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A Developmentally Informed Systematic Review and Meta-Analysis of the Strength of General Psychopathology in Childhood and Adolescence

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Abstract

Considerable support exists for higher-order dimensional conceptualizations of psychopathology in adults. A growing body of work has focused on understanding the structure of general and specific psychopathology in children and adolescents. No prior meta-analysis has examined whether the strength of the general psychopathology factor (*p* factor)—measured by explained common variance (ECV)—changes from childhood to adolescence. The primary objective of this multilevel meta-analysis was to determine whether general psychopathology strength changes across development (i.e., across ages) in childhood and adolescence. Several databases were searched in November 2021; 65 studies, with 110 effect sizes (ECV), nested within shared data sources, were identified. Included empirical studies used a factor analytic modeling approach that estimated latent factors for child/adolescent internalizing, externalizing, and optionally thought-disordered psychopathology, and a general factor. Studies spanned ages 2–17 years. Across ages, general psychopathology explained over half (~56%) of the reliable variance in symptoms of psychopathology. Age-moderation analyses revealed that general factor strength remained stable across ages, suggesting that general psychopathology strength does not significantly change across childhood to adolescence. Even if the structure of psychopathology changes with development, the prominence of general psychopathology across development has important implications for future research and intervention.

Keywords

general psychopathology; p factor; explained common variance; meta-analysis; childhood; adolescence

The *Diagnostic and Statistical Manual of Mental Disorders (DSM;* American Psychiatric Association, 2022) is in its fifth edition with text revisions, and traditionally distinguishes psychopathology into categorical diagnoses. However, there is growing evidence that a more reliable nosology would reflect dimensionality of psychopathology (i.e., spectrum

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syndromes) and would more appropriately account for co-occurrence and covariation among disorders (Caspi et al., 2014; Clark et al., 2021; Lahey et al., 2012; Murray et al., 2016). Different aspects of psychopathology, including internalizing (e.g., anxiety and depression), externalizing (e.g., aggression and rule breaking behaviors), and thought-disordered psychopathology (e.g., obsessions, compulsions, and mania), tend to covary. One review found that 10–20% of preschoolers with oppositional defiant disorder (ODD) diagnoses, an externalizing disorder, also present with internalizing disorders (Boylan et al., 2007). Estimates of the degree of covariation between internalizing and externalizing problems have shown to range from $r = .38–.62$ in a sample of adolescents ages 12–18 (Cosgrove et al., 2011). The strong covariation between internalizing and externalizing psychopathology suggests that higher-order processes may account for their commonality and co-occurrence (Carragher et al., 2015; Caspi et al., 2014; Gluschkoff et al., 2019; Kessler et al., 1999; Kotov et al., 2017). The higher-order factor that accounts for the strong covariation of specific psychopathologies (e.g., internalizing, externalizing, and thought-disorder psychopathology) is referred to as general psychopathology or *p* factor (Caspi et al., 2014).

Some have suggested that including a general psychopathology or *p* factor is necessary to fully conceptualize the structure of psychopathology (Avinun et al., 2021; Lahey et al., 2012). A general psychopathology model posits that a single factor influences all symptoms across a range of known categorical and dimensional diagnoses, while specific psychopathology accounts for what is unique to a given set of symptoms above and beyond the general factor (Caspi et al., 2014; Lahey et al., 2012). General psychopathology, as measured by factor analytic modeling (e.g., bifactor model), is meant to reflect the common variance that influences responses on measures of multiple psychopathology dimensions. This common variance indicates that individual differences in people's levels on some symptoms are concurrently associated (Lahey et al., 2021).

General Psychopathology Modeling Approaches

Co-occurrence and correlation among dimensions of psychopathology provide a reasonable justification for a hierarchical structure of psychopathology, but there is no single acceptable method to structuring a general psychopathology factor model (Lahey et al., 2021). Using factor analysis, there are several accepted methods of describing dimensionality among specific and general psychopathology, including higher-order (also called second-order) models, bifactor models, and modified bifactor models (Carragher et al., 2016; Lahey et al., 2021). A bifactor model of general psychopathology is the most common approach to modeling general psychopathology (e.g., Aitken et al., 2020; Clark et al., 2021; Hankin et al., 2017; Huang-Pollock et al., 2017; Neumann et al., 2016; Sheldrick et al., 2012; Vine et al., 2020; Wade et al., 2019; Waldman et al., 2016). In a bifactor model, individual indicators load onto one specific psychopathology factor. The symptoms additionally load directly onto an orthogonal general factor (see Figure 1, Model A). A traditional bifactor model relies on the general factor to account for commonality among the specific factors, rendering the specific factors to represent the residual correlations among common items in each specific factor, after accounting for what is shared due to the general factor (Lahey et al., 2021). Another modeling approach is the modified bifactor model. Modified bifactor

models have an orthogonal general or *p* factor, like bifactor models, but the specific factors (e.g., internalizing, externalizing, and thought disorder) are allowed to correlate (see Figure 1, Model B). Some have argued that allowing specific factors to correlate provides a more ecologically valid representation of psychopathology (Afzali et al., 2018; Carragher et al., 2016). Psychopathology may also be modeled using a higher-order model. In a higher-order model, symptom or diagnosis indicators load onto only one first-order specific factor. These first-order factors then load onto a higher-order factor, which represents the general factor (see Figure 1, Model C). Despite the lack of consensus on the best modeling approach, there is support for modeling the covariation among psychopathology using factor analytic approaches (Canivez, 2016; Lahey et al., 2015, 2021; Patalay et al., 2015).

The Conceptualization of General Psychopathology

The proliferation and wide acceptance of general psychopathology models is due in part to a growing adoption of hierarchical nosologies, such as Hierarchical Taxonomy of Psychopathology or HiTOP (Kotov et al., 2017). HiTOP and other general psychopathological models have inspired a growing body of research aimed at disentangling the structure and manifestation of psychopathology and how it changes across the lifespan (e.g., Forbes et al., 2019; Gomez et al., 2019; Martel et al., 2017; Murray et al., 2016; Waldman et al., 2016). However, HiTOP and other conceptualizations of general psychopathology were developed largely based on adult samples primarily comprised of 15- to 65-year-olds, and there is uncertainty as to whether this conceptualization generalizes to younger ages because children rarely exhibit symptoms of end-stage psychopathology (Forbes et al., 2019; Kotov et al., 2021). Studies have replicated findings that the *p* factor and specific factors exist in both adults and in children (e.g., Laceulle et al., 2015; McElroy, Belsky, et al., 2018; Olino et al., 2014). Some have argued that HiTOP does not adequately capture developmental changes in behavioral manifestations of psychopathology such as: (a) sex-related differences in depression and antisocial behavior during puberty (Hamlat et al., 2019; Van Hulle et al., 2009); (b) restricted access to elicit substances in childhood (Kotov et al., 2021); or (c) the decreasing base rate of aggressive and rule-breaking behavior in adulthood (Achenbach, 2020). Thus, a hierarchical taxonomy of psychopathology in children might not simply be a translation of the adult model, and instead would require more attention to developmentally informed changes in the presentation and covariation of psychopathology (Kotov et al., 2021).

Criticisms of General Psychopathology Modeling

Hierarchical approaches to modeling psychopathology, such as bifactor modeling, are often criticized for numerous reasons. Some have argued that latent factor modeling approaches make assumptions about the data that may be questionable, such as imprecise explanations and predictions on data supporting weak theories (Fried, 2020; van Bork et al., 2017; Watts et al., 2019). For example, bifactor models of general psychopathology are often favored over unidimensional and other correlated factor models because bifactor models yield the best model fit. However, studies have shown that bifactor models fit well even when there are spurious reasons for it, e.g., random patterns and not valid responses, suggesting that both signal and noise are overfitting the data (Fried, 2020; Haeffel et al., 2022; Reise et

al., 2016; Snyder, Young, et al., 2017). In light of overreliance on goodness of fit and other flaws in bifactor modeling approaches, Bonifay and colleagues (2017) suggested that other bifactor statistics, such as explained common variance, may prove more useful than goodness of fit in evaluating indices of general factor modeling.

