeneity within diagnostic categories coupled with similarity and overlap across the same categories. This comment argues that bottom-up approaches are insufficient; to make progress, we need to take an explicitly functional perspective, as illustrated by the fast–slow–defense model.

*Keywords:* evolutionary psychopathology, life history theory, “p” factor, transdiagnostic models

Caspi et al. (2026) have written a masterful overview of how mental disorders overlap within people and families, both throughout individual lives and across generations. The construct of “p,” originated in factor-analytic models of psychopathology, crystallizes the notion that “all disorders go together” and hence should be studied and understood together.

I absolutely agree with the spirit of this approach, but a crucial question remains: Is there a level of description or a mode of classification that allows one to find true specificity beyond the generalized overlap summarized by “p”? For reasons touched upon in the article, neither purely empirical diagnostic subtypes (derived bottom-up from biomarkers, symptom profiles, etc.) nor the transdiagnostic spectra of the HiTOP (derived bottom-up from correlations among symptoms or diagnoses) seem able to convincingly

answer this question (for critical assessments see, e.g., Del Giudice, 2018; Del Giudice & Haltigan, 2023; Haeffel, Jeronimus, Fisher, et al., 2022; Haeffel, Jeronimus, Kaiser, et al., 2022).

In this commentary, I argue that we can make progress by taking an explicitly functional, top-down perspective on the structure of mental disorders. The idea is to remap psychopathology onto broad, evolutionarily relevant dimensions of individual variation that determine individual patterns of risk for mental disorders, both concurrently and across developmental stages. In previous publications (Del Giudice, 2018; for a quick overview see Del Giudice & Haltigan, 2023), I proposed the fast–slow–defense (FSD) model, which is based on the concept of a fast–slow continuum in life history-related traits. The “core” psychological traits associated with fast and slow life history strategies are impulsivity, present versus future orientation, risk-taking, and sensation seeking; precocious versus delayed onset of sexual desire and sexual debut; restricted versus unrestricted sociosexuality (i.e., the preference for uncommitted sex with multiple partners); sensitivity to sexual/moral disgust; orientation toward long-term mating; stable versus unstable romantic attachments; and exploitative versus cooperative social attitudes. Taken together, these traits paint a contrast between “risky, short term” and “safe, long term” psychologies, both in the domains of mating and social relationships and in the weighting of potential dangers against rewards (see Del Giudice, 2020, 2025).

Aidan G. C. Wright served as action editor.

Marco Del Giudice  <https://orcid.org/0000-0001-8526-1573>

The author has no conflicts of interest to declare.

Marco Del Giudice served as lead for conceptualization, writing—original draft, and writing—review and editing.

Correspondence concerning this article should be addressed to Marco Del Giudice, Department of Life Sciences, University of Trieste, Via Weiss 2, Trieste 34128, Italy. Email: [marco.delgiudice@units.it](mailto:marco.delgiudice@units.it)

The FSD model combines the fast–slow continuum with a dimension of defense activation that captures individual differences in the readiness and intensity of protective psychological mechanisms such as anxiety, fear, panic, and sadness/depression. Heightened defense activation can occur at both the slow and (especially) fast ends of the fast–slow continuum. In total, the model identifies three functional spectra of psychopathology, which simultaneously describe “kinds of disorders” and “kinds of people”: a fast spectrum and slow spectrum that exist in a diametrical reciprocal relation, plus a defense activation spectrum that overlaps with the other two. Individual differences in cognitive ability are also taken into account; low and/or impaired cognitive abilities contribute to the associations between multiple disorders, even if they do not define a distinct pathology spectrum. Of course, different taxonomies can be useful for different purposes; the FSD fully recognizes that this is not the only way to draw meaningful functional distinctions that are relevant to psychopathology (see Del Giudice, 2018).

My goal here is not to describe the specifics and theoretical underpinnings of the FSD model in any detail. Instead, I want to focus on how the model meets what Caspi et al. (2026) aptly describe as the “dual challenge” of heterogeneity within diagnostic categories coupled with similarity and overlap across those same categories. It does so in a unique way, which makes it radically different from alternatives like the HiTOP: Namely, it employs trait constellations related to fast strategies, slow strategies, and defense activation to (a) identify putative functional subtypes within diagnostic categories, and (b) assign those subtypes to the three corresponding spectra. This approach might be called lumping via splitting, because diagnostic categories are subtyped only to be re-assembled into a small number of broad-band spectra—thus reducing the dual challenge to a single, tractable problem.

To give just two examples: eating disorders (EDs) can be split into an impulsive, fast spectrum subtype (F-EDs) that overlaps with cluster B personality disorders; and an overcontrolled, slow spectrum subtype (S-EDs) that overlaps with autistic traits and cluster C personality disorders. While bulimic presentations are relatively more common in fast spectrum EDs, these subtypes are defined by their personality/motivational correlates, rather than by (unstable, nonspecific) symptom profiles. Obsessive–compulsive disorder (OCD) can be similarly split into a defense activation subtype (D-OCD) that is more prevalent in females, centers around themes of harm prevention, and overlaps with other defense activation disorders as well as psychosis; and a male-biased slow spectrum subtype (S-OCD) that is characterized by perfectionism, themes of symmetry/ordering and “just not right experiences,” and overlaps with autism and obsessive–compulsive personality disorder. Germane to the argument developed in Caspi et al.’s (2026) article, conditions in different spectra of the FSD model are also expected to show distinctive developmental patterns, from typical age of onset to remission trajectories (details in Del Giudice, 2018).

It is interesting to consider the association between EDs and OCD in this light. S-OCD is expected to overlap with S-EDs (which are part of the same functional spectrum), but not with F-EDs; however, D-OCD can be associated with both ED subtypes. If one were to ignore the functional subtypes and treat EDs and OCD as unitary categories, they would show moderate overlap, both with one another and with virtually all the other mental disorders (since the association

networks of their subtypes would merge into a mixed, heterogeneous network). Intriguingly, if one considers the data on assortative mating and parent–offspring associations presented by Caspi et al. (2026, Figures 1 and 3), OCD and EDs stand out for their comparatively small associations with other disorders—consistent with the idea that they comprise functionally distinct subtypes with partly different networks of overlap.

The broader and more important point is that the FSD model should not be tested naïvely; despite the diametric nature of the fast–slow continuum, it does not predict a broad pattern of negative associations between standard diagnostic categories, but only between specific conditions or (more often) disorder subtypes. Moreover, the ubiquitous defense activation disorders (a category similar, but not identical, to the internalizing spectrum) overlap with both fast and slow spectrum conditions, while cognitive impairment and other nonspecific factors tend to create positive associations across spectra, partly masking the underlying negative associations.

As a result, the FSD model is fully compatible with the observed comorbidity structure of psychopathology. This can be demonstrated constructively by means of simulation: when the model is used to generate data based on standard diagnostic categories, and those data are analyzed with factor-analytic techniques, the results closely reproduce the structure found in empirical datasets—including two internalizing and externalizing factors as well as a general p factor (Del Giudice, 2016). Crucially, this happens even if the p factor as such is not part of the generating model: in the FSD, “p” emerges from the combination of three largely independent aspects of individual variation—fast life history strategies, heightened defense activation, and reduced cognitive ability (see Del Giudice & Haltigan, 2023). In this sense, the FSD model allows for an even deeper deconstruction (and reconstruction) of “p,” one that goes beyond descriptive patterns of association—rich and informative as they may be—to embrace the challenge and power of functional explanation.

## References

- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Anderssen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Del Giudice, M. (2016). The life history model of psychopathology explains the structure of psychiatric disorders and the emergence of the p factor: A simulation study. *Clinical Psychological Science*, 4(2), 299–311. <https://doi.org/10.1177/2167702615583628>
- Del Giudice, M. (2018). *Evolutionary psychopathology: A unified approach*. Oxford University Press.
- Del Giudice, M. (2020). Rethinking the fast–slow continuum of individual differences. *Evolution and Human Behavior*, 41(6), 536–549. <https://doi.org/10.1016/j.evolhumbehav.2020.05.004>
- Del Giudice, M. (2025). A turning point for the life history approach to individual differences. In S. Kanazawa (Ed.), *Genes, environments, and differential susceptibility: Current topics in evolutionary developmental psychology* (pp. 28–57). Cambridge University Press.
- Del Giudice, M., & Haltigan, J. D. (2023). An integrative evolutionary framework for psychopathology. *Development and Psychopathology*, 35(1), 1–11. <https://doi.org/10.1017/S0954579421000870>
- Haefel, G. J., Jeronimus, B. F., Fisher, A. J., Kaiser, B. N., Weaver, L. J., Vargas, I., Goodson, J. T., Soyster, P. D., & Lu, W. (2022). The

Hierarchical Taxonomy of Psychopathology (HiTOP) is not an improvement over the DSM. *Clinical Psychological Science*, 10(2), 285–290. <https://doi.org/10.1177/21677026211068873>

Haeffel, G. J., Jeronimus, B. F., Kaiser, B. N., Weaver, L. J., Soyster, P. D., Fisher, A. J., Vargas, I., Goodson, J. T., & Lu, W. (2022). Folk classification and factor rotations: Whales, sharks, and the

problems with the Hierarchical Taxonomy of Psychopathology (HiTOP). *Clinical Psychological Science*, 10(2), 259–278. <https://doi.org/10.1177/21677026211002500>

Received October 3, 2025

Accepted October 6, 2025 ■

## COMMENTARY

## The Promise of Transdiagnostic Prevention and Treatment for Youth and Caregiver Psychopathology: Commentary on Caspi et al. (2026)

Jill Ehrenreich-May and Lauren Milgram  
Department of Psychology, University of Miami

The prevalence of mental health concerns among children and adolescents is on the rise, and most youth presenting for treatment exhibit symptoms of more than one diagnostic category. Caspi et al. (2026) present data from multiple population-level data sets to highlight the pathways through which youth inherit transdiagnostic vulnerability for psychopathology from parents and the trajectories this psychopathology can take throughout the lifespan. Given high rates of diagnostic comorbidity both cross-sectionally and over time, it is not surprising that traditional, diagnosis-specific treatments often fall short of meeting the complex mental health needs of such youth and their caregivers. Building on the future directions highlighted by Caspi et al. (2026), this commentary discusses the rationale for and potential benefits of transdiagnostic approaches to youth psychotherapy and general psychopathology prevention. In particular, we describe how these interventions can target cross-cutting psychiatric vulnerabilities to potentially prevent sequential comorbidity, allow for a smooth incorporation of caregivers with disparate mental health concerns into treatment, and facilitate the adoption and scaling of evidence-based practices in mental health systems. We conclude with a call for additional research and training efforts to maximize transdiagnostic intervention effects and their broader implementation.

**General Scientific Summary**

Following from recommendations in Caspi et al. (2026), this commentary describes how transdiagnostic prevention and intervention programs target cross-cutting youth psychiatric vulnerabilities, potentially preventing developmental-sequential comorbidity. Youth-focused transdiagnostic interventions also incorporate caregivers with disparate mental health concerns into treatment and may be scalable models of evidence-based practices suitable for implementation in youth mental health systems.

*Keywords:* prevention, transdiagnostic, child psychopathology, caregiver, treatment

The field of youth mental health care is at a crossroads. The prevalence of psychopathology among children and adolescents was on a troubling rise well before the COVID-19 pandemic exacerbated the youth mental health crisis (Samji et al., 2022). Traditional, diagnosis-specific treatment models, while effective in some cases, often fall short of meeting the complex mental health needs of youth and families. As highlighted by Caspi et al. (2026) and previous works (e.g., Hankin et al., 2016), comorbidity is the norm in youth psychopathology. Consistent with a general psychopathology or p-factor concept, youth presenting for

treatment in routine clinical settings commonly present with more than one mental health concern, and youth's concerns may frequently change over a standard treatment course or the longer term of youth development. Building on future directions highlighted by Caspi et al. (2026), this commentary discusses the rationale for and potential benefits of transdiagnostic therapy for youth and caregivers. We posit that transdiagnostic treatments are emerging not just as promising alternatives to diagnosis-specific treatments but as pragmatic and sensible standards for the delivery of youth psychotherapy and related prevention efforts.

Aidan G. C. Wright served as action editor.

Jill Ehrenreich-May  <https://orcid.org/0000-0001-7436-5393>

Jill Ehrenreich-May receives a royalty from the sales of the Unified Protocols for Transdiagnostic Treatment of Emotional Disorders in Children and Adolescents from Oxford University Press. She also has grant funding related to this intervention from the National Institute of Health, Patient Centered Outcomes Research Institute, Institute of Education Sciences, the Children's Trust, and the Batchelor Foundation. She is also paid for clinical training and implementation support services

related to this intervention by a variety of institutions. No other potential conflicts of interest were reported by the authors.

Jill Ehrenreich-May served as lead for writing–review and editing. Lauren Milgram served in a supporting role for writing–review and editing. Jill Ehrenreich-May and Lauren Milgram contributed equally to writing–original draft.

Correspondence concerning this article should be addressed to Jill Ehrenreich-May, Department of Psychology, University of Miami, Coral Gables, 5665 Ponce de Leon Boulevard, Coral Gables, FL 33146, United States. Email: [j.ehrenreich@miami.edu](mailto:j.ehrenreich@miami.edu)

## The Transdiagnostic Approach to Youth Psychotherapy

The fundamental premise of the transdiagnostic approach to psychotherapy is simple: many seemingly distinct psychiatric disorders share core psychological processes, consistent with the p-factor concept. Internalizing, externalizing, and even certain thought disorders can be conceptualized as emerging from similar emotion regulation challenges, such as cognitive inflexibility, distress intolerance, and use of maladaptive action tendencies in the face of strong emotion. These same challenges may give rise to and maintain a range of psychiatric disorders concurrently or in sequence. Rather than teaching separate skills for each presenting diagnosis, transdiagnostic prevention and treatment models provide a pragmatic and flexible toolkit designed to address these cross-cutting psychiatric vulnerabilities. A growing body of literature indicates that targeting core dysfunctions across mental disorders via evidence-based treatment strategies is a more efficient and effective approach to alleviating mental health distress and preventing the development of further psychopathology later in life (Ghizzoni et al., 2025; Radunz et al., 2025).

### Transdiagnostic Programs Offer Flexibility and Practicality in Real-World Settings

The benefits of a transdiagnostic psychotherapy approach extend beyond efficiency. By shifting the focus from rigid diagnostic categories to the shared mechanisms of youth distress and common elements of effective therapies, transdiagnostic treatments offer clinicians flexibility to tailor intervention content to the symptoms most distressing for a given child or family on a given week without straying from fidelity to the intervention. Transdiagnostic approaches address a significant challenge in the current mental health landscape: the fragmentation of care. If clinicians rigidly adhere to traditional, diagnosis-specific protocols, a patient who presents with multiple problem types may require entirely separate or sequential courses of therapy. Such a process is inefficient and straining to mental health systems, which are currently facing a marked shortage of mental health providers (U.S. Department of Health and Human Services, Bureau of Health Workforce, 2024). Instead of transferring a youth patient from one clinician to another or requiring clinicians to become experts in many different interventions, transdiagnostic approaches provide a unifying framework and evidence-based techniques that can be applied to an array of concerns and a diverse clinical caseload. The transdiagnostic approach can thus aid in the prevention of sequential comorbidities and facilitate the adoption and scaling of evidence-based practices in mental health systems.

### The Bidirectional Relationship Between Youth and Parent Psychopathology and Implications for Transdiagnostic Treatment

While transdiagnostic models have a growing evidence base, more research is needed to refine their application to youth populations. There is a particular need for continued focus on the role of parents and caregivers in shaping the transdiagnostic therapeutic process. Caspi et al.'s (2026) findings on assortative mating and the intergenerational transmission of psychopathology provide a

novel lens to understand what many clinical researchers and practitioners observe: youth with mental health concerns often have parents with mental health concerns. Parental psychopathology confers genetic and environmental risks for youth psychopathology; that notwithstanding, it is important not to dismiss the bidirectional and modifiable learning pathways between the two. As one example, parents who are distressed by their child's distress often accommodate their child's symptoms in ways that relieve distress in the short term but maintain distress in the long term (e.g., allowing the child to avoid distressing situations in part to alleviate the parent's own distress). Parental accommodation behaviors negatively reinforce the parent and can spur and exacerbate youth and parental mental health concerns over time (Birk et al., 2022).

Parents seeking treatment for their children do not always seek or prioritize treatment for their own symptoms, and there is little systemic effort toward identifying caregivers in need, even during times of crisis (e.g., Peris & Ehrenreich-May, 2021). As suggested by Caspi et al. (2026), a thorough assessment of family psychiatric history and current family functioning is critical to inform the provision of youth psychiatric services. A potential benefit of transdiagnostic approaches could be to facilitate the application of treatment skills to both youth and parent symptoms simultaneously, even if youth and parents do not necessarily present with the same diagnosis. For example, youth and their parents may be taught to identify the antecedent, behavior, and consequence of respective symptoms and how these function to reinforce each other's distress (e.g., Double Before, During, and After within the Unified Protocols for Transdiagnostic Treatment of Emotional Disorders in Children and Adolescents; Ehrenreich-May et al., 2017), and youth and parents can be assigned home-learning activities to "try the opposite" (e.g., behavioral activation, exposure, and conflict resolution within the FIRST Program; Weisz et al., 2017) during times of heightened youth or caregiver emotionality.

### Caveats and Future Directions

Transdiagnostic approaches offer many exciting future directions for research and clinical practice; yet the transition to a transdiagnostic paradigm for youth and families is not without its challenges. More needs to be understood regarding clinician preferences for and usage of flexible transdiagnostic interventions and prevention programs. In addition, while transdiagnostic interventions may allow for the flexible integration of parent-directed content into youth psychotherapy, parents experiencing significant distress and psychopathology may not have the resources necessary to engage with parent-directed material (Caspi et al., 2026). Continued research and training efforts are needed to assess the effectiveness of transdiagnostic interventions compared to existing diagnosis-specific treatments for youth and caregivers, optimize predictive algorithms that allow easier clinician navigation through these approaches, and ensure that core treatment principles are implemented effectively in real-world settings. It will be especially important to better understand the limits of certain transdiagnostic interventions or prevention programs for certain clinical presentations that require diagnosis-specific care.

Despite these hurdles, the rising prevalence of comorbidity in youth, the need for more efficient and accessible care, the concurrent presence of caregiver psychopathology, and the growing body of evidence supporting these approaches point to a future where

transdiagnostic treatments may be the new standard for youth and family psychotherapy. By shifting from a focus on discrete, diagnosis-specific symptoms presenting at one moment in time toward a more bird's-eye view of the shared psychological processes that perpetuate mental distress across families and across the lifespan, transdiagnostic interventions offer a holistic path to recovery.

## References

- Birk, S. L., Sung, J. Y., Schleider, J. L., & Olino, T. M. (2022). Unpacking parental accommodation: Relationship to parent distress tolerance and cognitive styles. *Journal of Anxiety Disorders*, *92*, Article 102639. <https://doi.org/10.1016/j.janxdis.2022.102639>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, *135*(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Ehrenreich-May, J., Kennedy, S. M., Sherman, J. A., Bilek, E. L., Buzzella, B. A., Bennett, S. M., & Barlow, D. H. (2017). *Unified protocols for transdiagnostic treatment of emotional disorders in children and adolescents: Therapist guide*. Oxford University Press.
- Ghizzoni, G., Mirandi, M., Garofalo, C., Mazzeschi, C., & Delvecchio, E. (2025). A systematic review comparing four transdiagnostic programmes for school-age children. *Clinical Psychology & Psychotherapy*, *32*(3), Article e70072. <https://doi.org/10.1002/cpp.70072>
- Hankin, B. L., Snyder, H. R., Gulley, L. D., Schweizer, T. H., Bijttebier, P., Nelis, S., Toh, G., & Vasey, M. W. (2016). Understanding comorbidity among internalizing problems: Integrating latent structural models of psychopathology and risk mechanisms. *Development and Psychopathology*, *28*(4, Pt 1), 987–1012. <https://doi.org/10.1017/S0954579416000663>
- Peris, T. S., & Ehrenreich-May, J. (2021). The parents are not alright: A call for caregiver mental health screening during the COVID-19 pandemic. *Journal of the American Academy of Child and Adolescent Psychiatry*, *60*(6), 675–677. <https://doi.org/10.1016/j.jaac.2021.02.007>
- Radunz, M., Johnson, C., Dalgleish, T., Shafran, R., & Wade, T. D. (2025). Transdiagnostic interventions in prediagnostic youth with elevated distress: A meta-analysis of outcomes. *Journal of Consulting and Clinical Psychology*, *93*(9), 627–641. <https://doi.org/10.1037/ccp0000968>
- Samji, H., Wu, J., Ladak, A., Vossen, C., Stewart, E., Dove, N., Long, D., & Snell, G. (2022). Review: Mental health impacts of the COVID-19 pandemic on children and youth—A systematic review. *Child and Adolescent Mental Health*, *27*(2), 173–189. <https://doi.org/10.1111/camh.12501>
- U.S. Department of Health and Human Services, Bureau of Health Workforce. (2024). *State of the behavioral health workforce report*. <https://bhw.hrsa.gov/sites/default/files/bureau-health-workforce/state-of-the-behavioral-health-workforce-report-2024.pdf>
- Weisz, J., Bearman, S. K., Santucci, L. C., & Jensen-Doss, A. (2017). Initial test of a principle-guided approach to transdiagnostic psychotherapy with children and adolescents. *Journal of Clinical Child & Adolescent Psychology*, *46*(1), 44–58. <https://doi.org/10.1080/15374416.2016.1163708>

Received October 21, 2025

Accepted October 22, 2025 ■

## COMMENTARY

The p-Factor and Associations Across Partners, Time, and Generations:  
Commentary on Caspi et al. (2026)

Erik Pettersson

Department of Medical Epidemiology and Biostatistics, Karolinska Institutet

Drawing on both surveys and (primary and specialist care-assigned) register-based diagnoses, Caspi et al. (2026) displayed that the positive manifold among psychiatric conditions is present not only between individuals, but also across partners, time, and generations. To this impressive work, I add three comments.

First, regarding intergenerational transdiagnostic transmission, our research group observed a similar pattern of results in a sample of three million Swedes linked to their parents, and that the results extended to school and labor market performance (Zhou et al., 2024). Furthermore, in a follow-up study where we fit a bifactor model to the parental psychiatric conditions, the general factor of psychopathology was significantly associated with all outcomes in the offspring, whereas the specific factors were primarily associated with similar types of outcomes. The results are summarized in Figure 1, which displays the mean of the odds ratios within each group of outcomes (the associations with the 32 individual outcomes, with corresponding confidence intervals, are available in eFigure 2 of Zhou et al., 2023). These results support Caspi et al.'s (2026) hypothesis that p can account for the transdiagnostic pattern of intergenerational transmission.

Second, regarding transdiagnostic patterns over time, Caspi et al. (2026) argued that most disorders eventually morph into different disorders. Following a review of longitudinal studies of the stability of mental conditions, however, Oldehinkel and Ormel (2023) concluded that whereas individuals with multiple conditions at Time 1 tended to exhibit multiple conditions at Time 2, the cessation of one diagnosis and the initiation of a new diagnosis appeared relatively rare. More recently, a similar pattern was observed in parent-rated symptoms of children in the Adolescent Behavior Cognitive Development (ABCL) study, with few children displaying cessation of a symptom at Time 1 and the emergence of a new symptom at Time 2. Instead,

stability of one (or more symptoms) over time appeared more typical (Applegate & Lahey, 2025). Similarly, when applying a bifactor model to examine the longitudinal stability of broad mental health spectra in adolescents, although each factor was highly stable over 18 months, the cross-factor, cross-time associations were close to the null (Snyder et al., 2017). Thus, one possibility is that among individuals with a low liability toward all mental health problems (i.e., low scores on p), diagnostic switching across broad spectra over time (e.g., from internalizing problems at Time 1 to externalizing problems at Time 2) might be relatively rare.

Nevertheless, the above research is limited in that most of the studies are (a) restricted to relatively brief intervals of a couple of years and (b) focused primarily on internalizing and externalizing conditions. Therefore, it remains unclear if diagnostic switching—after controlling for the p-factor—might be more prevalent when examining stability over decades and when including psychotic conditions.

Third, although the observed transdiagnostic patterns across partners, time, and generations are consistent with an underlying p-factor, so are other models without a p-factor (Caspi & Moffitt, 2018). Notably, this challenge is not unique to the p-factor, but applies to all latent constructs (e.g., the Big Five, general intelligence). Attempts to elucidate, or at least rule out, alternate data-generating mechanisms include incorporating genetic information or comparing observed data to different data-generating mechanisms (Clapp Sullivan et al., 2024; Pettersson, 2023). Nevertheless, considering that empirical economists tend to go to great lengths to identify the causal effect of a single observed exposure on a single observed outcome, attempting a similar feat when dealing with tens or hundreds of observed outcomes (i.e., factor indicators) and multiple unobserved exposures (i.e., latent factors) is bound to be challenging. As an alternate approach, I have argued that there might be nonempirical reasons in favor of measuring a general factor of psychopathology independently of specific factors capturing subsets of symptoms (Pettersson, 2023).

Regardless of how well the p-factor might capture the underlying data-generating mechanism, one possibility is that the current psychiatric nosology, which focuses primarily on specific conditions, might do a disservice to individuals with multiple psychological problems. As an example, in 2018, a Swedish public service investigative journalism television show displayed the challenges facing Sanne, a young woman suffering from problems related to eating, and anxiety, and harmful substance use. Despite her suffering, she was repeatedly rejected by substance treatment centers and referred to psychiatric care centers, and vice versa, based on the notion that she had to recover from her other condition first. Public outrage ensued, leading the Swedish government to appoint a “Comorbidity Investigation.”

---

Aidan G. C. Wright served as action editor.

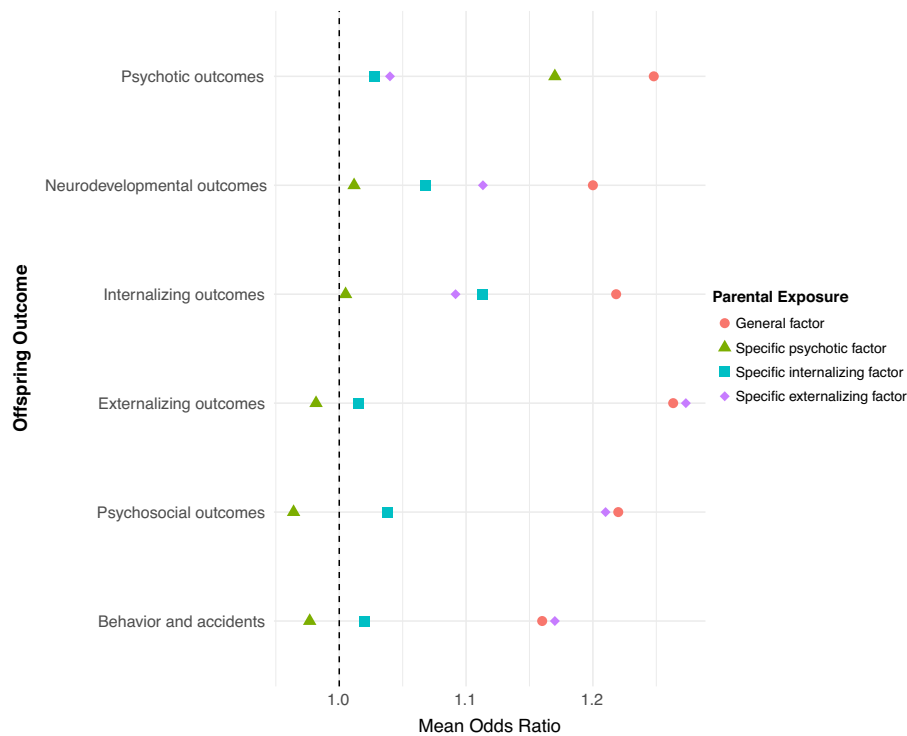
Erik Pettersson  <https://orcid.org/0000-0003-0149-9002>

This work was supported by the Swedish Research Council [Grant 2023–01999], the Swedish Research Council for Health, Working Life and Welfare [Grant STY-2023/0008], and the Brain Foundation [Grant FO2024-0067].

Erik Pettersson served as lead for conceptualization and funding acquisition.

Correspondence concerning this article should be addressed to Erik Pettersson, Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Nobels väg 12A, P.O. Box 281, SE-171 77 Stockholm, Sweden. Email: [erik.pettersson@ki.se](mailto:erik.pettersson@ki.se)

**Figure 1**  
Associations of Parental Bifactor Model With Offspring Outcomes



Note. See the online article for the color version of this figure.

In 2021, it concluded that harmful substance use and psychiatric conditions should be treated by the same caregiver, preventing individuals like Sanne from being repeatedly rejected and referred elsewhere (Printz, 2021). Though speculative, one possibility is that if diagnostic manuals were to include an explicit measure of a liability toward all psychiatric conditions (e.g., a p-factor), then Sanne might have had an easier time accessing care.