Others have argued that the general factor in a bifactor model is merely a methodological artifact. Watts and colleagues (2019) found that general psychopathology factors differ greatly as a function of which indicators are included in the bifactor model. They estimated 15 separate bifactor models that each had a distinct single indicator dropped, leaving a unique combination of 14 indicators in each model. The results indicated that some indicators' loading strength and sign were highly dependent on the presence of the other indicators in the model (Watts et al., 2019). For example, the conduct disorder indicator had a loading strength on the general factor of .63 when tics were excluded, but a strength of −.45 when obsessions were excluded. Evidence from these studies raises questions about whether the general factor of psychopathology might be a methodological artifact and might not be meaningful, when estimated using a bifactor model (Fried et al., 2021; Watts, Makol, et al., 2021). However, there is growing support that the general factor represents severity or comorbidity among items of psychopathology (Fried et al., 2021). To better understand the development of the general factor of psychopathology, it is important to examine the general factor strength across ages.

Measurement of General Psychopathology Strength

Many studies have tested higher-order psychopathology models in children that index the amount of variance accounted for by a general psychopathology factor at a given age or a span of ages across development (e.g., Gomez et al., 2019; Haltigan et al., 2018; Hankin et al., 2017; Levin-Aspenson et al., 2019; Waldman et al., 2016). The proportion of variance in ratings of psychopathology accounted for by the general factor is often called explained common variance (ECV). ECV is considered a reliable estimate of the strength of the general factor, when estimating the relative contributions of both the general and specific factors (Martel et al., 2017; Rodriguez et al., 2016). In this regard, explained common variance is a useful metric from which stability of the strength of the general factor can be estimated when examined longitudinally (Murray et al., 2016; Rodriguez et al., 2016). There is a growing need to examine the strength of the general factor meta-analytically and how it changes across development to better understand the contributions of higher order psychopathology across childhood and adolescence to inform the development of interventions (Forbes et al., 2019; Hopwood et al., 2020; Ruggero et al., 2019).

Developmentally Informed Conceptualizations of Psychopathology

Developmental psychopathology is a framework in which practitioners and researchers study how individuals may or may not develop pathology (e.g., externalizing or internalizing disorders), given social, biological, and psychological risks (Cicchetti, 2020). To this end, it is important to consider the timing of development, new challenges that may arise, and the degree to which the individuals are able to navigate these challenges (Cicchetti, 2020). The earlier and longer that an individual continues along a maladaptive pathway, the more

difficult it becomes for them to return toward a normal developmental trajectory (Nigg, 2006). However, individuals may move between states of pathology and non-pathology functioning across development (Cicchetti, 2020; Cicchetti & Sroufe, 2000).

Behavioral manifestations of psychopathology vary at different ages (i.e., heterotypic continuity; Cicchetti & Rogosch, 2002). For example, externalizing problems in a 3-year-old may take the form of overt behavior (e.g., temper tantrum), whereas in a 16-year-old adolescent, the manifestation may take a more covert form (e.g., substance use; Miller et al., 2009). Changes in the manifestation of psychopathology at roughly predictable ages may reflect changes in the tasks that children face across development. A review by Hankin and colleagues highlighted that within the internalizing spectrum, specific disorders and syndromes follow developmentally informed patterns (Hankin et al., 2016). Separation anxiety and specific phobias are highest at early ages, then decrease in adolescence. Social anxiety and generalized anxiety are most prevalent in middle childhood, but panic related symptoms become most prevalent in adolescence (Beesdo et al., 2009; Costello et al., 2011; Hankin et al., 2016). In summary, there is mounting evidence that there are developmentally informed changes in lower-order spectra of psychopathology that span across diagnoses.

The culmination of the order and consequences of how a child copes with these developmental tasks are called *developmental cascades*, and they are thought to map onto one's course of pathology development (Cicchetti, 2020). From this perspective, some children have successfully surpassed a given milestone, while others have not (Sroufe, 2009). Because less attention has been paid to general psychopathology in childhood from a developmentally informed perspective, little is known about whether changes in manifestation of psychopathology correspond to periods when children typically encounter developmental tasks. One example is that 5–6-year-old children spend more time away from parents at primary school compared to their younger-aged selves and peers, which occurs concurrently with a developmental task where children start demonstrating a desire for more autonomy-seeking behaviors (Cicchetti & Rogosch, 2002; Lahey et al., 2021; Sroufe, 2009, 2016). Therefore, developmental tasks, and when they occur, may provide a useful metric for accounting for individual differences in behavior at given ages.

This shift in the presentation of psychopathology across development has led to many questions about whether there are higher-order factors, such as *p* factor, that might account for why some individuals are more likely to develop pathology than others (Smith et al., 2020). There are two theoretical frameworks that suggest that the general factor changes in strength over development. *Dynamic mutualism* suggests that the general factor represents local interactions of symptoms that directly influence and reinforce one another, resulting in the increase in the strength of the general factor over time, due to an increased number of symptoms and correlations (Caspi et al., 2014; McElroy, Belsky, et al., 2018; Murray et al., 2016). Another theory is that the general factor represents a general liability for psychopathology and is strongest at a young age. This theory, *p-differentiation*, posits that as a child ages, the symptoms of psychopathology differentiate, more specific symptoms emerge, and the strength of general psychopathology decreases (McElroy, Belsky, et al., 2018; Murray et al., 2016; Patalay et al., 2015).

Prior Studies Examining Stability Versus Change in the General Factor

Prior work has examined stability versus change in multiple aspects of general psychopathology, including stability and change of individual differences, structure, and strength. Studies have shown relative stability in individual differences in the *p* factor, even when different measurements of psychopathology are used to assess symptoms and diagnoses, suggesting that *p* factor is relevant and meaningful across development (Smith et al., 2020). Studies have found stability of individual differences in the *p* factor across ages 2 to 14 (β = .52– .76; McElroy, Belsky, et al., 2018), and ages 13 to 15 (β = .86; Snyder, Young, et al., 2017).

Studies have also examined the stability in the structure of the *p* factor, and conflicting evidence has emerged. Castellanos-Ryan and colleagues (2016) found that substance use and internalizing indicator loadings on the *p* factor were stronger at age 16 compared to age 14 years, consistent with changes, and therefore instability, in the structure of the *p* factor across development (heterotypic continuity). By contrast, Snyder and colleagues (2017) found that loadings on the *p* factor were largely invariant from ages 13 to 15 years, suggesting relatively stable structure in the general factor during this adolescent period.

As evidenced by dynamic mutualism and *p*-differentiation theories, there is a lack of consensus of whether explained common variance (ECV) or an equivalent metric of factor strength, is stable, increases, or decreases with age. To our knowledge, only a few longitudinal studies (Castellanos-Ryan et al., 2016; Choate et al., 2022; Constantinou, 2019; McElroy, Belsky, et al., 2018; McElroy, Shevlin, et al., 2018; Murray et al., 2016) have explored the changes in strength of a general psychopathology factor, i.e., ECV, throughout childhood and adolescence. Findings in these studies have varied. Some studies have shown fluctuations of increases and decreases (Choate et al., 2022)—others have shown no change (McElroy, Belsky, et al., 2018)—in general factor strength across childhood and adolescence. Due to inconsistent findings, there is a need to explore changes, i.e., increases, decreases, or stability, in general psychopathology factor strength in childhood and adolescence through a developmental psychopathology lens to better conceptualize and prevent development of psychopathology. Developmental psychopathologists would be interested in understanding the amount of explained common variance and the timing of fluctuations, because these changes may map onto known developmental tasks and circumstances.

One longitudinal study examined explained common variance in an expanded age range of 14–21-year-olds and found that explained common variance in the general factor appeared to increase stepwise (see Figure 2; Choate et al., 2022). Explained common variance from ages 14–16 years slightly decreased (from .60 to .57), then increased from ages 16–18 (from .57 to .71) where it remained until hitting a peak at age 21 (ECV = 0.75), but overall stayed relatively stable across this period (Choate et al., 2022).

A longitudinal study of children ages 2 to 14 years also found fluctuations, but relative stability, in the strength of the general factor across ages; (ECV = .60–.71; McElroy, Belsky, et al., 2018). Taken together, findings from two longitudinal studies suggest that the general

factor accounts for approximately 60–75% of the reliable variance, and that there are modest fluctuations that occur at different developmental periods (see Figure 2; Choate et al., 2022; McElroy, Belsky, et al., 2018).

One review of cross-sectional and longitudinal studies found that, when fit to a nonlinear trajectory, explained common variance in childhood (ages 2–12) showed that the general factor accounted for 56% of explained common variance, whereas in adolescence (ages 13–17), this value declined subtly to 54%, and then increased in adulthood (ages 18–40) to 60%, following a *u-shape* trajectory (Constantinou, 2019). Interestingly, this finding suggests that explained common variance might decrease across childhood and adolescence before increasing into adulthood. Taken together, the studies suggest that there may be small fluctuations in the strength of the general factor in explaining individual differences across the lifespan.

To our knowledge, only one study has conducted a comprehensive systematic review on the changes in explained common variance across early childhood, middle childhood, and adolescence (Constantinou, 2019). However, this review calculated an average explained common variance to estimate general factor strength, rather than a meta-analysis which would have provided confidence intervals in estimations. This review also evaluated general factor strength across development by plotting a study's explained common variance against the study's mean age, rather than a meta-regression. Meta-regression is needed to evaluate whether explained common variance changes across time and as a function of other factors. To our knowledge, no previous meta-analysis has aggregated the relevant literature and used robust multilevel meta-regression to test whether the proportion of variance in ratings of psychopathology differs across early childhood, middle childhood, and adolescence. Nor has such a review been conducted in adults. Fortunately, a growing number of cross-sectional and a few longitudinal child and adolescent studies have generated general psychopathology models using factor analysis, which provide the information needed to conduct a meta-analysis to address this gap in the literature. In summary, there is little consensus from prior work as to whether the strength of the general factor of psychopathology increases, decreases, or is stable across development. Therefore, there is a need for studies that examine general psychopathology to account for developmentally informed changes across childhood and adolescence. However, prior research has shown strong support for differentiation of specific symptoms of psychopathology across development, which would result in a decrease in general factor strength, supporting the *p-differentiation* hypothesis (Choate et al., 2022; McElroy, Belsky, et al., 2018; Murray et al., 2016; Patalay et al., 2015). When paired with evidence that only 10% of mental disorders begin to manifest as observable behaviors at or before age 5, it might be the case that psychopathology is more general at younger ages and then becomes more specific throughout development, leading to a decrease in general factor strength throughout development (Kessler et al., 2005).

The Present Review

The aim of the present meta-analysis is to examine factor analytic models of general psychopathology in children and adolescents (e.g., bifactor, modified bifactor, and higher-order factor models) to determine whether explained common variance, a measure of general

factor strength, changes across childhood and adolescence. We hypothesize that general psychopathology will account for more variance in childhood than adolescence, functionally taking the form of a negative age moderation from a meta-regression analysis, supporting the *p*-differentiation theory. We expect change in general psychopathology strength because previous research has indicated that specific symptom expression and presentations are likely not at their "end-stage" earlier in development and that psychopathology becomes more specified throughout the lifespan (Forbes et al., 2019). Additionally, informants are likely to observe a heterogeneous expression of psychopathology and may be unable to differentiate specific forms of psychopathology at younger ages, resulting in more broad representation of psychopathology. If we find differences in general factor strength across development, a secondary aim of the present review would be to map these differences onto expected developmental tasks (e.g., Sroufe, 2016). If, for example, we find that general factor strength decreases during preschool age, we might investigate whether development of, or challenges to the development of, self-regulation plays a role in this change in psychopathology strength (Sroufe, 2016).

If the hypothesis is not supported, and general factor strength either increases across development or does not change, these results would still have implications for future interventions and research. For example, if general factor strength does not change across development, it would suggest that general psychopathology strength is stable across development and is interpretable as an overall impairment or liability, identifiable (and potentially treatable) from a young age. We anticipate that several factors may interact with general factor strength to alter the slope of factor strength over development. Several exploratory moderators were evaluated in the present review.

Subgroup Sensitivity Analyses

We examine the strength of the general factor and how it differs between subgroups. These analyses include subgrouping by: model type (i.e., bifactor or higher-order factor models), study wave (i.e., timepoint or measurement occasion), studies that have a small or large variability in age at a given wave, developmental period (i.e., preschool, school-age, adolescence), longitudinal studies, studies that established at least partial longitudinal metric invariance, and explained common variance of specific factors (ECVs). The subgroup analyses are motivated by prior research indicating that general factor strength may differ as a function of differences in sample or modeling characteristics. For example, hierarchical models and bifactor models tend to differ in their factor loading strengths (Lahey et al., 2021), and data from longitudinal studies are thought to provide a stronger test of change in factor strength compared to cross-sectional studies (Ringwald et al., 2021, 2022). Additionally, developmental stages—preschool age, school age, and adolescence—and the associated developmental tasks with these key developmental stages have impact on development of psychopathology (Cicchetti, 2020; Cicchetti & Rogosch, 2002). We also examined moderation sensitivity analyses.

Moderation Sensitivity Analyses

As a contrast to the sum-based estimate of ECV, we calculate a mean-based estimate of general factor strength (see Equation 4 in Supplemental Appendix 3) to determine if the

method by which general factor strength is estimated alters the results of the study. This analysis is motivated by concerns that a larger number of indicators on the general factor likely inflates sum-based ECV estimates (Watts, Makol, et al., 2021). Similarly, we conduct additional analyses on modeling approaches, including general factor indicator count, and factor count, to further explore method and modeling related variables (Watts et al., 2019). According to Rodriguez and colleagues (2016), PUC is a metric of how a measurement of a general factor is 'uncontaminated' by multidimensionality due to specific factors, and represents the suitability of the model to assess a general factor of psychopathology. PUC is calculated as the number of correlations explained by the general factor compared to the number of within-specific factor item correlations. PUC was found to moderated the association between ECV across age, where higher PUC resulted in a strong positive slope, where lower PUC resulted in stronger negative slope (Constantinou, 2019). Therefore, we calculated PUC and included it as a moderator to reexamine these prior analyses.

Due to well-established sex-related differences in the development of specific psychopathology; e.g., boys experience more externalizing symptoms whereas girls experience more internalizing symptoms (Hinnant & El-Sheikh, 2013; Mayes et al., 2020; Mesman et al., 2001); and boys show higher levels of general psychopathology than girls (Lynch et al., 2021), we also examine whether general factor strength across development differs by sex. Ratings of psychopathology were collected from parent, self, and teacher reports from either a questionnaire or from a structured clinical interview. Previous research suggests that measure type and informant-related biases influence estimates of general factor strength (Conway et al., 2019; Laceulle et al., 2015; Lahey et al., 2012; Martel et al., 2017). Some have suggested that method effects, such as informants and measure, may account for about 25% of variance in general factor strength (Constantinou, 2019; Cote & Buckley, 1987). Thus, we also examine the strength of the general factor as a function of the measure and informant type. Additionally, we evaluated whether ECV changes across development when including sample size as a moderator and setting the sampling variance to constants.

These moderation analyses are informed by prior research and they aim to elucidate areas for future study. Understanding the degree to which developmental trajectory is associated with differing degrees of general psychopathology risk from a developmental psychopathology perspective, is a novel and important gap in the literature that can be used to inform research and clinical evaluation of child and adolescent psychopathology.

Method

Procedure

We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines for conducting this meta-analysis and reporting findings (Page et al., 2021). See https://osf.io/pyc9r for our PRIMA 2020 checklist.

Search Strategy and Eligibility Criteria

Studies were compiled by the first author with the assistance of a psychology librarian in November 2021 through a systematic search using the following electronic databases:

PsycINFO, PubMED, Embase, CINAHL Plus, Scopus, Web of Science (Core Collection), ProQuest Dissertations and Theses Global. See https://osf.io/d3a8n for full list of search terms from all databases. In total, 3,200 articles were screened for inclusion. Additional description of deduplication and screening procedures are in Supplemental Appendix 1.