## References

- Applegate, B., & Lahey, B. B. (2025). Mapping persistence and change in psychological problems during the transition to adolescence: Adding, subtracting, shifting, and persisting. *JCPP Advances*, Article e70043. Advance online publication. <https://doi.org/10.1002/jcv2.70043>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Caspi, A., & Moffitt, T. E. (2018). All for one and one for all: Mental disorders in one dimension. *American Journal of Psychiatry*, 175(9), 831–844. <https://doi.org/10.1176/appi.ajp.2018.17121383>
- Clapp Sullivan, M. L., Schwaba, T., Harden, K. P., Grotzinger, A. D., Nivard, M. G., & Tucker-Drob, E. M. (2024). Beyond the factor indeterminacy problem using genome-wide association data. *Nature Human Behaviour*, 8(2), 205–218. <https://doi.org/10.1038/s41562-023-01789-1>
- Oldehinkel, A. J., & Ormel, J. (2023). Annual research review: Stability of psychopathology: Lessons learned from longitudinal population surveys. *Journal of Child Psychology and Psychiatry*, 64(4), 489–502. <https://doi.org/10.1111/jcpp.13737>
- Pettersson, E. (2023). Opportunities of measuring hierarchical models of psychopathology. *JCPP Advances*, 3(4), Article e12187. <https://doi.org/10.1002/jcv2.12187>
- Printz, A. (2021). *Från delar till helhet—En reform för samordnade, behovsanpassade och personcentrerade insatser till personer med samsjuklighet* [From parts to whole—A reform for coordinated, needs-adapted and person-centered interventions for people with comorbidities]. Statens Offentliga Utredningar 2021:93. <https://www.regeringen.se/rattsligadokument/statens-offentliga-utredningar/2021/11/sou-202193/>
- Snyder, H., Young, J., & Hankin, B. (2017). Strong homotypic continuity in common psychopathology, internalizing and externalizing specific factors over time in adolescents. *Clinical Psychological Science*, 5(1), 98–110. <https://doi.org/10.1177/2167702616651076>
- Zhou, M., Lageborn, C. T., Sjölander, A., Larsson, H., D’Onofrio, B., Landén, M., Lichtenstein, P., & Pettersson, E. (2024). Psychiatric diagnoses in parents and psychiatric, behavioral, and psychosocial outcomes in their offspring: A Swedish population-based register study. *American Journal of Psychiatry*, 181(8), 761–773. <https://doi.org/10.1176/appi.ajp.20230353>
- Zhou, M., Larsson, H., D’Onofrio, B. M., Landén, M., Lichtenstein, P., & Pettersson, E. (2023). Intergenerational transmission of psychiatric conditions and psychiatric, behavioral, and psychosocial outcomes in offspring. *JAMA Network Open*, 6(12), Article e2348439. <https://doi.org/10.1001/jamanetworkopen.2023.48439>

Received October 22, 2025

Accepted October 22, 2025 ■

## COMMENTARY

# The Value of Focusing on Psychosocial Dysfunction Rather Than Categorical Disorders: Commentary on Caspi et al. (2026)

Emily A. Atkinson<sup>1</sup>, Joshua R. Oltmanns<sup>2</sup>, and Gregory T. Smith<sup>3</sup><sup>1</sup> Department of Psychiatry and Behavioral Neuroscience, The University of Chicago<sup>2</sup> Department of Psychological and Brain Sciences, Washington University in St. Louis<sup>3</sup> Department of Psychology, University of Kentucky

Caspi et al. (2026) conducted an ambitious population-level investigation that provided robust support for assortative mating and the transfer of psychiatric dysfunction across generations. These findings are key to our understanding of the nature and clinical utility of the general factor of psychopathology “p” and support the possibility that “p” reflects a nonspecific index of psychosocial dysfunction, where common dysfunction associated with multiple specific behaviors drives psychiatric comorbidity and cross-generational transmissibility. In addition, we argue the following. First, categorical *Diagnostic and Statistical Manual of Mental Disorders* diagnoses hinder the recognition of these patterns, as they arbitrarily divide shared dysfunction into distinct disorders. Second, recognition of “p” can have direct clinical value. Third, though Caspi et al. report striking odds ratios, these effects should be interpreted as probabilistic, rather than deterministic, and should not be misinterpreted as inevitable outcomes. Fourth, while the population-level scope of this investigation is a major strength, the racial and ethnic homogeneity of the sample may limit global generalizability. The target article provides a critical contribution to psychological science and can facilitate significant improvements in our understanding of psychopathology.

**General Scientific Summary**


This commentary on Caspi et al. (2026) argues that psychosocial dysfunction is a common factor underlying many mental health conditions, contributing to frequent overlap between diagnoses and persistence across generations. It suggests that relying on traditional diagnostic categories may limit our understanding and treatment of mental illness. Focusing on the general factor of psychopathology could help clinicians offer more personalized, effective care that addresses broader patterns of emotional and social impairment.

**Keywords:** psychosocial dysfunction, general factor of psychopathology “p,” dimensional psychopathology, assortative mating

Caspi et al. (2026) have conducted a remarkable, population-level multigenerational investigation aimed at examining the interrelated nature of psychiatric disorders. The findings provide clear support for both assortative mating by the presence of disorder and the transfer of psychiatric dysfunction across generations. These two findings help provide a clearer view of the nature and clinical utility of the general factor of psychopathology “p.” Their work represents an invaluable contribution to the literature with the potential to facilitate profound improvements in our understanding of psychopathology.

We begin with two related reactions to their findings. First, they are consistent with the perspective that “p” represents a nonspecific index of psychosocial dysfunction (Smith et al., 2020). A defining feature of disorder is the presence of life impairment, most typically characterized by some degree of social, emotional, and/or occupational dysfunction. Almost certainly, the relationship between symptoms and dysfunction is reciprocal, such that symptoms lead to dysfunction, which in turn leads to additional or perhaps more pronounced symptoms (Riley & Smith, 2017; Stern et al., 2025). Common forms of dysfunction are shared across disorders, helping

Aidan G. C. Wright served as action editor.

Emily A. Atkinson  <https://orcid.org/0000-0002-9239-6297>

All authors declare no conflicts of interest.

Emily A. Atkinson served as lead for writing—original draft. Joshua R. Oltmanns served in a supporting role for writing—original draft. Gregory

T. Smith served as lead for writing—original draft. Emily A. Atkinson, Joshua R. Oltmanns, and Gregory T. Smith contributed equally to conceptualization.

Correspondence concerning this article should be addressed to Emily A. Atkinson, Department of Psychiatry and Behavioral Neuroscience, The University of Chicago, 5841 South Maryland Avenue, MC 3077, Chicago, IL 60637, United States. Email: [Emily.atkinson@uchicagomedicine.org](mailto:Emily.atkinson@uchicagomedicine.org)

to explain disorder comorbidity and the existence of “p.” The most dysfunctional symptoms of any putative disorder will load on the general factor reflecting what disorders have in common.

From this perspective on “p,” individuals with higher levels of social, emotional, and occupational dysfunction are more likely to associate with, and ultimately mate with, others who also experience elevated levels of dysfunction. For both genetic (e.g., personality traits) and environmental (e.g., social and emotional dysfunction in parenting) reasons, the offspring of such individuals will tend to experience dysfunction and associated life impairment.

Our second reaction is to highlight that psychological dysfunction exists along continua (Kotov et al., 2021; Krueger et al., 2021). The artificial dichotomous chunking of dimensional functioning into present or absent behaviors, and thus present or absent specific *Diagnostic and Statistical Manual of Mental Disorders (DSM)* disorders, inevitably involves the construction of supposedly distinct *DSM* disorders that, in part, represent the same dimensions of dysfunction. Virtually by definition, such disorders will correlate. In part, disorder comorbidity may occur when the same dimensions of dysfunction result in multiple, different sets of maladaptive behaviors. Furthermore, it is not surprising that specific behavioral expressions of dimensions of dysfunction vary over time, contributing to the finding that individuals are often diagnosed with different *DSM* disorders at different times.

One thing that has slowed down the recognition of assortative mating and generational transfer of “p” is the reliance on *DSM* categorical diagnoses, rather than embracing, fully, the shift to dimensions of dysfunction. Had researchers, historically, conceptualized psychopathology as dimensional, it would have made clear sense to hypothesize and test whether both assortative mating and generational transfer were operative. Even in the target article, there are occasional statements made as if *DSM* disorders really are distinct entities from each other. For example, the authors suggest that some individuals’ psychiatric disorders “morph” from one disorder into another over time; this gives the impression that the person has changed in some fundamental or dramatic way. It is very reasonable to doubt that dramatic, morphing changes are frequent. Such doubt has perhaps contributed to skepticism regarding “p” and too little interest in testing assortative mating and generational transfer hypotheses.

We believe that recognition of “p” can have clear, direct value clinically. Imagine two individuals seeking treatment for moderate alcohol use disorder (AUD). One developed AUD in college has no other markers of dysfunction or comorbid psychopathology, and little or no dysfunction in his/her family. The other also developed AUD in college; however, they reported persistent depressed mood and, in their immediate family, a history of substance misuse. In this example, assessing “p” will help the clinician know whether to view the case as uncomplicated addiction or one characterized by elevations in multiple dimensions of dysfunction, thus needing more comprehensive transdiagnostic treatment such as the Unified Protocol for Transdiagnostic Treatment of Emotional Disorders (Barlow et al., 2011).

We next shift to another characteristic of the findings presented by Caspi et al. (2026). The odds ratios, in predicting both assortative mating and intergenerational dysfunction transfer, though quite strong, do not describe anything inevitable. Typical odds ratios presented were in the 2.0 range, which is both quite striking and far from determinative. Typically, it was more likely that dysfunction would

not be transferred across generations than it would be transferred. We want to emphasize that readers should not misconstrue the findings of Caspi et al. to believe, for example, that individuals with dysfunction should be avoided because they somehow inevitably align with others with dysfunction and inevitably produce troubled offspring. That was certainly not the argument that Caspi et al. were making.

Our third comment concerns the nature of the samples studied by Caspi et al. (2026). On the very positive side, their choices enabled them to study virtually whole societies, across the full socioeconomic range. Just as do all sampling choices, even this one has an important limitation. The samples appear to have been largely homogeneous with respect to race and ethnicity, which raises several important questions. Do the findings of this remarkable study apply to individuals of color or from the southern hemisphere? We do not believe that Caspi et al. should have somehow solved this problem and only published with samples representative of the world’s population—such a requirement would be both absurd and hypocritical. We were surprised, though, that no mention was made of this limitation by the authors. A small percentage of the world’s population is White or European. Given this reality, what arguments should be considered regarding how confident we can or should be that the current findings generalize across the globe?

We thank Caspi et al. (2026) for their remarkable contribution to the psychopathology literature. We hope that one benefit of their work is a heightened focus on diagnosis and treatment of social, emotional, and occupational dysfunction, and perhaps less focus on specific symptomatic expression of such dysfunction. Such a focus will be particularly beneficial for individuals with high scores on the p-factor of psychopathology.

## References

- Barlow, D. H., Farchione, T. J., Fairholme, C. P., Ellard, K. K., Boisseau, C. L., Allen, L. B., & Ehrenreich-May, J. (2011). *Unified protocol for transdiagnostic treatment of emotional disorders: Therapist guide*. Oxford University Press.
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Kotov, R., Krueger, R. F., Watson, D., Cicero, D. C., Conway, C. C., DeYoung, C. G., Eaton, N. R., Forbes, M. K., Hallquist, M. N., Litzman, R. D., Mullins-Sweatt, S. N., Ruggero, C. J., Simms, L. J., Waldman, I. D., Waszczuk, M. A., & Wright, A. G. C. (2021). The Hierarchical Taxonomy of Psychopathology (HiTOP): A quantitative nosology based on consensus of evidence. *Annual Review of Clinical Psychology*, 17(1), 83–108. <https://doi.org/10.1146/annurev-clinpsy-081219-093304>
- Krueger, R. F., Hobbs, K. A., Conway, C. C., Dick, D. M., Dretsch, M. N., Eaton, N. R., Forbes, M. K., Forbush, K. T., Keyes, K. M., Litzman, R. D., Michelini, G., Patrick, C. J., Sellbom, M., Slade, T., South, S. C., Sunderland, M., Tackett, J., Waldman, I., Waszczuk, M. A., ... HiTOP Utility Workgroup. (2021). Validity and utility of Hierarchical Taxonomy of Psychopathology (HiTOP): II. Externalizing superspectrum. *World Psychiatry*, 20(2), 171–193. <https://doi.org/10.1002/wps.20844>
- Riley, E. N., & Smith, G. T. (2017). Childhood drinking and depressive symptom level predict harmful personality change. *Clinical Psychological Science*, 5(1), 85–97. <https://doi.org/10.1177/2167702616661716>

Smith, G. T., Atkinson, E. A., Davis, H. A., Riley, E. N., & Oltmanns, J. R. (2020). The general factor of psychopathology. *Annual Review of Clinical Psychology, 16*(1), 75–98. <https://doi.org/10.1146/annurev-clinpsy-071119-115848>

Stern, M., Rohde, P., Desjardins, C. D., Perry, J. S., & Stice, E. (2025). Prospective reciprocal relations between psychosocial impairment and

eating disorder symptoms in a high-risk sample. *Eating Behaviors, 57*, Article 101958. <https://doi.org/10.1016/j.eatbeh.2025.101958>

Received October 30, 2025

Accepted November 1, 2025 ■

## COMMENTARY

## The Relationship's the Thing: Commentary on Caspi et al. (2026)

Susan C. South

Department of Psychological Sciences, Purdue University

To provide partial support for the “p” factor, Caspi et al. (2026) provide meta-analytic and large sample data evidence of assortative mating for various forms of psychopathology. From this, they conclude that assortative mating leads to transdiagnostic risk for symptoms of mental illness in offspring. In this commentary, I focus on why individuals assortatively mate for psychopathology, even in the face of evidence that they do not assort for similarity of personality. Personality is firmly entrenched as a correlate of mental disorders, to the point of being included in structural models of psychopathology. Individuals with different personalities end up in relationships that become distressed and trigger psychopathology; each partner's unique personality traits shape the form of psychopathology they experience. As Caspi et al. argue, interventions for mental illness do need to move beyond the traditional therapist-patient model to incorporate spouses and other family members. There may be a segment of the population, however, because of low socioeconomic status, prone to marital distress and psychopathology and more likely to benefit from financial support than psychological interventions.

*Keywords:* assortative mating, psychopathology, p factor

In this target article, Caspi et al. (2026) marshal evidence from three basic areas to support the existence and need to study “p,” a tendency to develop many different forms of psychopathology as opposed to a specific type of mental disorder. One of these lines of evidence is assortative mating for psychopathology. As the authors correctly note, there is evidence that romantic partners, particularly spouses, are correlated on symptoms or diagnoses of mental illness (Horwitz et al., 2023). Using data drawn from hospital records and data taken from primary care records, the authors find that people who have experienced a mental health disorder at some point in their adult lives are more likely to marry someone who has also experienced a mental health disorder. Assortative mating for the same disorder was more common for assortative mating for a different disorder, although there was evidence of the latter. This fits with work from a recent meta-analysis and analysis of data from the U.K. biobank, which reported substantial spousal correlations for, among other things, smoking status and substance use disorder (Horwitz et al., 2023).

What might be driving this assortative mating for psychopathology? Harper and Zietsch (2025) propose that assortative mating can be explained, in part, by genetic correlations between preferred traits

in one partner and the presence of the trait in the other partner. This may partly explain assortative mating for some forms of psychopathology. For instance, partners who like to drink may seek each other out. Even though marriage tends to act as a maturing force for most, leading to less drinking and other externalizing behaviors, research suggests that concordance for drinking, even high levels of drinking, is associated with more satisfying marriages (Homish & Leonard, 2007). In reading the target article and its focus on understanding developmental trajectories, I also thought of how cohorts might differ over time. For instance, it has been reported that members of Gen Z are drinking less (Cleveland Clinic, 2023). Will this continue, and will we see assortative mating for less drinking for this cohort?

One might hypothesize that assortative mating for personality might be driving assortative mating for psychopathology. There are strong links between personality and psychopathology, to the point that some have argued that the domains of psychopathology in a hierarchical, dimensional model can be considered personality traits (DeYoung et al., 2022). However, a striking finding from personality psychology, consistent over decades of research, is that spouses do not resemble each other on personality. Even the recent meta-analytic and large data analysis of assortative mating by Horwitz et al. (2023) found very low (but nonzero) partner agreement on the major domains of personality (openness, conscientiousness, neuroticism, agreeableness, and extraversion). This follows from recent research using advanced statistical methods that shows little similarity across couples for personality traits (Weidmann et al., 2023). A highly extroverted person may be just fine being married to a highly introverted individual, if that highly introverted spouse is willing to spend time with them.

Mate selection is a function of similar education, religion, and political values (and luck and timing)—many of which are explained by

---

Aidan G. C. Wright served as action editor.

Susan C. South  <https://orcid.org/0000-0003-0341-7074>

The authors have no conflicts of interest to declare.

Susan C. South served as lead for conceptualization and writing—original draft.

Correspondence concerning this article should be addressed to Susan C. South, Department of Psychological Sciences, Purdue University, 703 Third Street, West Lafayette, IN 47907, United States. Email: [ssouth@purdue.edu](mailto:ssouth@purdue.edu)

proximity. We tend to date people who are geographically close, and that brings similarities with it (e.g., if you meet your future partner in college, you will be similar on education). In the target article, the authors compare actual partners with random pairings of men and women in the data set. The fact that these random pairings resulted in few significant associations suggests that partner agreement on psychopathology is not simply a statistical artifact of mental disorders being common in the population. However, creating random pairings is a tough test. A fairer comparison would have been associations of own psychopathology with that of close friends (living in proximity, not Facebook friends). Then the question becomes, is there something about the marital relationship that lends itself to partnering with someone else with mental illness, as opposed to a sort of “social contagion” that affects people living in the same place and time (again, back to cohort effects).

The target article reports cross-disorder assortative mating as further evidence of “p” and of contributing to risk for “p” in offspring. It also fits with the lack of consistent evidence for the similarity of spouses on basic personality domains. Personality traits of each partner, but not similarity, predict the quality of one’s romantic relationship (Weidmann et al., 2023). There is abundant evidence at this point that personality traits, including pathological traits and personality disorders, negatively affect the quality of one’s romantic relationship (South, 2021). It is likely that, for many couples, assortative mating for psychopathology arises from the fact that they are both unhappy in the relationship and this stressor, alone or in combination with other stressors, triggers psychopathology. The form of psychopathology rests, in part, on each partner’s unique personality profile. A partner higher in neuroticism, for instance, may experience depression whereas a partner higher on disinhibition may be more likely to experience harmful substance use. It is unclear from the analyses in the target article if both members of the couple had a disorder at the same time; future research should investigate how disorders that impact individuals within romantic dyads wax and wane over the course of the relationship. Do partners experience a disorder (even if not the same disorder) at the same time? It is also important for future research to tease apart mechanisms driving how relationship distress triggers pathology. For instance, is distress a result of realizing that one’s partner has different needs, goals, and wants, and/or their partner is not helping meet their needs and goals (Fitzsimons et al., 2015), but feeling stuck with few alternatives?

I appreciated Caspi et al.’s (2026) application of their findings to suggestions for future intervention efforts. There are couple-focused therapies (i.e., for depression, substance use), but they tend to focus on a “target” patient. The findings of this article would suggest that couples-focused intervention should incorporate a lifetime assessment of both members of the couple and include coping and skills training for both partners and any children living in the family.

Finally, special attention should be paid to couples who may not benefit from family interventions because of the totality of challenges they face. Assortative mating is higher for things related to socioeconomic status (SES; e.g., educational attainment and IQ) than for personality and psychopathology. Notably, SES is a strong correlate of psychopathology and can increase the genetic influences on mental illness (South & Krueger, 2011). Individuals at the lowest levels of SES may have few if any resources to buffer against the stressors that would lead to marital strain and mental health problems. Research has shown that there are small but significant groups with the most severe mental

disorders (Kessler et al., 2005) and in the most distressed and unhappy relationships (Lavner et al., 2012). Future work needs to investigate how these groups track over time—are those in the lowest SES the most likely to be in distressed relationships and dealing with the most severe forms of psychopathology?

## References

- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Cleveland Clinic. (2023, November 1). *Is Generation Z drinking less?* <https://health.clevelandclinic.org/why-gen-z-is-drinking-less>
- DeYoung, C. G., Chmielewski, M., Clark, L. A., Condon, D. M., Kotov, R., Krueger, R. F., Lynam, D. R., Markon, K. E., Miller, J. D., Mullins-Sweatt, S. N., Samuel, D. B., Sellbom, M., South, S. C., Thomas, K. M., Watson, D., Watts, A. L., Widiger, T. A., Wright, A. G. C., & HiTOP Normal Personality Workgroup. (2022). The distinction between symptoms and traits in the Hierarchical Taxonomy of Psychopathology (HiTOP). *Journal of Personality*, 90(1), 20–33. <https://doi.org/10.1111/jopy.12593>
- Fitzsimons, G. M., Finkel, E. J., & vanDellen, M. R. (2015). Transactive goal dynamics. *Psychological Review*, 122(4), 648–673. <https://doi.org/10.1037/a0039654>
- Harper, K. T., & Zietsch, B. P. (2025). Assortative mating is a natural consequence of heritable variation in preferences and preferred traits. *Psychological Science*, 36(10), 771–779. <https://doi.org/10.1177/09567976251365900>
- Homish, G. G., & Leonard, K. E. (2007). The drinking partnership and marital satisfaction: The longitudinal influence of discrepant drinking. *Journal of Consulting and Clinical Psychology*, 75(1), 43–51. <https://doi.org/10.1037/0022-006X.75.1.43>
- Horvitz, T. B., Balbona, J. V., Paulich, K. N., & Keller, M. C. (2023). Evidence of correlations between human partners based on systematic reviews and meta-analyses of 22 traits and UK Biobank analysis of 133 traits. *Nature Human Behaviour*, 7(9), 1568–1583. <https://doi.org/10.1038/s41562-023-01672-z>
- Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 617–627. <https://doi.org/10.1001/archpsyc.62.6.617>
- Lavner, J. A., Bradbury, T. N., & Karney, B. R. (2012). Incremental change or initial differences? Testing two models of marital deterioration. *Journal of Family Psychology*, 26(4), 606–616. <https://doi.org/10.1037/a0029052>
- South, S. C. (2021). Pathology in relationships. *Annual Review of Clinical Psychology*, 17(1), 577–601. <https://doi.org/10.1146/annurev-clinpsy-081219-115420>
- South, S. C., & Krueger, R. F. (2011). Genetic and environmental influences on internalizing psychopathology vary as a function of economic status. *Psychological Medicine*, 41(1), 107–117. <https://doi.org/10.1017/S0033291710000279>
- Weidmann, R., Purol, M. F., Alabdullah, A., Ryan, S. M., Wright, E. G., Oh, J., & Chopik, W. J. (2023). Trait and facet personality similarity and relationship and life satisfaction in romantic couples. *Journal of Research in Personality*, 104, Article 104378. <https://doi.org/10.1016/j.jrp.2023.104378>

Received October 30, 2025  
Accepted November 1, 2025 ■

## COMMENTARY

## Transdiagnostic Formulations Can Inform Intervention and Academic Training Programs: Commentary on Caspi et al. (2026)

Maureen Zalewski<sup>1</sup>, Sheila E. Crowell<sup>1</sup>, and Stephanie D. Stepp<sup>2</sup><sup>1</sup> Psychology Department, University of Oregon<sup>2</sup> Department of Psychiatry, University of Pittsburgh

Caspi et al. (2026) present intergenerational and developmental evidence demonstrating that psychopathology is fundamentally transdiagnostic, reflecting a general liability for mental disorder (p). We extend their argument by emphasizing how transdiagnostic psychotherapy models and training frameworks can actualize this concept in clinical practice. Using dialectical behavior therapy as an exemplar, we illustrate how targeting shared mechanisms, such as emotion dysregulation, can reduce psychopathology across diagnoses and generations. Finally, we call for clinical training programs to adopt curricula centered on transdiagnostic science to align the next generation of clinicians with the empirical realities of p and developmental psychopathology.

**General Scientific Summary**

Extending the work of Caspi et al. (2026), this commentary highlights the benefits and need of prioritizing transdiagnostic over disorder-specific psychotherapy treatments. Further, there is a need for academic programs to teach curricula explicitly rooted in transdiagnostic approaches, as this will ultimately shape the next generation of mental health clinicians.

**Keywords:** transdiagnostic interventions, emotion dysregulation, dialectical behavior therapy, academic training programs

As developmental psychopathologists who study intergenerational mental health, we appreciated Caspi et al.'s (2026) inclusion of developmental and intergenerational evidence as an empirical foundation for understanding p. In their article, Caspi et al. synthesize data from population-based registries in Denmark and Norway and the Dunedin Longitudinal Study to show that risk for psychopathology is transdiagnostic rather than disorder-specific. They demonstrate that individuals with a history of any mental disorder often partner with others who have their own, often distinct, mental disorders, creating children who inherit overlapping genetic and environmental

vulnerabilities. Accordingly, parents' mental disorders predict a range of offspring psychopathology. Furthermore, individuals' own diagnostic patterns shift across the lifespan, underscoring the dynamic nature of p. Caspi et al. argue that this pervasive interconnectedness of mental disorders calls for a fundamental reorientation of research, treatment, and training models toward shared mechanisms and developmental processes rather than discrete diagnostic categories.

Throughout our clinical scientist careers, we have engaged in transdiagnostic over disorder-specific research and practice. The findings presented by Caspi et al. (2026) are consistent with this framework and highlight important opportunities to extend their implications, particularly for psychotherapy and clinical training. Below, we focus on how transdiagnostic models of treatment and training, exemplified by dialectical behavior therapy (DBT), operationalize the principles articulated in Caspi et al.'s framework and provide a pathway for integrating p into both clinical science and practice.

**Transdiagnostic Psychotherapy Models as Clinical Expressions of p**

Caspi et al. (2026) argue that transdiagnostic risk factors are ubiquitous, which requires the field to rethink “design, measurement, and treatment—as well as funding, publication, and training” (p. 489). We take this call seriously and view transdiagnostic psychotherapy models as critical for translating p into practice. For several decades, there

Aidan G. C. Wright served as action editor.

Maureen Zalewski  <https://orcid.org/0000-0001-5594-5296>

Sheila E. Crowell declares a financial interest in the Utah Center for Evidence Based Treatment, a private psychotherapy practice, where she holds a co-ownership position.

Maureen Zalewski served as lead for conceptualization, writing—original draft, and writing—review and editing. Sheila E. Crowell served in a supporting role for conceptualization, writing—original draft, and writing—review and editing. Stephanie D. Stepp served in a supporting role for conceptualization, writing—original draft, and writing—review and editing.

Correspondence concerning this article should be addressed to Maureen Zalewski, Psychology Department, University of Oregon, 1227 University of Oregon, Eugene, OR 97403, United States. Email: [zalewski@uoregon.edu](mailto:zalewski@uoregon.edu)

has been growing recognition that traditional, disorder-specific interventions cannot fully address the complexity of co-occurring disorders and presenting problems seen in research and community settings. This realization, coupled with advances in developmental and etiological science, has fueled a rise in transdiagnostic treatments that target shared vulnerabilities across diagnoses (Sauer-Zavala et al., 2017). Such treatments explicitly target core processes that confer risk for multiple disorders (e.g., emotion dysregulation, avoidance, cognitive inflexibility); they have clear etiological models, specify mechanisms of change, and demonstrate efficacy across diagnostic categories. Importantly, these treatments move beyond symptom reduction toward modifying underlying developmental and interpersonal processes, which is precisely the level at which *p* is theorized to operate.

DBT (Linehan, 1993) is among the most widely disseminated and empirically supported transdiagnostic treatments. While DBT was historically recognized as an evidence-based treatment for borderline personality disorder, its theoretical foundation and mechanisms of change are explicitly transdiagnostic. The biosocial model that grounds DBT posits that emotion dysregulation arises from a transactional process between biological vulnerabilities and invalidating environments (Crowell et al., 2009). This framework has been applied to effectively treat a range of clinical presentations characterized by emotional, behavioral, and interpersonal instability, including suicidality, depression, substance use, and eating disorders. From the perspective of *p*, DBT provides an applied example of a treatment designed to address a shared underlying liability rather than any single disorder.

Improving emotion regulation is the primary mechanism of change in DBT, accomplished primarily through skills acquisition and generalization. Weekly DBT skills groups teach mindfulness, distress tolerance, emotion regulation, and interpersonal effectiveness, which strengthen adaptive regulation across contexts. While DBT skills are only one component of the original treatment model, they have proven so potent that they are increasingly applied as a transdiagnostic treatment (Delaquis et al., 2022). Emotion dysregulation change has been empirically validated as both a mechanism and outcome of DBT (Neacsiu et al., 2010), further supporting the model's alignment with a developmental and mechanistic understanding of *p*.

### Intergenerational Implications of Transdiagnostic Treatments

Caspi et al.'s (2026) integration of intergenerational data provides a crucial empirical foundation for understanding how transdiagnostic risk is transmitted across generations. However, these findings also highlight a novel intervention opportunity: targeting shared mechanisms within parents to disrupt transdiagnostic risk transmission to children. Our ongoing research applies DBT as a transdiagnostic prevention and intervention model for parents with significant emotion dysregulation and histories of suicidality. Specifically, our work leverages DBT's effect on adult emotion dysregulation, as a way of examining intergenerational effects on children's emotion regulation (Zalewski et al., 2023).

In one randomized controlled trial, we demonstrated that improvements in maternal emotion regulation following DBT skills predicted steeper growth in children's emotion regulation across early development (Byrd et al., 2021). Current trials extend this work to mothers of school-age children and to pregnant women at elevated

risk for psychopathology. These findings provide experimental evidence of the intergenerational reach of a transdiagnostic intervention, demonstrating that modifying parental regulatory capacity can have downstream effects on child emotion regulation trajectories. This research complements Caspi et al.'s (2026) population-level findings by showing how *p* can be addressed at a mechanistic level within families. It also underscores the importance of designing and testing interventions that are not only transdiagnostic but transgenerational by targeting processes that sustain risk within families.

### Integrating *p* into Clinical Training and Education

For the field to fully embrace *p*, it must invest in training practitioners and clinical scientists who are fluent in transdiagnostic conceptualizations and committed to testing interventions that target shared developmental mechanisms. Academic training programs play a central role in shaping how emerging scientists and practitioners conceptualize psychopathology. We argue that training programs should explicitly prioritize teaching transdiagnostic frameworks integrating developmental, biological, and contextual processes and highlight shared mechanisms, such as emotion regulation, reward sensitivity, and cognitive control. Newer developments in academic training programs show promise of this approach. As an example, the Psychological Clinical Science Accreditation System (PCSAS), which accredited its first clinical psychology program in 2009, positions science, and specifically the integration of research and practice, as a cornerstone of its mission (Levenson, 2017). Because PCSAS emphasizes flexibility in training approaches, PCSAS-accredited programs are well-suited to be leaders in fully realizing the application of *p* across components of their training programs. Further, new advancements to train bachelor's-level practitioners for youth mental health services roles increasingly rely on transdiagnostic approaches, where trainees apply common elements of effective interventions across a wide range of common mental health problems (McLaughlin et al., 2025).

These training approaches not only expand access to care but also operationalize *p*-aligned principles in applied settings. While categorical diagnostic frameworks (e.g., *Diagnostic and Statistical Manual of Mental Disorders*, fifth edition) are currently necessary for interprofessional communication and health care reimbursement, training programs should embed students within a broader, critical understanding of the limitations of diagnostic tools and the value of transdiagnostic, process-based perspectives.

### Conclusion

Caspi et al.'s (2026) integrative analysis of assortative mating, intergenerational transmission, and developmental trajectories provides a powerful empirical case for *p*, underscoring the shared liability underlying diverse mental disorders. We extend their argument by emphasizing that transdiagnostic treatments and training models are the clinical and educational embodiments of this framework. Treatments such as DBT exemplify how interventions grounded in etiological models and mechanisms of change can address multiple forms of psychopathology simultaneously and even interrupt intergenerational cycles of risk. We contend that integrating *p* into psychotherapy and training will not only accelerate clinical science but also improve our capacity to prevent and treat psychopathology across generations.