Inclusion and Exclusion Criteria

The exclusion criteria were: (1) the study did not report empirical findings (e.g., reviews or meta-analyses); (2) mean participant age in the study was > 18.00 years; (3) the study did not use factor analysis to model psychopathology; (4) internalizing and/or externalizing (or their sub-factors) were not evaluated in the study; (5) the study separately evaluated psychopathology factors (i.e., the study did not examine the covariation of internalizing and externalizing); and (6) the psychopathology model included extraneous latent factors that cannot be categorized as sub-factors of internalizing, externalizing, and/or thought disordered problems (e.g., model includes latent factors of personality, stress, wellbeing, etc.). However, models with latent factors that represented a sub-factor of internalizing or externalizing problems were retained. An example of a sub-factor is the use of both fear and distress in substitution of a single internalizing disorders factor (Martel et al., 2017). Moreover, attention problems and conduct problems may be represented as distinct but related factors that comprise total externalizing problems (Clark et al., 2021; Haltigan et al., 2018; Harden et al., 2020; Neumann et al., 2020; Sheldrick et al., 2012). The sixth exclusion criterion was intended to retain a uniform definition of general psychopathology. Models were limited to what might fall under the scope of Caspi and colleagues' *p* factor, including only factors of psychopathology conceptualized as thought disordered, internalizing, and externalizing dimensions (2014). Models including these three factors have the most empirical support (Bates et al., 2014; Forbes, Sunderland, et al., 2021; Kotov et al., 2017, 2021). Therefore, studies with extraneous non-psychopathology factors (e.g., well-being, stress, and personality dimensions) would introduce heterogenous conceptualizations of general psychopathology, changing the meaning of *p* factor, and were thus excluded.

Title and Abstract Screening

The first and second authors both independently screened all titles and abstracts using Rayyan (Ouzzani et al., 2016). Studies that clearly met eligibility criteria or were inconclusive were passed to the data extraction phase where the full text was reviewed.

Data Extraction Criteria and Study Selection

Prior to undergoing the full data extraction process, inclusion and exclusion criteria were assessed using information from the full text. Information on how exclusion criteria were reported are in Supplemental Appendix 1. A subset of approximately 20% of the studies were independently coded by the first two authors to determine their reliability. Intraclass correlation coefficient (ICC) reliability between the coders ranged from .92–1 on five key variables (e.g., exclusion criteria, general factor strength, sum of squared factor loadings of externalizing, internalizing, and thought disordered specific factors). Following reliability check, all discrepancies were discussed and resolved. Remaining studies were divided among the two coders. The meta-analysis coding manual is available on the Open Science Framework (https://osf.io/fvbsu). If an included study did not contain a table or figure in the

full-text or supplemental material, we requested relevant information from the study authors by email. If the authors did not respond, and there was no other factor loading information available, the study was not included in the present meta-analysis.

Statistical Analysis

Effect Size—The study effect size was taken to be the explained common variance in ratings of psychopathology. Standardized (β) loadings for the specific and general/*p* factor were extracted from a table or figure in the manuscript or supplementary materials. The explained common variance was calculated by dividing the variance explained by the general factor (i.e., sum of squared general factor loadings) by the total reliable variance (i.e., sum of squared general and specific factor loadings) using Equation 1 (Constantinou, 2019; Rodriguez et al., 2016). Reliable variance is similar to a total variance estimation, but reliable variance, as measured in the present meta-analysis, does not include an error estimate. The sum of squared loadings from a given sub-factor are summed to represent the variance explained by the given specific factor.

$$\frac{\left(\sum \beta^2_{Gen}\right)}{\left(\sum \beta^2_{Gen}\right)+\left(\sum \beta^2_{Ext}\right)+\left(\sum \beta^2_{Int}\right)+\left(\beta^2_{TD}\right)} \quad (1)$$

To calculate ECV for higher-order models, an additional step was taken. For higher-order models, we follow path tracing rules (Loehlin, 2003), where the βs between the general factor and the indicator, typically passing through at least a specific factor, are multiplied to derive the value that represents the regression coefficient from indicator to general factor. The specific factor may also require subordinate sub-factors that require similar path tracing multiplication. The derived βs are then calculated into the explained common variance using Equation 1.

Given that the effect size was taken to be a calculated proportion score (*p*), there was no provided sampling variance, therefore one needed to be estimated to provide a metric of standard error to the effect size due to sample size. Based on prior literature (e.g., Moeyaert et al., 2017), we derived the sampling variance for each proportion (*p*) using the proportion score and the sample size (*n*). As seen in Equation 2, the sample size (*n*) appears in the denominator of the square root, indicating that larger samples have less sampling variability compared to smaller samples. Effect sizes are normally distributed and the sample sizes are large enough to justify this method of sampling variance calculation (Moeyaert et al., 2017).

$$\sqrt{\frac{p(1-p)}{n}} \quad (2)$$

Accounting for Nonindependence of Nested Data—Given the prevalence of large cohort study datasets and independent samples used across multiple studies, we accounted

for nonindependence of observations by nesting within a given study, within wave (i.e., prospective measurement occasion or timepoint from a given data source), and within sources of participant data (i.e., large cohort studies or independently collected samples shared by multiple studies). Each unique data source was given a categorical code, e.g., ABCD was given the corresponding code of 1. If the study used, for example, wave 2 of the data source (e.g., wave 2 of ABCD = 11 years of age), then all studies that used the same data source at a given wave were assigned the same corresponding wave code (e.g., 2), in addition to the same data source code (e.g., 1). This method also allowed us to retain as many effect sizes at as many ages as possible, which was essential for testing our primary hypotheses. These methods of nesting data are known to be robust for accounting for nonindependence of data within meta-analyses (e.g., Konstantopoulos, 2011; McCurdy et al., 2020). A multilevel meta-analysis approach in the R package *metafor* was used to derive the pooled statistical effect size, explained common variance, while accounting for the nested structure of the data (i.e., study within waves(s) within data sources(s); Viechtbauer & Viechtbauer, 2021).

Evidence of heterogeneity from effect sizes was examined using the Q statistic found in *metafor*, which outputs a chi-square distribution based on k − 1 degrees of freedom in which k is the number of effect sizes derived from studies to account for between-study variance (Cochran, 1954; Huedo-Medina et al., 2006; Ringwald et al., 2021). Total amount of heterogeneity was calculated by I^2 statistic, a robust estimate of the amount of heterogeneity present in a given dataset (Higgins & Thompson, 2002). Multilevel meta-analyses with nonindependence of data (i.e., nested data) have used the I^2 to determine the percentage of heterogeneity due to between-cluster and within-cluster levels, given nested data (Konstantopoulos, 2011). To calculate I^2, we used analysis script templates provided by experts in multivariate meta-analyses (e.g., Konstantopoulos, 2011; Viechtbauer & Viechtbauer, 2021).

Age-Moderated Changes—The primary goal of the present meta-analysis was to determine whether strength of the general factor of psychopathology changes across childhood to adolescence. Meta-regression moderation analysis was used to calculate the degree to which this strength changes across the mean ages of included studies. Data were nested within a given study, within wave, and within sources of participant data. Sample mean age was examined as a potential moderator in *metafor* to conduct meta-regression analysis (Viechtbauer & Viechtbauer, 2021). A significant age moderation would indicate that the slope of ECV as a function of sample mean age is different from 0. We hypothesized a decrease, or negative slope, of ECV across sample mean ages. If the moderation is not significantly different from 0, that would indicate that general factor strength does not significantly change across ages.

Publication Bias—Currently, multilevel meta-analyses are unable to use graphical and quantitative methods of publication bias (e.g., funnel plot, trim and fill plot) using nested data because these approaches do not adequately account for multiple effect sizes that come from a single study or sample (Assink & Wibbelink, 2016; Rodgers & Pustejovsky, 2021). Nevertheless, we generated a contour-enhanced funnel plot and a trim and fill plot with

analyses using the effect sizes without nesting the data. A traditional funnel plot visualizes the effect size of each study plotted against the standard error, a function of the study's sample size, to determine whether effects from smaller studies are more variable than effects from larger studies (Peters et al., 2008). A contour-enhanced funnel plot includes colored areas of significance thresholds, one showing effects between $p = .05–.1$, and another showing $p = .01–.05$, representing significant deviations from the pooled meta-analyzed effect size (Peters et al., 2008).