## References

- Byrd, A. L., Lee, A. H., Frigoletto, O. A., Zalewski, M., & Stepp, S. D. (2021). Applying new RDoC dimensions to the development of emotion regulation: Examining the influence of maternal emotion regulation on within-individual change in child emotion regulation. *Development and Psychopathology*, 33(5), 1821–1836. <https://doi.org/10.1017/s0954579421000948>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan’s theory. *Psychological Bulletin*, 135(3), 495–510. <https://doi.org/10.1037/a0015616>
- Delaquis, C. P., Joyce, K. M., Zalewski, M., Katz, L. Y., Sulymka, J., Agostinho, T., & Roos, L. E. (2022). Dialectical behaviour therapy skills training groups for common mental health disorders: A systematic review and meta-analysis. *Journal of Affective Disorders*, 300, 305–313. <https://doi.org/10.1016/j.jad.2021.12.062>
- Levenson, R. W. (2017). Clinical psychology training: Accreditation and beyond. *Annual Review of Clinical Psychology*, 13(1), 1–22. <https://doi.org/10.1146/annurev-clinpsy-021815-093559>
- Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. Guilford Press.
- McLaughlin, K. A., Bearman, S. K., & Zalewski, M. (2025). *Transforming the youth mental health workforce* [Manuscript submitted for publication]. The Ballmer Institute for Children’s Behavioral Health, University of Oregon.
- Neacsiu, A. D., Rizvi, S. L., & Linehan, M. M. (2010). Dialectical behavior therapy skills use as a mediator and outcome of treatment for borderline personality disorder. *Behaviour Research and Therapy*, 48(9), 832–839. <https://doi.org/10.1016/j.brat.2010.05.017>
- Sauer-Zavala, S., Gutner, C. A., Farchione, T. J., Boettcher, H. T., Bullis, J. R., & Barlow, D. H. (2017). Current definitions of “transdiagnostic” in treatment development: A search for consensus. *Behavior Therapy*, 48(1), 128–138. <https://doi.org/10.1016/j.beth.2016.09.004>
- Zalewski, M., Maliken, A., Lengua, L. J., Martin, C. G., Roos, L., & Everett, Y. (2023). Integrating dialectical behavior therapy with child and parent training interventions: A narrative and theoretical review. *Clinical Psychology: Science and Practice*, 30(4), 365–376. <https://doi.org/10.1111/cpsp.12363>

Received October 31, 2025

Accepted November 3, 2025 ■

## COMMENTARY

# Transdiagnostic Psychopathology Research Requires Comprehensive, Lifespan Coverage of Psychological Symptoms *and* Function and Ability: Commentary on Caspi et al. (2026)

Sylia Wilson<sup>1</sup>, Whitney Ringwald<sup>2</sup>, and Giorgia Michelini<sup>3</sup><sup>1</sup> Institute of Child Development, University of Minnesota<sup>2</sup> Department of Psychology, University of Minnesota<sup>3</sup> Department of Biological and Experimental Psychology, Queen Mary University of London

Contemporary conceptualizations of mental health conditions are transdiagnostic and dimensional, and based on structural models. We suggest it is necessary to include comprehensive coverage of the full range of possible diagnoses, symptoms, and signs/behaviors, as well as function and ability, and take a longitudinal, lifespan approach, to fully understand the range of human experience.

**General Scientific Summary**

What is understood about mental health conditions builds upon assessment of psychiatric diagnoses and symptoms. We emphasize how critical it is that assessment be comprehensive across the entire lifespan and suggest assessment of function and ability, in addition to dysfunction and maladaptation, is also necessary to fully understand the range of human experience.

*Keywords:* psychopathology, transdiagnostic, structural models

Caspi et al. (2026) give an elegant synthesis of intergenerational and developmental data to argue convincingly for transdiagnostic psychopathology research, suggesting that focusing on one or even a few mental health conditions at a time hinders research and clinical practice. Comprehensive coverage is key because all statistical models are a function of the variables that are included in analyses, meaning that conclusions about psychopathology based on models derived without the full range of possible diagnoses, symptoms, and signs/behaviors will be limited and incomplete. We further suggest that a narrow focus on psychopathology and symptoms is likewise limiting—understanding the full range of human experience requires consideration of broader domains of function and ability, and the systems that enable adaptation. Moreover, because the expression of mental health conditions, function, and ability shifts over time, as Caspi et al. (2026) so compellingly illustrate, moving

transdiagnostic psychopathology research forward necessarily requires taking a longitudinal, lifespan approach.

## Coverage of Psychopathology in Structural Models Is Not Yet Comprehensive

Contemporary transdiagnostic dimensional frameworks of psychopathology, such as hierarchical models with higher order dimensions (e.g., internalizing, externalizing, thought disorder, and p factor), hold considerable promise to advance understanding of the nature of psychopathology. At the same time, it is increasingly clear that coverage of relevant diagnoses, symptoms, and signs/behaviors in current models is incomplete. A major reason is that psychopathology is dynamic, manifesting differently over time and development, which means that samples assessed at any single time point will, necessarily, provide incomplete coverage. Some mental health conditions are by nature thought to be transient and episodic (e.g., depression), while others long thought to be stable and persistent may actually show considerable variation in course and impact (e.g., autism). Some symptoms or behaviors do not typically become evident until adolescence or adulthood (e.g., psychosis) and thus are not reflected in models derived from child samples. Others typically emerge in childhood (e.g., neurodevelopmental conditions, oppositional defiant disorder) or manifest differently in children (e.g., irritability in depression) and are thus not reflected in adult samples. Consequently, some conditions, especially those most commonly seen earlier or later in life (e.g.,

Aidan G. C. Wright served as action editor.

Sylia Wilson  <https://orcid.org/0000-0003-0144-524X>

Sylia Wilson served as lead for conceptualization and writing—original draft. Whitney Ringwald served in a supporting role for conceptualization and writing—review and editing. Giorgia Michelini served in a supporting role for conceptualization and writing—review and editing.

Correspondence concerning this article should be addressed to Sylia Wilson, Institute of Child Development, University of Minnesota, 51 East River Parkway, Minneapolis, MN 55455, United States. Email: [syliaw@umn.edu](mailto:syliaw@umn.edu)

neurodevelopmental and neurocognitive conditions), are less frequently included in structural models.

### A Lifespan Approach Is Necessary for Transdiagnostic Psychopathology Research

Truly comprehensive transdiagnostic models of psychopathology require coverage across the full range of possible diagnoses, symptoms, and signs/behaviors, during the developmental periods in which they are evident, and considering dynamic courses with potentially varying manifest indicators. This work is challenging. Taking a lifespan approach that includes assessments from infancy into older adulthood by definition takes a lifetime—requiring generations of researchers to collect the data. Mental health conditions and functioning are dynamic, with relevant indicators emerging, changing, co-occurring, and disappearing, and with potentially different meanings in different developmental periods and contexts. Despite these challenges, we agree with Caspi et al. (2026) that this work is worth the effort. Tackling these issues as a field will require complementary study designs, collaboration, and methodological innovation. For example, large-scale cohort and panel studies with regular (e.g., yearly) assessments can be complemented by smaller samples with deeper phenotypic assessment and finer grained timescales (e.g., ecological momentary assessment) and enriched with population-level records (e.g., health, education, and criminal justice). Promising methodological advances include dynamic statistical models that incorporate time series and harmonization across multiple data sets to span developmental periods. These approaches will open new possibilities to measure and model psychopathology and function, thereby enabling comprehensive coverage of developmental trajectories across the lifespan.

### Comprehensive, Lifespan Coverage Leads to Changes in Conceptualizations of Psychopathology

Because our basic understanding of transdiagnostic psychopathology and related function is built upon the results of structural models, decisions about what content to include in these models are highly consequential—differential or incomplete content coverage influences whether dimensions are identified at all, how items are captured by each dimension, and the hierarchical relationships among dimensions. For example, although evidence for an externalizing dimension is longstanding, earlier work in samples of children with limited assessment of or variability in substance use did not (could not) identify the separable disinhibited (e.g., substance use) and antagonistic (e.g., psychopathy) externalizing factors that are evident in samples of adolescents and adults. In addition, with the notable exception of attention-deficit/hyperactivity disorder, the neurodevelopmental conditions (e.g., autism and learning disorders) have not been commonly included in structural models. However, our recent research suggests comprehensive models include a neurodevelopmental dimension (Michelini et al., 2024), with attention-deficit/hyperactivity disorder features mainly captured by this new dimension (rather than the externalizing dimension) when other neurodevelopmental items are included in the model. This work also suggests a more radical change in conceptualizing psychopathology that accounts for links to cognitive function (e.g., executive functions)—a domain of functioning that is not yet explicitly included in transdiagnostic models. Building upon theory and empirical work showing that some aspect of cognitive

function difficulties is evident for nearly all mental health conditions (Abramovitch et al., 2021; Forbes, 2026; Morris et al., 2025; Ringwald et al., 2025; Wilson & Michelini, 2026), we have proposed the inclusion of a higher order “cognitive function” factor that is broad, nonspecific, and transdiagnostic. This cognitive function factor can be conceptualized similar to the general factor of psychopathology and placed at the top of hierarchical models—correlated with the p factor and broadly linked to all forms of psychopathology.

### Assessment of Function and Ability Is Also Needed

Although we have emphasized the need for comprehensive coverage of diagnoses, symptoms, and signs/behaviors in structural models, we believe even this is incomplete. Any understanding of psychological dysfunction must also account for psychological function in both conceptualization and measurement. Dysfunction, abnormality, and maladaptation are necessarily defined by and contrasted with function, normality, and adaptation (and reflect an individual’s interactions with their environments, as well as societal norms). Although distress and impairment are required for diagnosis, we suggest that function and ability are not already adequately represented in structural models simply by a lack of dysfunction or being low on psychopathology dimensions. For example, lower detachment does not necessarily mean capacity for sustaining intimate, mutually satisfying relationships nor does lower disinhibition necessarily mean greater planfulness, organization, or well-reasoned decision making. That is, the absence of dysfunction does not in and of itself imply the presence of positive functioning. A complete understanding of the full range of psychological dysfunction and adaptive functioning requires measurement across the full distribution. This work will require assessment of broader domains (e.g., cognitive function, social/interpersonal abilities, motivation, and quality of life) and methods other than the usual self-/informant-reports and clinical interviews, such as neurocognitive tests and behavioral tasks. Thus, in addition to the need for transdiagnostic psychopathology research that Caspi et al. (2026) highlight, we suggest that a multimethod approach encompassing multiple domains is also necessary to determine and model the true structure of psychopathology and function. This approach also aligns with clinical practice that considers both challenges and strengths—by helping to identify what those strengths are.

### Moving Transdiagnostic Psychopathology Research Forward

Contemporary transdiagnostic dimensional frameworks of psychopathology, based on structural models, have changed how we conceptualize mental health conditions. Comprehensive coverage of the full range of possible diagnoses, symptoms, and signs/behaviors, as well as function and ability, and a longitudinal, lifespan approach are now necessary to move transdiagnostic psychopathology research to the next stage. So doing will result in more complete and accurate structural models and support the critical next step for this work—to understand the mechanisms that cause and maintain mental health conditions.

### References

- Abramovitch, A., Short, T., & Schweiger, A. (2021). The C factor: Cognitive dysfunction as a transdiagnostic dimension in psychopathology. *Clinical Psychological Review, 86*, Article 102007. <https://doi.org/10.1016/j.cpr.2021.102007>

- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Forbes, M. K. (2026). Cognitive and neurodevelopmental disorders in dimensional models of psychopathology. In C. J. Hopwood & C. Sharp (Eds.), *Dimensional diagnosis: Practical and conceptual issues in the integration of personality and psychopathology* (pp. 311–324). Guilford Press.
- Michelini, G., Carlisi, C. O., Eaton, N. R., Elison, J. T., Haltigan, J. D., Kotov, R., Krueger, R. F., Latzman, R. D., Li, J. J., Levin-Aspensson, H. F., Salum, G. A., South, S. C., Stanton, K., Waldman, I. D., & Wilson, S. (2024). Where do neurodevelopmental conditions fit in transdiagnostic psychiatric frameworks? Incorporating a new neurodevelopmental spectrum. *World Psychiatry*, 23(3), 333–357. <https://doi.org/10.1002/wps.21225>
- Morris, I., Michelini, G., & Wilson, S. (2025). Moving toward transdiagnostic dimensional models of neurodiversity and mental health (and away from models of psychopathology). *Journal of Psychopathology and Clinical Science*, 134(5), 483–485. <https://doi.org/10.1037/abn0001007>
- Ringwald, W. R., Abramovitch, A., van Rentergem, J. A. A., & Kotov, R. (2025). Do cognitive functions belong in the Hierarchical Taxonomy of Psychopathology model? A meta-analysis. *Perspectives on Psychological Science*, 20(6), 1049–1064. <https://doi.org/10.1177/17456916251347926>
- Wilson, S., & Michelini, G. (2026). Incorporating neurodevelopmental and neurocognitive conditions in transdiagnostic dimensional models of psychopathology. In C. J. Hopwood & C. Sharp (Eds.), *Dimensional diagnosis: Practical and conceptual issues in the integration of personality and psychopathology* (pp. 305–310). Guilford Press.

Received November 3, 2025

Accepted November 4, 2025 ■

## COMMENTARY

## P Is for Personality: Commentary on Caspi et al. (2026)

Jennifer L. Tackett<sup>1</sup> and Benjamin A. Katz<sup>2</sup><sup>1</sup> Department of Psychology, Northwestern University<sup>2</sup> School of Psychological Science, University of Haifa

Caspi et al. (2026) highlight substantive questions over methodological debates when considering covariation of disorders across development. In this commentary, we argue that personality and temperament offer models through which such heterogeneity should be understood. Personality traits index a wide range of biological, psychological, and behavioral individual differences and share common structures with dimensional models of psychopathology. As such, they are particularly useful for identifying both transdiagnostic and domain-specific processes of psychopathology. For decades, editorials in journals such as this have argued that personality should be incorporated into developmental psychopathology to a larger degree. We hope that discussions initiated by the target article being discussed may be a catalyst to do so.

**General Scientific Summary**

In this commentary, we argue that personality and temperament provide a powerful, developmentally grounded framework for understanding both transdiagnostic vulnerability (often labeled the “p-factor”) and domain-specific pathways of psychopathology, because personality traits reflect stable yet malleable individual differences in emotion regulation, behavior, and cognition that unfold across the lifespan. Drawing on decades of theory and recent empirical evidence, we highlight how personality models clarify why different forms of psychopathology cluster, diverge, and transform over time, offering critical insight into mechanisms such as heterotypic continuity and equifinality. Incorporating personality into developmental psychopathology research therefore represents a missed but recoverable opportunity to move beyond descriptive models toward more explanatory, developmentally informed accounts of mental disorder.

*Keywords:* developmental psychopathology, general factor of psychopathology, heterotypic continuity, personality, temperament

We appreciate the opportunity to reflect on the target article by Caspi et al. (2026) and the spotlight it shines on some of the larger questions related to disorder covariation from young development through adulthood. We particularly appreciate the target article’s focus on substantive research findings and questions. Although early work on such questions showed similar scope, much recent dialogue has been bogged down by smaller scale debates around analytic technique. We hope that the target article may help our field move into a more substantive and productive discussion around how to understand systematic and

robust covariation across psychopathology domains and spectra and across time.

Although we share points of difference with the authors of the target article on some smaller points, we are far more alike than different. We wholeheartedly agree that research on psychopathology would benefit from better accounting for broad disorder covariation, referenced here as the p-factor. We also strongly agree regarding the importance of questions related to within-person homogeneity versus heterogeneity of disorders across time and that existing dimensional structural approaches to psychopathology do not fully incorporate developmental considerations to the detriment of understanding phenomena such as heterotypic continuity (as we have also discussed; Tackett & Hallquist, 2022). Such issues shape our understanding of etiology, presentation, developmental course, and prevention/intervention efforts. Here we offer some additional points of consideration.

The present commentary remains agnostic with regard to mechanisms underlying their findings related to etiological covariation (e.g., equifinality/multifinality) and developmental change. We argue that comprehensive models of personality and temperament are a helpful psychological context to understand such mechanisms. Models of personality and temperament are largely consistent with (and

Aiden G. C. Wright served as action editor.

Jennifer L. Tackett  <https://orcid.org/0000-0002-5084-3511>

Jennifer L. Tackett served as lead for conceptualization and writing—original draft. Benjamin A. Katz served in a supporting role for conceptualization and writing—original draft.

Correspondence concerning this commentary should be addressed to Jennifer L. Tackett, Department of Psychology, Northwestern University, 2029 Sheridan Road, Evanston, IL 60208, United States. Email: [jennifer.tackett@northwestern.edu](mailto:jennifer.tackett@northwestern.edu)

even analogous to) structural dimensional models of psychopathology (Tackett & Hallquist, 2022). For example, not only does broad trait neuroticism highly correlate with the p-factor, but factor analytic sub-factors of trait neuroticism also show dimension-specific overlap with internalizing and externalizing (Brandes et al., 2019). By intentionally incorporating normal-range manifestations of individual differences, personality trait models offer a comprehensive and rich psychological context within which to advance our understanding of psychopathology emergence and development (Tackett et al., 2009). Personality reflects a range of biological, cognitive, and behavioral processes (Bainbridge et al., 2022) but offers incremental information about psychopathology variation as well. For example, the inclusion of models of personality offers incremental etiologic information in understanding associations between markers of executive functioning and psychopathology (Shields et al., 2022). Longitudinally, personality-based models point out how the heterotypic continuity that the authors describe is quite common and hereditary (Tackett & Hallquist, 2022). In one study (Shields et al., 2021), we found that even *grandparent's* internalizing personality psychopathology was associated with *grandchildren's* externalizing personality psychopathology, further support for the points made by Caspi et al. (2026). Thus, both structurally and etiologically, there is ample evidence to support personality as a critical context for understanding psychopathology covariation and development.

A recent meta-analysis of 147 studies ( $N = 46,369$ ) on the associations between personality and youth psychopathology (Katz et al., *in press*) showed how personality can be used to understand both transdiagnostic factors such as the p-factor and specific factors that differentiate between disorder presentations. Specifically, examination of zero-order correlations showed some differentiation between internalizing and externalizing psychopathology (e.g., both domains showed large associations with trait neuroticism), but new and starker differences emerged when accounting for their covariation (à la “p”). Specifically, when including the covariance between the internalizing and externalizing domains within the model, we found that internalizing was most associated with low extraversion and high neuroticism, and to a lesser extent higher agreeableness and conscientiousness. Externalizing, on the other hand, was most associated with high extraversion, low agreeableness, and low conscientiousness. This was found to be the case even when removing items commonly shared between personality and psychopathology assessments (e.g., sadness items in neuroticism assessment and the internalizing dimension). Personality contains both “p-factor” traits that have bivariate associations with all of psychopathology (i.e., high neuroticism) and traits that are dimension specific (i.e., low extraversion in the internalizing dimension and high extraversion in the externalizing dimension).

Personality's relevance to development and psychopathology, as well as ease of high-quality assessment, make it a valuable candidate for large-scale clinical research that focuses on the structure of psychopathology. Broader models of youth personality and temperament are ideally suited to advance our understanding of the p-factor, transdiagnostic mechanisms for psychopathology, and the presence of heterotypic continuity across the lifespan. Yet they remain woefully understudied within both personality psychology and developmental psychopathology. Perhaps some of this issue reflects decades-old debates about the existence of early life personality, the ability to measure it well, and its associations with clinical outcomes. These debates, however, have largely long since been settled (Shiner & Caspi, 2003). Thus the exclusion of

these models in current research reflects a lost opportunity to advance our scientific understanding of youth psychopathology. An editorial in this journal over a decade ago lamented the same, calling for empirical attention to these issues (Goodman, 2012), but we have yet to see it realized in current research outputs and applications.

We fully agree with Caspi et al. (2026) that current and future psychopathology research must prioritize cross/transdiagnostic studies; the contribution of single-disorder studies is increasingly modest and potentially incorrectly classifies the role of the pathological process being studied. For those interested in transdiagnostic developmental work, we encourage them to consider personality and temperament frameworks that offer a more comprehensive and rich context of individual differences and may highlight biological, psychological, and cognitive processes that will be potentially fruitful areas of inquiry.

## References

- Bainbridge, T. F., Ludeke, S. G., & Smillie, L. D. (2022). Evaluating the big five as an organizing framework for commonly used psychological trait scales. *Journal of Personality and Social Psychology, 122*(4), 749–777. <https://doi.org/10.1037/pspp0000395>
- Brandes, C. M., Herzhoff, K., Smack, A. J., & Tackett, J. L. (2019). The p factor and the n factor: Associations between the general factors of psychopathology and neuroticism in children. *Clinical Psychological Science, 7*(6), 1266–1284. <https://doi.org/10.1177/2167702619859332>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science, 135*(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Goodman, S. (2012). Editorial: Approaching 125. *Journal of Abnormal Psychology, 121*(1), 1–3. <https://doi.org/10.1037/a0026618>
- Katz, B. A., Shields, A. N., Watts, A., & Tackett, J. L. (*in press*). Temperament, personality, and psychopathology in youth: A preregistered multi-level meta-analysis and preregistered large-scale replication and extension. *Psychological Bulletin*.
- Shields, A. N., Malanchini, M., Vinnik, L., Tucker-Drob, E. M., Harden, K. P., & Tackett, J. L. (2022). Genetic variance in conscientiousness relates to youth psychopathology beyond executive functions. *Journal of Psychopathology and Clinical Science, 131*(8), 830–846. <https://doi.org/10.1037/abn0000781>
- Shields, A. N., Oltmanns, T. F., Boudreaux, M. J., Paul, S. E., Bogdan, R., & Tackett, J. L. (2021). The impact of personality pathology across three generations: Evidence from the St. Louis Personality and Intergenerational Network Study. *Clinical Psychological Science, 9*(5), 900–918. <https://doi.org/10.1177/2167702621989665>
- Shiner, R., & Caspi, A. (2003). Personality differences in childhood and adolescence: Measurement, development, and consequences. *Journal of Child Psychology and Psychiatry, 44*(1), 2–32. <https://doi.org/10.1111/1469-7610.00101>
- Tackett, J. L., Balsis, S., Oltmanns, T. F., & Krueger, R. F. (2009). A unifying perspective on personality pathology across the life span: Developmental considerations for the fifth edition of the diagnostic and statistical manual of mental disorders. *Development and Psychopathology, 21*(3), 687–713. <https://doi.org/10.1017/S095457940900039X>
- Tackett, J. L., & Hallquist, M. (2022). The need to grow: Developmental considerations and challenges for modern psychiatric taxonomies. *Journal of Psychopathology and Clinical Science, 131*(6), 660–663. <https://doi.org/10.1037/abn0000751>

Received November 3, 2025

Accepted November 4, 2025 ■

## COMMENTARY

Shuffling Through Hierarchies in Developmental Psychopathology:  
Commentary on Caspi et al. (2026)Daniel P. Moriarity<sup>1</sup> and Jamie L. Hanson<sup>2</sup><sup>1</sup> Department of Psychology, University of Pennsylvania<sup>2</sup> Department of Psychology, University of Pittsburgh

Caspi et al. (2026) use the metaphor of “a deck of cards” in their work to describe psychopathology over the lifespan—specifically, how an individual’s experience of psychopathology can change across development (both changing between different disorders and different presentations of the same diagnosis). Yet mental health research typically fragments these trajectories: Cross-sectional studies examine discrete time points, whereas longitudinal studies often track isolated disorders. Neither captures the dynamic, multidisorder life course patterns that Caspi et al. document. For example, someone may demonstrate anxious symptoms in adolescence, depressive symptoms in early adulthood, substance abuse in midlife, and psychosis symptoms even later in life.

**General Scientific Summary**

Caspi et al. (2026) provide compelling evidence for how an individual’s experience of mental health can shift meaningfully across development. This commentary extends this conversation to highlight the theoretical and clinical benefits of considering the degree that these shifts happen within-levels of a hierarchy (e.g., anxiety disorder in youth influencing depression disorder in adolescence) versus cross-levels (e.g., anxiety disorder in youth influencing specific symptoms such as feelings of guilt and insomnia).

**Keywords:** psychopathology, developmental psychopathology, comorbidity, precision psychiatry, hierarchical phenotypes

Most contemporary psychopathology frameworks, whether they are designed for diagnostic (e.g., *Diagnostic and Statistical Manual of Mental Disorders, International Statistical Classification of Diseases and Related Health Problems*), descriptive (Hierarchical Taxonomy of Psychopathology [HiTOP]; Kotov et al., 2017), or mechanistic research purposes (e.g., Research Domain Criteria [RDoC]) embrace a hierarchical perspective of mental health. That is, individual facets (e.g., specific symptoms such as sad mood, RDoC subconstructs such as reward satiation) are nested within broader groupings (e.g., disorders such as depression, RDoC constructs such as

reward processing), both of which communicate nonredundant information. For example, different levels of these hierarchies (e.g., p-factor vs. depression symptoms vs. fatigue) can have different pathophysiological correlates, responses to treatment, and stabilities over time (Moriarity et al., 2026).

Overlaying a hierarchical structure to the issues of multifinality presented by Caspi et al. (2026) invites the question: To what extent do developmental changes operate through within-level (i.e., the degree that anxiety as a disorder in youth might manifest as depression in adolescence) versus cross-level shifts in psychopathology (using the HiTOP framework: the sexual problems subfactor leading to intimacy avoidance within the lower-order homogenous symptom components/maladaptive traits level). Embracing transdiagnostic studies, as encouraged by the target article, is essential to exploring the phenotyping specificity that may lie behind lack of developmental stability in the diagnoses noted by Caspi et al.

Distinguishing between “within-level” and “cross-level” movement is essential to understand the scope of flow between mental health constructs and build the foundation for precision developmental psychopathology. We also cannot—without definitively mapping developmental sequences of interest—elaborate on characteristic developmental patterns that would help to assess risk (e.g., as part of increasing efforts to conduct mental health screenings in schools; Connors et al., 2022).

Aidan G. C. Wright served as action editor.

Daniel P. Moriarity  <https://orcid.org/0000-0001-8678-7307>

Jamie L. Hanson  <https://orcid.org/0000-0002-0469-8886>

Daniel P. Moriarity contributed equally to writing—original draft and writing—review and editing and served in a supporting role for conceptualization. Jamie L. Hanson served as lead for conceptualization and contributed equally to writing—original draft and writing—review and editing.

Correspondence concerning this article should be addressed to Daniel Moriarity, University of Pennsylvania, Department of Psychology, NBS, Room 453, 425 South University Avenue, Philadelphia, PA 19104, United States. Email: [dmori@upenn.edu](mailto:dmori@upenn.edu)

These complications extend to an understanding of mechanisms and causal pathways. Continuing the target article's example using the immunopsychiatry of depression, interleukin-6 partially mediates the association between anxiety and subsequent depression among adolescents who perseverate on unpleasant emotions (Moriarty et al., 2018). Critically, different components of this pathway may operate at different hierarchical levels. Given both the established comorbidity between anxiety and depression (Fava et al., 2000), the fact that youth anxiety often precedes depression (Cummings et al., 2014; Starr et al., 2016), and factor analytic studies highlighting a shared nonspecific component of anxiety and depression (Clark & Watson, 1991), some of the anxiety to depression risk may be occurring at the syndromal level (i.e., within-level pathway). Furthermore, given that negative affect upregulates inflammatory signaling (Carroll et al., 2011; Moons et al., 2010), it is plausible that the syndrome of anxiety impacts inflammatory biology. However, the pathway linking inflammation to depression symptoms appears more specific. The developmental neuroimmune network model (Nusslock et al., 2024) suggests inflammation preferentially influences particular symptoms: fatigue, appetite dysregulation, and anhedonia. Importantly, these symptoms are not depression exclusive. They span diagnostic boundaries, creating biological pathways to the diverging trajectories Caspi et al. (2026) report. A single mechanistic process may thus operate across levels (e.g., anxiety syndrome to inflammation to specific transdiagnostic symptoms) in ways that can catalyze components of new diagnostic constructs.

Such unknowns impede clinical translation. For example, risk prediction models developed from single diagnoses may fail to identify individuals whose risk "decks" may include developmental liability to shifting constructs. Similarly, interventions developed on the concept of "depression" may not be successful if the underlying developmental trajectory is unlikely to remain specific to the depression syndrome (and thus, require a different combination of skills to prevent activation of new problems). Motivated by Caspi et al.'s (2026) results and the research directions they suggest, we propose three complementary strategies to more richly interrogate the dynamic, multidimensional nature of mental health.

First, leverage the transdiagnostic data Caspi et al. (2026) advocate for to conceptualize any one person's life course as a multivariate time-series across disorders. Instead of modeling depression, anxiety, and other problems as separate univariate trajectories, model each person's "deck" as a multidimensional vector evolving over time. Using the HiTOP framework as an example, this would incorporate different levels such as the p-factor for general psychopathology; intermediate spectra levels of internalizing, externalizing, and thought disorder; and specific subfactors. This allows for an empirical distinction between within-level versus cross-level shifts using techniques such as latent growth-curve models or hidden Markov models. These models could allow for testing whether developmental epochs, life events, or treatments differentially predict within-level versus cross-level movement, which would help identify when it may be effective to target broad transdiagnostic mechanisms versus when interventions targeting more precise outcomes might be beneficial.

Second, biomarkers and other mechanisms should be explicitly modeled in ways that determine the hierarchical specificity of their

associations with mental health (Moriarty et al., 2026). This involves both cross-sectional research focused on co-occurrence and longitudinal research that identifies the extent that changes in mechanisms are associated with (a) change in a singular clinical outcome's severity, (b) changes within levels of the chosen hierarchy, and/or (c) cross-level transitions between levels of the hierarchy. It is important to highlight that due to the resources involved this will often require large, multisite studies (e.g., Dunedin, Adolescent Brain Cognitive Development Study) with longitudinal measures of both potential mechanisms and comprehensive, transdiagnostic assessments of psychopathology.

Third, integration of deck-based profiles into evaluation and implementation of interventions. Consider the context of a developmentally specific targeted early intervention/prevention program. In childhood and adolescence, a person's deck might be externalizing dominant. However, the work outlined above might highlight plausible, transdiagnostic paths based on factors such as family history, cognitive vulnerabilities, and biological profiles. Understanding the plausible paths can empower treatments to (a) use measurement-based care to flag the beginning of potential transitions and (b) create maximally comprehensive treatment plans to treat current problems while developing resources to prevent transdiagnostic shifts.

Caspi et al. (2026) encourage us to contemplate mental health as it is really experienced—not as isolated, stable diagnoses but instead to take a more expansive view that considers it to be a "deck of cards" that is often shuffled and reshuffled throughout the course of a lifetime. For our research to be capable of capturing this nuance, we must move from single-disorder longitudinal designs or cross-sectional snapshots to transdiagnostic, temporally characterized investigations of developmental psychopathology. This could provide a more comprehensive and clinically effective understanding of the dynamic shifts in mental illness and health that many of the people we serve experience during their lives.