A trim and fill plot attempts to correct for asymmetry by estimating the number of studies needed to be imputed on one side of the figure to balance the asymmetry (Duval & Tweedie, 2000; Rodgers & Pustejovsky, 2021). A commonly used method for assessing publication bias, even in multilevel meta-analyses, is Egger's regression test (Egger et al., 1997). The metric of interest is the intercept, β_0, because a regression line through symmetrical data in a funnel plot would have a β_0 not significantly different from 0, whereas asymmetrical data due to small sample size influences will have an intercept significantly different from 0 (Egger et al., 1997). Egger's test tends to overestimate bias and may lead to false positives and thus should be interpreted carefully (Pustejovsky & Rodgers, 2019). Egger's test is calculated by performing a meta-regression with the standard error of the effect size (i.e., sampling variance) as a moderator.

Study Quality—To assess the quality of the included studies, we modified the Downs and Black (1998) checklist for assessing methodological quality. See Supplemental Appendix 2 for the modified study quality checklist and scoring of study quality.

Subgroup Analyses—Several subgroup analyses were conducted to determine if general factor strength differed based on groups. These analyses included: model type (i.e., bifactor or higher-order model), longitudinal studies, studies that established at least partial measurement invariance, study wave (i.e., measurement occasion or timepoint), studies that have a small or large variability in age at a given wave, developmental period (i.e., preschool age, school age, adolescence), explained common variance of specific factors (ECVs), and a mean-based estimate of general factor strength. For each subgroup analysis, we first generated the meta-analytic estimate of ECV without age as a moderator. Second, within each subgroup, we examined age as a moderator to determine whether ECV differed by age. For details on subgroup analyses, see Supplemental Appendix 3.

Moderation Analyses—Several moderation analyses were conducted to determine if the strength of the general factor, and whether the age moderation, differs when including (separately) each of the following factors as a potential moderator: mean-based estimate of general factor strength, general factor indicator count, factor count, sex composition of the sample, informant type, mono- versus multi-informant, whether ratings of psychopathology came from questionnaire versus interview, sample size, and percent uncontaminated correlations. Among the combinations of informants and measures in the meta-analysis, 5 effect sizes—4.5%—derived from models that included multiple informants and both interviews and questionnaires. Nine effect sizes—8.2%—were derived from models that included only questionnaires and had multiple informants, and 10 effect sizes—9.1%—were derived from models that only included interviews and had multiple informants. For each

moderator examined, we first evaluated whether the moderator was associated with the estimate of ECV. Second, we added age as a moderator, to determine whether age was associated with ECV when accounting for a given moderator. For details on the moderator analyses, see Supplemental Appendix 3.

Nonlinear Trajectory of Age

We examined whether the change in general factor strength followed a nonlinear trajectory. We centered age such that the intercept was set at the youngest age, 2 years of age, by subtracting 2 from each age. Next, we squared these centered ages to derive a quadratic term (i.e., centered age^2) that allowed for a test of nonlinear moderation of age. To evaluate whether there was nonlinear age-moderation, the quadratic centered age term was added as a second moderator along with the linear centered age term. To further evaluate the possibility of nonlinear age-moderation, a cubic centered age term (i.e., centered age^3) was added as a third moderator along with the linear and quadratic centered age terms in a separate analysis.

Results

Inclusion of Studies

As shown in Figure 3, 3,200 deduplicated studies were identified. After screening abstracts and titles, 233 studies were sought for retrieval. 63 of these studies were conference poster or symposium abstracts and the authors were contacted for additional details, 10 articles were not in English and their abstracts were translated to determine inclusion, and 6 articles were found to be duplicates of other retrieved studies. Authors were contacted, yielding an additional 14 studies to be assessed for eligibility. Including additional studies from contacted authors, a total of 168 articles were assessed for eligibility. Of these 168 articles, a total of $k = 65$ articles were included in this review. Given the nested data structure in which multiple effect sizes might be found in a given study, a total of 110 distinct effect sizes were derived from the 65 studies. The included studies and a snapshot of study characteristics is shown in Table 1.

Overall Explained Common Variance and Heterogeneity

The 110 distinct effect sizes derived an aggregate effect size representing the proportion of variance in psychopathology ratings accounted for by the general factor of 0.56, $SE = 0.02$, $p < .001$. These results indicate that general psychopathology accounted for approximately 56% of the reliable variance across the included studies. The forest plot is shown in Figure 4. The homogeneity Q statistic ($Q = 250.24$, $p < .001$) indicated significant variability in the 110 individual effect sizes nested within the 65 studies. A total I^2 value of 54.83% indicated that approximately half of the heterogeneity is attributable to the included nested components of data source, wave, and study. By proxy, just under half (45.17%) of the heterogeneity is due to sampling variance. The 54.83% of heterogeneity due to nested components is broken down into 38.11% of heterogeneity accounted for by data source, with the remaining 16.72% accounted for by the study. Study wave did not account for any additional heterogeneity.

Age Moderation Analyses

We hypothesized that general factor strength would decrease across development. Study mean ages ranged from 2–17 years of age, M_{age} (*SD*) = 10.95 (*3.72*). Figure 5 describes the distribution of effect sizes by sample mean age included in the review. Screened studies that had 17- or 18-year-old participants tended to be included in adult samples with a mean sample size of > 18.00, thus there were no studies that included a mean sample size of ≥ 18.0 years of age. Age moderation analysis results yielded a Test of Moderator (*QM*) statistic, *QM*(1) of 0.63, $p = .43$. The slope of ECV as a function of age was $\beta = -0.003$, SE = 0.004, $p = .43$. Findings suggest that general psychopathology strength did not change significantly across childhood and adolescence.

Mean-Based Estimate of General Factor Strength

We also examined general factor strength using the mean (rather than sum) of squared factor loadings, to reduce the impact of the number of indicators on estimates of general factor strength (see Supplemental Appendices 3 and 4). Using the mean of squared standardized factor loadings, the estimate of general factor strength was somewhat smaller (0.34, *SE* = 0.02, $p < .001$). Age moderation analysis results yielded a Test of Moderator (*QM*) statistic, *QM*(1) of 0.96, $p = 0.33$. The slope of the moderation was −0.004, SE = 0.004, $p = 0.33$. These results increase confidence in the finding that, even when accounting for potential ECV inflation due to indicator count, general psychopathology strength does not change significantly across childhood and adolescence.

Publication Bias

The results of the Egger's test of the present meta-analyses indicated an intercept of $\beta_0 = 0.63$, *SE* = .07 95% CI = 0.50–0.76. *t*-test results were $t(108) = 9.40$, $p < .001$. That is, the intercept of the sampling variance as a moderator was significantly different from 0, indicating the possibility of publication bias due to fewer studies with small sample sizes being published compared to larger samples.

A contour-enhanced funnel plot is in Figure 6. Results show that there is likely a bias toward publishing results that indicated larger explained common variance estimates, albeit only a small bias. There was significant variability in explained common variance at different sample sizes, and there was some indication that studies with smaller sample sizes (i.e., larger standard errors) tended to have smaller explained common variance values, depicted on the left side of the plot.

Results from the trim and fill analysis indicate that 8 effect sizes, depicted as white dots in Figure 7, would need to be imputed to render the current findings symmetrical. All imputed points were placed to the right of the plot, resulting in an increased estimate where the general factor accounts for ~60% of total reliable variance; ECV = .60, *SE* = .01, 95% CI = .57–.63. Because these analyses do not account for nesting, they should be interpreted carefully. The results likely indicate that the meta-analysis result of an explained common variance of .56 is likely a slight underestimate, falling just short of the confidence interval of the trim and fill estimate (.57–.63). These values are close enough to one another to suggest that publication bias may exist but does not greatly affect the findings of the present study.