## References

- Carroll, J. E., Low, C. A., Prather, A. A., Cohen, S., Fury, J. M., Ross, D. C., & Marsland, A. L. (2011). Negative affective responses to a speech task predict changes in interleukin (IL)-6. *Brain, Behavior, and Immunity*, 25(2), 232–238. <https://doi.org/10.1016/j.bbi.2010.09.024>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding "p." *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, 100(3), 316–336. <https://doi.org/10.1037/0021-843X.100.3.316>
- Connors, E. H., Moffa, K., Carter, T., Crocker, J., Bohnenkamp, J. H., Lever, N. A., & Hoover, S. A. (2022). Advancing mental health screening in schools: Innovative, field-tested practices and observed trends during a 15-month learning collaborative. *Psychology in the Schools*, 59(6), 1135–1157. <https://doi.org/10.1002/pits.22670>
- Cummings, C., Caporino, N., & Kendall, P. C. (2014). Comorbidity of anxiety and depression in children and adolescents: 20 years after. *Psychological Bulletin*, 140(3), 816–845. <https://doi.org/10.1037/a0034733>
- Fava, M., Rankin, M. A., Wright, E. C., Alpert, J. E., Nierenberg, A. A., Pava, J., & Rosenbaum, J. F. (2000). Anxiety disorders in major

- depression. *Comprehensive Psychiatry*, 41(2), 97–102. [https://doi.org/10.1016/s0010-440x\(00\)90140-8](https://doi.org/10.1016/s0010-440x(00)90140-8)
- Kotov, R., Krueger, R. F., Watson, D., Achenbach, T. M., Althoff, R. R., Bagby, R. M., Brown, T. A., Carpenter, W. T., Caspi, A., Clark, L. A., Eaton, N. R., Forbes, M. K., Forbush, K. T., Goldberg, D., Hasin, D., Hyman, S. E., Ivanova, M. Y., Lynam, D. R., Markon, K., . . . Zimmerman, M. (2017). The Hierarchical Taxonomy of Psychopathology (HiTOP): A dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology*, 126(4), 454–477. <https://doi.org/10.1037/abn0000258>
- Moons, W. G., Eisenberger, N. I., & Taylor, S. E. (2010). Anger and fear responses to stress have different biological profiles. *Brain, Behavior, and Immunity*, 24(2), 215–219. <https://doi.org/10.1016/j.bbi.2009.08.009>
- Moriarity, D. P., McArthur, B. A., Ellman, L. M., Coe, C. L., Abramson, L. Y., & Alloy, L. B. (2018). Immunocognitive model of depression secondary to anxiety in adolescents. *Journal of Youth and Adolescence*, 47(12), 2625–2636. <https://doi.org/10.1007/s10964-018-0905-7>
- Moriarity, D. P., Perkins, E. R., & Joyner, K. J. (2026). Hierarchical phenotyping of psychopathology: implications and opportunities for precision psychiatry when biology could be associated with both symptoms and syndromes. *Biological Psychiatry*, 99(2), 104–112. <https://doi.org/10.1016/j.biopsych.2025.04.015>
- Nusslock, R., Alloy, L. B., Brody, G. H., & Miller, G. E. (2024). Annual research review: Neuroimmune network model of depression: A developmental perspective. *Journal of Child Psychology and Psychiatry*, 65(4), 538–567. <https://doi.org/10.1111/jcpp.13961>
- Starr, L. R., Stroud, C. B., & Li, Y. I. (2016). Predicting the transition from anxiety to depressive symptoms in early adolescence: Negative anxiety response style as a moderator of sequential comorbidity. *Journal of Affective Disorders*, 190, 757–763. <https://doi.org/10.1016/j.jad.2015.10.065>

Received November 11, 2025

Accepted November 12, 2025 ■

### E-Mail Notification of Your Latest Issue Online!

Would you like to know when the next issue of your favorite APA journal will be available online? This service is now available to you. Sign up at <https://my.apa.org/portal/alerts/> and you will be notified by e-mail when issues of interest to you become available!

## COMMENTARY

Toward a Third Wave of Transdiagnostic Psychological Therapies:  
Commentary on Caspi et al. (2026)

Tim Dalglish

Department of Psychology and Medical Research Council Cognition and Brain Sciences Unit, University of Cambridge

**A Compelling Case for Heterotypic Continuity**

Caspi et al. (2026) present compelling data that individuals who suffer from mental ill health rarely experience only one diagnosable psychiatric disorder across their life course, but in fact are much more likely to have a unique and fluid mental disorder life history—so-called “heterotypic continuity.” Caspi et al. present new analyses from the seminal Dunedin Study showing that by midlife (age 45), an overwhelming majority of individuals (85%) who have experienced any mental health condition had in fact experienced comorbid diagnoses, with nearly all diagnoses predicting subsequent disorders that cut across diagnostic families (internalizing disorders, externalizing disorders, and thought disorders; see also Caspi et al., 2020). Similarly, they present new analyses from the Danish national register data (building on Plana-Ripoll et al., 2019), which demonstrate that receiving a diagnosis of one mental disorder is associated with a persistently elevated risk (for at least 15 years) of subsequently developing all other major mental disorders.

This marked fluidity across clinical manifestations within lifetime histories is likely to be even more marked than the presented data suggest because even within the minority of individuals who experience recurrence of the same psychiatric diagnosis, the clinical presentation in terms of the pattern of specific symptoms and their severity, will likely differ significantly from one episode to the next due to the marked symptomatic heterogeneity within disorders (e.g., Gili et al., 2011; see Dalglish et al., 2020, for a review).

If we accept the case that heterotypic continuity is the norm, the implications for mental health science and for psychological interventions are profound—as Caspi et al. (2026) warn, the evidence “invites a reckoning for psychopathology research” (p. 483).

In this commentary, I concur with Caspi et al. (2026) and argue that that their findings threaten some of the fundamental pillars

supporting the current edifice of mental health science and indeed herald the need for a third wave of transdiagnostic psychological interventions.

**Topping the Pillars of Mental Health Science**


The first pillar under threat is that of *finality*. A seminal notion in developmental psychiatry has been the concept of equifinality—that different starting points and trajectories of risk lead to the same single diagnostic outcome (Cicchetti & Rogosch, 1996).<sup>1</sup> If psychiatric disorders typically morph into different conditions across the life course, a single diagnosis at a single point in time can only provide a phenotypic snapshot, capturing just one moment in a shifting developmental trajectory, not a “final” outcome. The concept of “finality” is therefore obsolete and misleading.

The second somewhat adjacent pillar that begins to look shaky is the traditional notion of a “control group.” In their discussion, Caspi et al. (2026) articulate how their data undermine the traditional case-control design where a specific psychopathology group of “cases” is compared with a group of ostensibly healthy “controls” based on single-point-in-time diagnostic assessments. The evidence now suggests that the specific psychopathology is likely just a one-time snapshot and that an individual could present with an altogether different psychopathology on another testing occasion or indeed with no disorder at all, transitioning from being a “case” to being a “control.”

A partial solution is to include as “controls” only those individuals who have never suffered from a psychiatric condition. However, the Dunedin data indicate that by midlife only 14% of the sample had experienced sustained mental wellness of this kind. This already low proportion is likely to dwindle further as the sample is tracked from midlife into older age. This suggests that sustained mental wellness is very much an atypical experience and that there are important resilient features of this subsample that merit investigation in their own right. This renders this minority subsample inappropriate as a traditional “control group” as it is not clear whether any “case-control” differences reflect the atypicality of the “controls,” as opposed to features of the disorders under examination.

<sup>1</sup> And the related concept of multi-finality where the same aetiological factor (such as childhood adversity) can lead to a plurality of ‘final’ outcomes.

Aidan G. C. Wright served as action editor.

Tim Dalglish  <https://orcid.org/0000-0002-7304-2231>

Tim Dalglish served as lead for writing—original draft.

Correspondence concerning this article should be addressed to Tim Dalglish, Department of Psychology and Medical Research Council Cognition and Brain Sciences Unit, University of Cambridge, Downing Street, Cambridge CB2 3EB, United Kingdom. Email: [tim.dalglish@mrc-cbu.cam.ac.uk](mailto:tim.dalglish@mrc-cbu.cam.ac.uk)

A third nearby pillar with subsiding foundations is the notion of specificity as Caspi et al. (2026) address in their discussion. Mental health science has been preoccupied for a long time with the search for delineated prognostic factors relating to specific psychiatric disorder outcomes, whether they be genes, other distal biomarkers, psychological processes, or environmental features. If the same people experience different disorders when followed for long enough, the notion of specificity becomes moot. Rather, the ambition should be to identify the profiles of risk and resilience factors linked to different life-course trajectories of mental ill health.

A fourth pillar under threat is the traditional concept of relapse and recurrence of individual disorders (e.g., depression). The high rates of sequential comorbidity indicate that a focus on relapse/recurrence of any one disorder is simply too narrow to capture the typical underlying mental health profile and that the sequential morphing from one disorder to another is just as important.

The fifth pillar that is undermined is the traditional distinction between treatment and prevention, as Caspi et al. (2026) discuss. If nearly every disorder is temporally linked to predicting every other disorder, focusing solely on the treatment of a current diagnosis while mostly ignoring the high likelihood of future manifestations (through sequential comorbidity) is clinically unsustainable. The data suggest that any responsible clinical management should always reactively “treat” the current presentation, while also seeking to prevent future possible manifestations. Such prevention efforts, moreover, need to go beyond the current focus on relapse prevention of the same disorder and embrace the likelihood that future manifestations will take the form of completely different disorders.

The implications of the evidence in Caspi et al. (2026), for some of the core pillars of mental health science, are of course also echoed in the discussions and debates about the most central pillar—the psychiatric diagnostic rubric more generally. This discourse has given rise to a new emphasis on transdiagnostic discovery science and nosology (where the case for a p-factor is key component of the Caspi et al., 2026, argument) and interventions. It is the implications for the latter than I briefly turn to in conclusion.

### Toward a Third Wave of Transdiagnostic Psychological Therapies

Caspi et al. (2026) draw out a number of clinical implications of their findings. They highlight the case for a transdiagnostic approach that also pays careful attention to an individual’s developmental and familial context. As noted, they also advocate for the application of clinical staging models (e.g., McGorry et al., 2006) that take account both of any current clinical presentation that requires remediation as well as of risk factors that can be targeted to prevent putative future clinical manifestations. The clinical implications that Caspi et al. draw out, taken together, I contend, herald the need for a new “third wave” of transdiagnostic psychological interventions.

The first wave of transdiagnostic approaches comprised compelling and potent so-called “universal interventions,” often characterized as “one size fits all” protocols (Dalgleish et al., 2020; Schaeuffele et al., 2021). These therapies sought to overcome the fragmentation of established disorder-specific protocols by targeting pervasive factors such as neuroticism, across a range of

different diagnoses without adapting the content to the individual (e.g., Sauer-Zavala & Barlow, 2021). The second wave of transdiagnostic psychological interventions recognized that these universal approaches, by design, lacked the potency of personalized therapy, while potentially delivering unnecessary intervention components. This second wave shifted the focus toward personalization by tailoring treatment through the selection and sequencing of specific intervention components or modules based on a patient’s unique presentation, symptom clustering, and severity at a given point in time (Dalgleish et al., 2020; Schaeuffele et al., 2021).

These first two waves have generated a number of potent clinical interventions (see Cuijpers et al., 2023). The proposed third wave would build on these while taking account of the Caspi et al. (2026) data and arguments concerning the dynamic and fluid nature of psychopathology as a sequence of shifting conditions over the life course, rather than static diagnoses. This proposed third wave of psychological interventions would comprise a hybrid approach that combines the bespoke selection of second wave treatment modules to address an individual’s current clinical difficulties, with delivery of universal (first wave) elements targeting an individual’s pluripotent risk profile for transdiagnostic future problems (Shah et al., 2022). This proposed third wave would therefore incorporate the philosophy of clinical staging approaches with the exact balance of bespoke modular “treatment” components versus universal “prevention” components determined by the individual’s current stage and severity of difficulties (McGorry et al., 2006; Shah et al., 2022).

### References

- Caspi, A., Houts, R. M., Ambler, A., Danese, A., Elliott, M. L., Hariri, A., Harrington, H., Hogan, S., Poulton, R., Ramrakha, S., Rasmussen, L. J. H., Reuben, A., Richmond-Rakerd, L., Sugden, K., Wertz, J., Williams, B. S., & Moffitt, T. E. (2020). Longitudinal assessment of mental health disorders and comorbidities across 4 decades among participants in the Dunedin birth cohort study. *JAMA Network Open*, 3(4), e203221. <https://doi.org/10.1001/jamanetworkopen.2020.3221>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, 135(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, 8(4), 597–600. <https://doi.org/10.1017/s0954579400007318>
- Cuijpers, P., Miguel, C., Ciharova, M., Ebert, D., Harrer, M., & Karyotaki, E. (2023). Transdiagnostic treatment of depression and anxiety: a meta-analysis. *Psychological Medicine*, 53(14), 6535–6546. <https://doi.org/10.1017/S0033291722003841>
- Dalgleish, T., Black, M., Johnston, D., & Bevan, A. (2020). Transdiagnostic approaches to mental health problems: Current status and future directions. *Journal of Consulting and Clinical Psychology*, 88(3), 179–195. <https://doi.org/10.1037/ccp0000482>
- Gili, M., Garcia-Toro, M., Vives, M., Armengol, S., Garcia-Campayo, J., Soriano, J. B., & Roca, M. (2011). Medical comorbidity in recurrent versus first-episode depressive patients. *Acta Psychiatrica Scandinavica*, 123(3), 220–227. <https://doi.org/10.1111/j.1600-0447.2010.01646.x>
- McGorry, P. D., Hickie, I. B., Yung, A. R., Pantelis, C., & Jackson, H. J. (2006). Clinical staging of psychiatric disorders: A heuristic framework for choosing earlier, safer and more effective interventions. *Australian*

- and *New Zealand Journal of Psychiatry*, 40(8), 616–622. <https://doi.org/10.1080/j.1440-1614.2006.01860.x>
- Plana-Ripoll, O., Pedersen, C. B., Holtz, Y., Benros, M. E., Dalsgaard, S., de Jonge, P., Fan, C. C., Degenhardt, L., Ganna, A., Greve, A. N., Gunn, J., Iburg, K. M., Kessing, L. V., Lee, B. K., Lim, C. C. W., Mors, O., Nordentoft, M., Prior, A., Roest, A. M., . . . McGrath, J. J. (2019). Exploring comorbidity within mental disorders among a Danish national population. *JAMA Psychiatry*, 76(3), 259–270. <https://doi.org/10.1001/jamapsychiatry.2018.3658>
- Sauer-Zavala, S., & Barlow, D. H. (2021). *Neuroticism: A new framework for emotional disorders and their treatment*. Guilford Press Publications.
- Schaeuffele, C., Schulz, A., Knaevelsrud, C., Renneberg, B., & Boettcher, J. (2021). CBT at the Crossroads: The rise of transdiagnostic treatments. *International Journal of Cognitive Therapy*, 14(1), 86–113. <https://doi.org/10.1007/s41811-020-00095-2>
- Shah, J. L., Jones, N., van Os, J., McGorry, P. D., & Gülöksüz, S. (2022). Early intervention service systems for youth mental health: Integrating pluripotentiality, clinical staging, and transdiagnostic lessons from early psychosis. *The Lancet Psychiatry*, 9(5), 413–422. [https://doi.org/10.1016/S2215-0366\(21\)00467-3](https://doi.org/10.1016/S2215-0366(21)00467-3)

Received December 14, 2025

Accepted December 17, 2025 ■

### Members of Underrepresented Groups: Reviewers for Journal Manuscripts Wanted

If you are interested in reviewing manuscripts for APA journals, the APA Publications and Communications Board would like to invite your participation. Manuscript reviewers are vital to the publications process. As a reviewer, you would gain valuable experience in publishing. The P&C Board is particularly interested in encouraging members of underrepresented groups to participate more in this process.

If you are interested in reviewing manuscripts, please write APA Journals at [Reviewers@apa.org](mailto:Reviewers@apa.org). Please note the following important points:

- To be selected as a reviewer, you must have published articles in peer-reviewed journals. The experience of publishing provides a reviewer with the basis for preparing a thorough, objective review.
- To be selected, it is critical to be a regular reader of the five to six empirical journals that are most central to the area or journal for which you would like to review. Current knowledge of recently published research provides a reviewer with the knowledge base to evaluate a new submission within the context of existing research.
- To select the appropriate reviewers for each manuscript, the editor needs detailed information. Please include with your letter your vita. In the letter, please identify which APA journal(s) you “social psychology” is not sufficient—you would need to specify “social cognition” or “attitude change” as well.
- Reviewing a manuscript takes time (1–4 hours per manuscript reviewed). If you are selected to review a manuscript, be prepared to invest the necessary time to evaluate the manuscript thoroughly.

APA now has an online video course that provides guidance in reviewing manuscripts. To learn more about the course and to access the video, visit <http://www.apa.org/pubs/journals/resources/review-manuscript-ce-video.aspx>.

## COMMENTARY

Rethinking the p-Factor: A Model of Cumulative Risk Aggregation  
Across Development—Commentary on Caspi et al. (2026)Andrea Raballo<sup>1,2</sup>, Michele Poletti<sup>3</sup>, and Antonio Preti<sup>4</sup><sup>1</sup> REMEDI Lab Euler Institute, Faculty of Biomedical Sciences, University of Southern Switzerland<sup>2</sup> Cantonal Sociopsychiatric Organisation, Mendrisio, Switzerland<sup>3</sup> Department of Mental Health and Pathological Addiction, Child and Adolescent Neuropsychiatry Service, Azienda Unità Sanitaria  
Locale-IRCCS di Reggio Emilia<sup>4</sup> Department of Neuroscience, University of Turin

The general psychopathology factor, or p-factor, has emerged as a latent structure capturing shared liability across mental disorders. However, it may reflect more than statistical covariance and instead index a developmental vulnerability architecture, an emergent feature shaped by genetic, social, and environmental interactions over time. Viewed in this way, the p-factor represents the trace of a deeper risk process unfolding across generations. This perspective supports integrative models of psychopathology and motivates transdiagnostic, network-based approaches to prediction and prevention.

**General Scientific Summary**

This commentary argues that the p-factor is not merely a statistical artifact of comorbidity but reflects a developmentally unfolding and intergenerationally transmitted network of shared genetic, biological, and environmental risks. It conceptualizes general psychopathology as emerging from dynamic feedback processes that consolidate vulnerability across the life course and family context. The framework integrates developmental psychopathology and clinical staging to support transdiagnostic and preventive approaches to mental disorder research and intervention.

*Keywords:* p-factor, developmental vulnerability, risk factors, risk aggregation, psychopathology


Caspi et al.'s (2026) articulation of the p-factor represents a major synthesis in psychopathology research. In the updated intergenerational and developmental account, this latent structure is substantiated through large-scale registry and longitudinal data showing that disorders across internalizing, externalizing, and thought domains co-occur within individuals, recur across the life span, and aggregate within families beyond diagnostic boundaries.

This pattern challenges the long-standing etiological assumption that distinct disorders reflect distinct causes. Instead, shared genetic and environmental vulnerabilities as well as their dynamic interactions constitute the crucial ontogenetic structure of the p-factor, reflecting an emergent property of life-course processes that intertwine genetic, neurodevelopmental, and psychosocial risks across generations (Caspi et al., 2026).

From a clinical-developmental standpoint, this view invites to conceptualize the translation of the p-factor from a statistical correlational structure (Caspi et al., 2014) to a developmentally evolving network of mutually reinforcing risks. The interaction between genetic liability and early childhood adversities unfolds along development through progressively more evident phenotypic features and more severe adjustment difficulties, that at a certain point along the developmental trajectory breaks the clinical threshold with earliest symptomatic manifestations that, without external interventions respect to the family environment, continue or worsen along development, reaching a trait-like threshold toward adolescence, that is, when the p-factor begins to be statistically detected as present and thereafter stable (Allegrini et al., 2020).

During adolescence, neurobiological plasticity intersects with environmental instability: Stress-reactivity systems mature, reward

Aidan G. C. Wright served as action editor.

Andrea Raballo  <https://orcid.org/0000-0003-3709-4111>

Andrea Raballo is supported by the Swiss State Secretariat for Education, Research and Innovation (SERI), SERI Contract number 23.00413, as part of the broader Horizon Europe FAMILY “Understanding and predicting the intergenerational transmission of mental illness” consortium.

Andrea Raballo served as lead for supervision, validation, and writing–review and editing and contributed equally to conceptualization. Michele Poletti contributed equally to conceptualization and writing–original draft. Antonio Preti served as lead for writing–review and editing and contributed equally to conceptualization.

Correspondence concerning this article should be addressed to Andrea Raballo, Faculty of Biomedicine, Università della Svizzera Italiana, Via La Santa 1, 6900 Lugano, Svizzera. Email: [andrea.raballo@usi.ch](mailto:andrea.raballo@usi.ch)

circuitry recalibrates, and social hierarchies become salient. In this context, individuals with elevated p-factor may display multifinality—the tendency for a single liability to manifest as different phenotypes depending on contextual exposures—and equifinality, where distinct early insults converge on similar psychopathological outcomes (Cicchetti & Rogosch, 1996). From a systems-developmental viewpoint, the statistical correlations among diagnostic domains can be seen as feedback loops linking behavior, environment, and biology. For example, externalizing behavior in adolescence (e.g., impulsivity, substance use) may elicit punitive parenting, social rejection, or academic failure, which in turn reinforce internalizing symptoms such as depression or anxiety. Over time, these interacting vulnerabilities coalesce into a self-organizing risk network—a clinical analogue of the latent p-factor.

### Intergenerational Aggregation: Genetic and Environmental Coupling

One of the most compelling contributions of Caspi et al. (2026) is their demonstration of transdiagnostic assortative mating and intergenerational transmission of general liability. Parents with a history of mental disorders tend to pair with partners who also have mental health vulnerabilities, although not necessarily the same disorder. This assortative mating amplifies both genetic and environmental loadings for offspring, creating a compound inheritance of risk.

This intergenerational pattern can be interpreted as a mechanism of developmental coupling—a process by which genetic propensities and social environments align to stabilize vulnerability across generations. Genetic liabilities for neuroticism, impulsivity, or cognitive disorganization may not act in isolation but rather interact with partner selection, socioeconomic disadvantage, and parenting behaviors to shape the family's developmental ecology.

Polygenic risk scores capture only a portion of this inherited vulnerability. Caspi et al. (2026) argue that polygenic risk scores underestimate the true scope of intergenerational transmission because they omit gene–environment correlations and social inheritance—that is, the transmission of adverse environments alongside genetic material. For instance, children of two parents with psychiatric histories may inherit both a higher polygenic burden and an increased likelihood of exposure to household instability, poor parental mental health, poor parenting, and reduced social capital. Thus, the statistical structure of the p-factor reflects the coupled inheritance of multiple, correlated liabilities—genetic, epigenetic, and psychosocial—which converge developmentally to elevate overall risk.

### Early Environmental Embedding: Developmental Origins of Health and Disease and the Biological Imprint of Risk

The developmental origins of health and disease (DOHaD) framework provides an essential biological bridge between the statistical and clinical interpretations of the p-factor. DOHaD posits that early environmental exposures—particularly in utero and early childhood—shape long-term health trajectories through mechanisms of epigenetic programming, stress-system calibration,

and metabolic and neural plasticity (Gluckman & Hanson, 2006; O'Donnell & Meaney, 2017).

In the context of the p-factor, the DOHaD hypothesis suggests that early adversity biologically embeds a general liability for psychopathology. Prenatal stress and exposures, malnutrition, maternal depression, or exposure to inflammation may disrupt neurodevelopmental pathways governing emotional regulation and cognitive control, that is, core endophenotypic substrates shared across disorders. These alterations bias the individual toward a general vulnerability to stress and maladaptation, which later expresses as co-occurring mental disorders.

Caspi et al.'s (2026) intergenerational data corroborate this interpretation: Offspring of parents with high p-factor are not only genetically predisposed but also prenatally and postnatally exposed to adverse milieus. Such convergence of biological and psychosocial embedding exemplifies the developmental canalization of p-factor, where early environmental perturbations steer developmental trajectories toward a narrower range of maladaptive outcomes.

### The Clinical Staging Model: Progressive Risk Aggregation

The clinical staging model (Hickie et al., 2013; McGorry et al., 2006) offers a translational framework for mapping the unfolding of the p-factor over time. This model conceptualizes psychopathology as a progressive process, beginning with nonspecific risk states and advancing toward discrete syndromes and chronic impairment.

Caspi et al.'s (2026) developmental findings can be directly mapped onto this staging sequence:

- *Stage 0: Early Risk Exposure.* Genetic loading, prenatal adversity, and early-life stress constitute a preclinical stage where the groundwork for the p-factor is laid. Here, DOHaD mechanisms—such as hypothalamic–pituitary–adrenal axis dysregulation—create enduring susceptibility to stress and emotional dysregulation.
- *Stage 1: Subthreshold Vulnerability.* During childhood, subtle cognitive, emotional, or behavioral deviations (e.g., attentional dysregulation, irritability, social withdrawal) signal the emergence of endophenotypic vulnerabilities. These manifestations are often transdiagnostic precursors that can evolve into various clinical forms depending on environmental context.
- *Stage 2: Prodromal/Comorbid Emergence.* In adolescence, the network of risks becomes behaviorally and phenomenologically explicit. Externalizing, internalizing, and thought disturbances begin to co-occur, reflecting a transition from latent vulnerability to manifest disorder. Feedback among symptoms (e.g., anxiety-driven substance use) consolidates p-factor's structure at the individual level.
- *Stage 3: Chronic and Multimorbid Disorder.* In adulthood, recurrent or chronic conditions—such as depression coexisting with substance abuse or psychosis—represent the clinical crystallization of the p-factor. Treatment resistance and functional impairment reflect the accumulated impact of the risk network over time.

Through this lens, the p-factor is not merely a statistical proxy of comorbidity but a dynamic marker of stage progression risk.

The higher the individual's p-factor loading, the earlier and more pervasive the aggregation of risks, and the greater the probability of cross-domain symptomatology.

Translating Caspi et al.'s (2026) findings into developmental psychopathology entails reframing risk factors as nodes within an evolving network rather than as independent predictors. Nodes as genetic predispositions, temperament, parenting quality, socioeconomic adversity, peer influences—are interdependent and self-reinforcing.

For example, a child's impulsivity (partially heritable) may elicit harsh parenting, which increases emotional reactivity and depressive symptoms, thereby fostering both externalizing and internalizing vulnerability. Over time, these mutual amplifications create statistical covariance among disorders, that is, the empirical signature of p-factor.

Clinically, this implies that interventions targeting isolated symptoms or risk factors may be insufficient or less effective to break the stable unfavorable and maladaptive equilibrium. Instead, effective prevention may require network disruption strategies—such as supporting parental sensitivity, reducing social disadvantage, and bolstering self-regulatory capacities early in life—to weaken the structural cohesion of the p-factor network before it consolidates.

Moreover, negative social determinants such as poverty, early stigma, and marginalization contribute in perpetuating the p-factor across generations. These social determinants operate both directly (through exposure to adversity) and indirectly (by shaping access to education, health care, and supportive environments). Stigma in particular functions as a social amplifier: Children of parents with mental illness may internalize feelings of shame or exclusion, which in turn augment internalizing symptoms and social withdrawal. Chronic exposure to stigma-related stress has been shown to induce epigenetic modifications in glucocorticoid receptor pathways (Pariante, 2017), further linking the social and biological substrates of the p-factor.

### Clinical Implications: Toward Transdiagnostic and Preventive Interventions

Clinically, recognizing the p-factor as a marker of generalized liability rather than disorder-specific pathology calls for a transdiagnostic approach to assessment and intervention. Screening should focus on early indicators of multidomain vulnerability—such as emotion dysregulation, cognitive inflexibility, and impaired stress tolerance—rather than waiting for categorical syndromes to emerge.

Moreover, interventions should be developmentally timed to intercept the aggregation of risks.

Early-life strategies (e.g., maternal mental health support, reduction of prenatal stress, parenting programs) can mitigate the intergenerational embedding of the p-factor. In adolescence, transdiagnostic therapies with broad targets as emotional regulation, cognitive control, and social connectedness may disrupt the consolidation of comorbidity.

At the population level, addressing social inequities—poverty, housing instability, and educational exclusion—constitutes a form of the p-factor prevention, as these determinants both shape and are shaped by the general liability for mental disorders.

Overall, a developmental account of the p-factor converges conceptually with the DOHaD hypothesis and the clinical staging framework into an integrated theory of progressive risk aggregation:

- DOHaD explains how early exposures biologically embed vulnerability.
- Clinical staging describes how these vulnerabilities manifest phenotypically over time.
- The p-factor quantifies the covariance structure resulting from these processes.

Together, such triad outlines a lifespan and transgenerational model of psychopathology: Risk factors emerge and cluster in early development, interact through feedback processes, and eventually stabilize as chronic multimorbidity, both within individuals and across generations. 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.

### References

- Allegrini, A. G., Cheesman, R., Rimfeld, K., Selzam, S., Pingault, J. B., Eley, T. C., & Plomin, R. (2020). The p factor: Genetic analyses support a general dimension of psychopathology in childhood and adolescence. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *61*(1), 30–39. <https://doi.org/10.1111/jcpp.13113>
- Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., Meier, M. H., Ramrakha, S., Shalev, I., Poulton, R., & Moffitt, T. E. (2014). The p factor: One general psychopathology factor in the structure of psychiatric disorders. *Clinical Psychological Science*, *2*(2), 119–137. <https://doi.org/10.1177/2167702613497473>
- Caspi, A., Houts, R. M., Tegner Anker, A. S., Richmond-Rakerd, L. S., Andersen, S. H., Theodore, R., Poulton, R., Moffitt, T. E., & Torvik, F. A. (2026). Why psychopathology research should avoid studying one mental disorder at a time: An intergenerational and developmental evidence base for understanding “p.” *Journal of Psychopathology and Clinical Science*, *135*(4), 461–494. <https://doi.org/10.1037/abn0001042>
- Cicchetti, D., & Rogosch, F. A. (1996). Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology*, *8*(4), 597–600. <https://doi.org/10.1017/s0954579400007318>
- Gluckman, P. D., & Hanson, M. A. (2006). *Developmental origins of health and disease*. Cambridge University Press.
- Hickie, I. B., Scott, E. M., Hermens, D. F., Naismith, S. L., Guastella, A. J., Kaur, M., Sidis, A., Whitwell, B., Glozier, N., Davenport, T., Pantelis, C., Wood, S. J., & McGorry, P. D. (2013). Applying clinical staging to young people who present for mental health care. *Early Intervention in Psychiatry*, *7*(1), 31–43. <https://doi.org/10.1111/j.1751-7893.2012.00366.x>
- McGorry, P. D., Hickie, I. B., Yung, A. R., Pantelis, C., & Jackson, H. J. (2006). Clinical staging of psychiatric disorders: A heuristic framework for choosing earlier, safer and more effective interventions. *Australian and New Zealand Journal of Psychiatry*, *40*(8), 616–622. <https://doi.org/10.1080/j.1440-1614.2006.01860.x>
- O'Donnell, K. J., & Meaney, M. J. (2017). Fetal origins of mental health: The developmental origins of health and disease hypothesis. *American Journal of Psychiatry*, *174*(4), 319–328. <https://doi.org/10.1176/appi.ajp.2016.16020138>
- Pariante, C. M. (2017). Why are depressed patients inflamed? A reflection on 20 years of research on depression, glucocorticoid resistance and inflammation. *European Neuropsychopharmacology*, *27*(6), 554–559. <https://doi.org/10.1016/j.euroneuro.2017.04.001>

Received December 18, 2025

Accepted December 26, 2025 ■