Taken together, the trim and fill and funnel plot provided some evidence that there may be a slight bias such that studies with larger samples published results that indicated larger explained common variance estimates. However, only 8 studies were needed on the right side of the plot to balance the symmetry. Furthermore, the values implied by the trim-and-fill plot were close to .85, which would indicate unidimensionality of the general factor (Forbes, Greene, et al., 2021; Reise & Revicki, 2014; Stucky & Edelen, 2014). Given the low number of studies that have found unidimensionality in the general factor, and that the publication bias analyses did not account for nonindependent effect sizes, the trim and fill and funnel plot results should be interpreted with caution.

Study Quality

We assessed the study quality of included studies using the modified Downs and Black (1998) checklist. Mean study quality had a mean score of .88, $SD = .11$. Scores ranged from .62 to 1.00 and had a median score of .93. See Supplemental Appendix 2 for more information.

A total of $k = 65$ effect sizes were derived from high quality studies, i.e., greater than or equal to a mean study quality score of 88%. Results indicated that the general factor strength accounted for 59% of the reliable variance. The results yielded a $QM(1) = 1.29$, $p = .256$. The slope of moderation was: β −.007, $SE = .006$, $p = .256$. A total of $k = 43$ effect sizes were derived from lower quality studies, i.e., less than a mean quality score of 87%. Results indicated that the general factor strength accounted for 52% of the reliable variance. The results yielded a $QM(1) = .30$, $p = .586$. The slope of the moderation was: β .004, $SE = .007$, $p = .586$. Taken together, results indicate that higher quality studies had stronger general factor strength, but the association between general factor strength and age was not moderated by study quality.

Nonlinear Trajectory of Age

We examined potential nonlinearity in the ECV estimates as a function of age. Neither the quadratic [$QM(2)$ of .85, $p = .65$] nor the cubic [$QM(3) = 1.42$, $p = .70$] terms showed evidence of moderation. Figure 8 depicts a bubble plot of model-implied estimates of ECV as a function of sample mean age, with the size of bubbles corresponding to the sample size.

Moderation and Subgroup Analysis Results

All moderation and subgroup analyses results, regardless of statistical significance, are reported in Supplemental Appendix 4. Below we highlight results that yielded at least trend-level statistical significance ($p \leq .10$).

Developmental Period Subgroups—When analyses separately analyzed whether ECV changes as a function of developmental period, school age (mean age of ≥ 6.00 & < 13.00 years; $k = 54$), and adolescents (mean age of > 13.00 years; $k = 45$) derived a general factor strength of .57 and .56 respectively, and ECV did not significantly change as a function of age.

For preschool age (mean age of < 6.00 years; $k = 11$), general factor strength was .66 and results indicated a significant increase across age within the age range of 2 to 5.9 years; M(SD) = 3.52 (*1.16*). The slope of moderation was: β = .065, $SE = .032$, $p = .043$.

To expand upon these results, we examined whether the mean factor loadings for general, internalizing, and externalizing factors changed across preschool ages, age moderation indicated that the mean internalizing factor loadings decreased across this age range: $QM(1)$ of 4.29, $p = .038$; slopeint: β = −.057, $SE = .027$, $p = .038$. Neither the mean externalizing factor loadings nor mean general factor loadings significantly changed across this age range.

Percent Uncontaminated Correlations (PUC)—When PUC was included as a moderator, greater PUC values were associated with higher ECV at a trend level (β = .304, SE = .160, $p = .057$). However, ECV was not associated with age when including PUC as a moderator.

General Factor Indicator Count—The number of indicators loading onto the general factor ranged from 5 to 116, $M_{\text{indicator}}$ (*SD*) = 27.81 (*28.31*). A greater number of indicators on the general factor was associated with greater ECV (β = .001, $SE = .001$, $p = .020$). ECV was not associated with age when including general factor indicator count as a moderator.

Factor Count—Among the separate analyses on factor count—total, externalizing, internalizing, and thought disorder—only the count of externalizing factors and the presence of a thought disorder factor emerged as at least trend level significant moderators in general factor strength. Having more than one externalizing factor, or subfactors, was associated with greater ECV at a trend level (β = .050, $SE = .029$, $p = .084$). The presence of a thought disorder factor was associated with weaker ECV (β = −.137, $SE = .041$, $p = .001$). ECV was not associated with age when controlling for factor count.

Measure Type Moderation—Among the included studies, 89 effect sizes were derived using results from questionnaires, while 38 were derived using results from structured clinical interview. Several factor analytic models included both questionnaires and interviews. Results from moderation analysis of whether a questionnaire or interview was used indicated that a moderation was present, $QM(2)$ of 6.09, $p = .05$; $\text{slope}_{\text{questionnaire}}$: β = .09, $SE = .04$, $p = .02$. Studies with questionnaire ratings tended to yield a stronger ECV estimate than studies with interviews. ECV was not associated with age when controlling for measure type.

Discussion

In the present meta-analysis, we aimed to determine whether general psychopathology strength changes across childhood to adolescence. The present meta-analysis expands the scope of what is understood about general psychopathology from evidence of aggregated studies to include children as young as 2 and includes information from almost every developmental period in childhood and adolescence (except 1 and 18 years). Included studies ($k = 65$) examined internalizing and externalizing psychopathology factors (or sub-factors) at a minimum, and included a thought disorder factor at a maximum (Caspi et al.,

2014). Standardized factor loadings were used to estimate explained common variance that represents the general factor's strength in relation to the total reliable psychopathology variance, while accounting for interdependencies due to shared data source, waves, and studies. When meta-analyzed, these results showed that general psychopathology accounted for approximately 56% of the reliable variance in ratings of child and adolescent psychopathology across the included studies, and this factor strength did not significantly change across development.

General Factor Strength

While there are no cutoffs for explained common variance values (McElroy, Belsky, et al., 2018), the suggested range to denote that the general factor is the main source of shared variance ranges from .6 or .7, which would indicate high importance of general factor relative to specific factors (Forbes, Greene, et al., 2021; Reise et al., 2013; Stucky & Edelen, 2014), to .85, which would indicate unidimensionality (Forbes, Greene, et al., 2021; Reise & Revicki, 2014; Stucky & Edelen, 2014). At a value of .56, our findings suggest that the general factor is not the main source of the shared variance, but does account for a nontrivial amount of variance of psychopathology symptoms across childhood and adolescence (Rodriguez et al., 2016). One interpretation for this finding is that general psychopathology (i.e., covariation of internalizing, externalizing, and thought disorder) meaningfully represents a considerable proportion of the total symptoms as reported by parents, teachers, secondary caregivers, and self-report across childhood and adolescence. These results support prior literature that has noted that symptom- and syndrome-specific diagnoses and treatments do not adequately cover the entirety of psychopathology, and that a general psychopathology representation would account for the considerable overlap in symptoms (Conway et al., 2019; Forbes et al., 2019; Hopwood et al., 2020; Kotov et al., 2017; Ruggero et al., 2019).

Role of Development in General Factor Strength

Inconsistent with hypotheses, general psychopathology strength did not differ as a function of sample mean age. When allowed to fit a nonlinear trajectory, the model-implied change in general factor strength was near-identical to the linear trajectory, suggesting that the general factor did not fluctuate in its strength at specific ages, and it did not significantly increase or decrease from early childhood to late adolescence when assessed meta-analytically. It is possible that prior studies that found random fluctuations in general factor strength may have captured sampling and measurement error (Watts, Makol, et al., 2021). In the present meta-analysis, general factor strength showed modest but nonsignificant decreases across school age and adolescent ages. By contrast, ECV significantly increased in the preschool developmental period encompassing ages 2 to 6 years. However, additional analyses indicated that these changes in ECV were driven by a decrease across this age range in the average factor loadings on the specific internalizing factor; mean general factor loadings did not change. One potential hypothesis for why psychopathology during the preschool ages may differ from other developmental periods is that many preschool-age children enter daycare or preschool settings and begin to spend more time away from parents. Preschool ages also coincide with a developmental task of more autonomy-seeking behaviors (Cicchetti & Rogosch, 2002; Lahey et al., 2021; Sroufe, 2009, 2016). The number

of effect sizes in the preschool age range was small at only $k = 11$, therefore it is important for future studies to estimate factor analytic models of general psychopathology in preschool age children to replicate these findings longitudinally.

The results suggest that—even with a potentially slight increase in preschool age—general psychopathology is as meaningful in young children as it is in adolescents nearing adulthood. These findings align with previous longitudinal studies (e.g., Choate et al., 2022; McElroy, Belsky, et al., 2018) that found that the general factor strength did not change with age. The findings in this meta-analysis cannot address changes in the general factor within individuals over development. Therefore, we are unable to make any claims about whether there are developmental changes in the level, structure, or strength of the general factor for an individual. Future longitudinal studies will be needed to examine these questions. However, our analysis of only longitudinal studies did not show changes in general factor strength across development.

When conducting a subgroup analysis on longitudinal studies that established at least partial metric invariance, only 4 of the 12 longitudinal studies met this criterion (e.g., Choate et al., 2022; Etkin et al., 2021; Snyder, Young, et al., 2017; Wade et al., 2019). The remaining 8 (e.g., Chen et al., 2022; Huang et al., 2020; McElroy, Belsky, et al., 2018; Neumann et al., 2020; Olino et al., 2018; Riglin et al., 2019; Rijlaarsdam, Cecil, et al., 2021; Tein, et al., 2023) either did not evaluate or attempted and determined that factor loadings of measures of general psychopathology were non-invariant across age. General factor strength also did not show changes across ages among studies that established longitudinal measurement invariance. Additionally, within studies that established longitudinal measurement invariance, there were very little fluctuations in ECV across the ages. Given the scarcity of studies that evaluated, let alone established, measurement invariance, the ability to detect an effect of age among the longitudinal studies in the present meta-analysis is limited. Future studies should evaluate longitudinal measurement invariance to estimate changes in general factor strength from longitudinal designs.

The present findings suggest that the general factor likely represents a variable that is both transdiagnostic and present at all stages of childhood and adolescent development, but we are unable to generalize these findings to the within-person level.

Potential Interpretations of General Factor

The finding that the strength of the general factor did not vary across ages has implications for how researchers and clinicians may conceptualize and interpret the meaning of the general factor. Stability in this factor's strength suggests that the general factor is not differentially strong at specific ages, but potentially: overall impairment (Smith et al., 2020); a risk factor for developing symptoms (Ringwald et al., 2021); or a dimensional alternative to categorical diagnoses conceptualization (Forbes et al., 2019; Kotov et al., 2017; Ringwald et al., 2021). Simply put, the evidence is consistent with the idea that the general factor represents something that influences the presentation of symptoms relatively evenly across development. Therefore, an interpretation that the general *p* factor represents overall impairment would suggest that experiencing general and transdiagnostic difficulties equally

affects children and adolescents, even though specific symptoms—and their frequency or severity—may change at different ages, i.e., heterotypic continuity (Smith et al., 2020). However, the general factor may instead be a statistical artifact, and therefore would not influence presentation of symptoms (Watts, Makol, et al., 2021; Watts, Meyer, et al., 2021).

There are many potential candidates for how we might interpret the general factor of psychopathology given the findings from the present meta-analysis. In a recent systematic review, Lynch and colleagues (2021) found that general psychopathology in young people aged 10 to 24 years of age was associated with a number of risk factors. Among biological risk processes for general psychopathology, they identified: genetic risk for ADHD and schizophrenia (Brikell et al., 2020; Riglin et al., 2020); being male (Riglin et al., 2020; Wade et al., 2018; Wang et al., 2020). They also identified early pubertal timing (Hamlat et al., 2019); and executive functioning deficits (Hatoum et al., 2018; Shields et al., 2019; Wade et al., 2019). Among psychological risk processes for general psychopathology, they identified: high negative affectivity (Deutz et al., 2020; Hankin et al., 2017; Mann et al., 2020; Wang et al., 2020); difficult temperament (Deutz et al., 2020; Levin-Aspenson et al., 2019); and low effortful control (Hankin et al., 2017; Shields et al., 2019). Among social risk processes for general psychopathology, they identified: stressful life events (Hamlat et al., 2019); and maternal depression (Deutz et al., 2020; McCutcheon et al., 2013).

Although the Lynch and colleagues' (2021) review did not include children under 10 years old, these findings highlight the diversity of potential candidates that influence the general factor of psychopathology. We contribute to this literature by describing developmentally informed conceptualizations of the general factor.

Risk of Developing Symptoms of Co-occurring Psychopathology—Plausibly, the findings in the meta-analysis could support the hypothesis that the general factor might represent the likelihood of experiencing co-occurring symptoms of psychopathology remains stable across development provided that an individual shows psychopathology symptoms (Ringwald et al., 2021). Although genetic and environmental risks for developing psychopathology would be higher in some individuals (e.g., Brikell et al., 2020; Chen et al., 2022; Grotzinger et al., 2019; Neumann et al., 2016), the present findings might indicate that, *on a population level*, the risk of developing symptoms of co-occurring psychopathology would be approximately equally likely across childhood and adolescence, rather than at certain age ranges. However, findings from analyses examining developmental periods suggest that there might be a slightly higher risk at younger ages.

Temperamental Negative Emotionality—Another possibility, given that the general factor is proposed to represent what is common among symptoms of psychopathology, is that the general factor and its relatively equal contributions across development might represent temperamental affective behavior. Temperamental affective behavior is easily observed by an informant and found across the lifespan. One aspect of temperamental affective behavior that is present across development and thus a potential interpretation for general psychopathology, is dysregulated emotionality, also called difficulty. Dysregulated emotionality changes in its behavioral manifestations throughout development and has been implicated as a transdiagnostic mechanism of psychopathology (Damme et al., 2020;

Weissman et al., 2019). Particularly at younger ages, dysregulated emotionality has been labeled irritability, a dispositional tendency to respond with anger when faced with slowed or blocked goal attainment (Damme et al., 2020; Wakschlag et al., 2018; Wiggins et al., 2018, 2021). Furthermore, irritability is present throughout development, even through adolescence (Hawes et al., 2020). Negative affect/irritability present at younger ages predicts future psychopathology, even when accounting for the introduction to novel contexts and challenges, e.g., child going to school, seeking more autonomy (Briggs-Gowan et al., 2006). Due to its presence throughout development during the ages assessed in the present meta-analysis, 2 to 17, irritability might be a candidate interpretation for the general factor.

Measurement and/or Informant Effects

Other considerations might include factors exterior to the symptoms themselves, such as method- or informant-related effects. The current review found a few instances where there was meaningful difference in ECV as a function of differences in method or analytic choices. The general factor was stronger in models that included questionnaires compared to models that included interviews. Including an interview weakens the general factor strength across development. An interview is typically administered and interpreted by a trained professional who may be more likely to assess psychopathology objectively. Nevertheless, Constantinou (2019) found that separating questionnaires from interviews and evaluating them in two separate models resulted in a non-significant ECV variability difference.

The weaker general factor strength from interviews may indicate that the use of interviews reduces reporter bias, compared to questionnaires completed by parents, teachers, or self-report. Alternatively, the difference between factor strength from interviews and questionnaires might be due to questionnaires containing more items that load onto the general factor, which would potentially inflate ECV estimations. Alternatively, due to more and diverse questionnaires compared to fewer interviews present in the study, the finding that questionnaires yielded a strong ECV might reflect a more reliable estimate of the general factor (Constantinou, 2019).

Another important consideration is informant-related biases, such as method biases specific to an informant type (e.g., child, parent, teacher). It is plausible that the general factor, what is common among ratings of psychopathology, might represent reporter bias to a degree (Constantinou, 2019; Martel et al., 2017). When Watts and colleagues (2021) compared ECV estimates from mono-informant models to ECV estimates from models that included multiple informants and accounted for method factors of informant type, ECV estimates decreased on average from .68 (range: .53–.80) to .37 (range: .20–.46). The proportion of variance in ECV estimates that were attributable to method factors ranged from .29 to .67 (M = .46). Thus, around half of the variance in ECV estimates may be attributable to method variance. ECV estimates from models that do not control for variance attributable to informant may over-estimate the true strength of the general factor. Applying this adjustment to account for method factors to the present study, one might expect that the true strength of the general factor in the present meta-analysis may be closer to .30 ([1 – 0.46] × 0.56). However, our moderation analyses on informants yielded nonsignificant results. One potential explanation is that while instances of self and parent report were

evenly split, there were only four instances of teacher report, which limited the variability in objective measurement across settings. Prior research has asserted that it is important to assess behavior problems from multiple raters in a given setting (e.g., mothers and fathers in the home), and across different settings (e.g., teachers in schools) to capture context-specific variability in reporting to reduce bias (De Los Reyes & Makol, 2021; Kraemer et al., 2003; Makol et al., 2020). It is important for future studies to examine measurement-related biases to determine the extent to which the general factor represents something other than the covariation of psychopathology.

Measuring General Psychopathology—A previous review had noted that measures differ in their validity and reliability to detect general psychopathology (Constantinou, 2019). Along with explained common variance to detect the general factor strength, another method that is often paired with ECV is percent uncontaminated correlations (PUC). Higher PUC values reflect a higher number of subscales with fewer items in each subscale, making them more suitable for estimating the general factor (Constantinou, 2019). Constantinou's review (2019) examined the interaction between age and PUC in predicting explained common variance values. As noted above, their results suggest that PUC might strongly influence interpretation of strength of the general factor over development (Constantinou, 2019).

In the present meta-analysis, we found that among bifactor models, PUC was associated with higher levels of ECV, which replicated the findings from Constantinou (2019). However, when assessed meta-analytically, including PUC as a moderator did not result in a change in ECV across development. Furthermore, when examining the role of PUC as a moderator of the mean-based estimate of general factor strength, PUC was negatively associated with this composite at a trend level, and there was no significant change in the mean-based estimate of general factor strength across development. One potential culprit that is responsible for these seemingly contradictory results might be due to differences in influence from the number of indicators loading onto the general factor. It is important to consider that PUC is calculated using the number of indicators and number of factors. In fact, the number of indicators loading onto the general factor was positively associated with higher levels of ECV, but not with the mean-based estimate of general factor strength. Therefore, deciding to calculate ECV using the sum, or the average of squared standardized factor loadings may influence the degree to which the number of indicators on the general factor indicator impacts the strength of the general factor. The number of specific factors, specifically of externalizing problems, and whether to include a thought disorder factor will also be important decision points for future research.

Clinical Implications

The stability of the strength of the general factor in ratings of psychopathology across childhood and adolescence suggests that what is common among symptoms of psychopathology, *p* factor, may be detectable from a young age. If the general factor represents a general liability, risk, or negative emotionality present at all stages of life course, then there is a need for early detection of general psychopathology in childhood and the use of domain-general treatment approaches. Examples of domain-general

approaches might include teaching emotion regulation skills and parenting training, which might potentially target and forestall development of end-stage specific psychopathology symptoms (Forbes et al., 2019; Martel et al., 2017).

The findings from the present meta-analysis suggest that emphasizing single symptoms or syndromes will not be sufficient to conceptualize the entirety of presenting concerns for youths seeking psychological treatment (Forbes et al., 2019). Evidence of a robust general factor of psychopathology across development has the potential to motivate the shift away from single-disorder treatment protocols toward transdiagnostic approaches that also better account for heterotypic continuity of problem behaviors, such as the Unified Protocol for Transdiagnostic Treatment for Emotional Disorders for Children and Adolescents (Ehrenreich-May et al., 2017). That is not to say that focusing on lower-order or specific symptoms or their treatment protocols should not also be a primary concern; in reality, we, along with others, argue that it is increasingly important to study homogeneous specific psychopathology concerns (McGrath, 2005; Smith et al., 2003, 2020; Strauss & Smith, 2009).

Higher general factor scores are associated with more functional impairment and an increased risk for suicidal behavior and non-suicidal self-injurious behavior (Haltigan et al., 2018; Hoertel et al., 2015; Lahey et al., 2015, 2021; Pettersson et al., 2018; Sallis et al., 2019). We urge clinicians and researchers, regardless of the presenting concern of the child, to assess for broad ranges of psychopathology (e.g., internalizing, externalizing, thought disordered, and other dimensions) in all clients or research participants. This perspective is in line with suggestions made from supporters of the HiTOP model (Conway et al., 2019; DeYoung et al., 2021; Forbes et al., 2019; Hopwood et al., 2020; Ruggero et al., 2019). We feel that assessing general psychopathology will better capture the full range of covarying symptoms to account for overlaps in symptoms often dismissed as a specific syndrome. Future work needs to develop measures that better account for heterotypic continuity to assess a wide scope of covarying symptoms that suitably estimate a general factor (Harris et al., 2023; Petersen & LeBeau, 2022). Ideally, measures might take the form of a computer adaptive test (CAT) such as the Overall Mental Illness (OMI) screener (Moore et al., 2019) that provide more rapid and accurate assessments to be used in research and clinical settings to develop more transdiagnostic treatment approaches.

One approach for treatment of general psychopathology is a transdiagnostic stepped-care approach to prevention is proposed by Forbes and colleagues (2019). This approach provides a framework for more universal interventions for broad and limited-modifiable risk factors (e.g., harsh parenting, emotional reactivity) at ages 3–6, and increases slightly in specificity at ages 7–10 to incorporate more targeted treatment, with a focus on emergent symptoms in adolescence and through adulthood (Forbes et al., 2019). Evidence from the present meta-analysis shows that general psychopathology strength is stable across ages at the population level and provides support for the need for prevention and early intervention of dimensional psychopathology problems. However, the present review did not account for functional impairment that would be relevant to consider in clinical treatment (Ruggero et al., 2019).

Limitations

Several limitations should be noted when interpreting the results of the present meta-analysis. First, we opted to include only those studies that assessed, at a maximum, internalizing, externalizing, and thought disordered specific factors (or multiple sub-factors representing these specific factors). Studies that included additional factors (e.g., stress, personality, well-being, prosocial behavior) were excluded (e.g., Black et al., 2019), because the conceptual interpretation of general psychopathology would differ greatly as a function of the specific factors from which it was composed. Additional factors, such as maladaptive personality traits, are important to consider because these traits informed the HiTOP structure (Kotov et al., 2017). Our defining characteristics of general psychopathology most closely align with the extant literature in children and adults (e.g., Caspi et al., 2014; Haywood et al., 2021; Lahey et al., 2017; Ringwald et al., 2021). Future studies should also include personality factors in the conceptualization of general psychopathology.

A second limitation is the method by which effect sizes were calculated. There are no single universal methods of factor analytic modeling. We calculated our effect sizes by using the information we had available, the standardized factor loadings to estimate the reliable variance. We ultimately chose the present method because it was a robust approach to estimating explained common variance, a metric of factor strength. The method chosen to calculate the effect size, explained common variance, inflates estimates of factor strength for factors that have more loadings (i.e., the general factor; Reise et al., 2013). However, averaging squared factor loadings, rather than summing them resulted in a reduced general factor strength of 34% in the present review, and these findings also did not significantly change across development. Published studies likely were biased to include only the best fitting version of models, which may have had many indicators. Additionally, variability in factor loadings across studies challenges the comparability of the latent factor itself. Therefore, estimation of ECV may be an overestimate and poses questions about possible interpretation of the general factor. However, because the averaging squared factor loadings also resulted in no change across development, we have further confidence that general factor strength does not change across development. We did not estimate “unreliable” error/residual variance of standardized factor loadings because the correlated nature of higher-order and modified bifactor models pose challenges to interpreting residual variance. Future studies should examine whether residual variance in the indicators or factors affects ECV interpretation, specifically for traditional bifactor models.

A third limitation is that there are potential limits to comparing ECV across models due to significant heterogeneity in methods of model estimation, including: measures, indicator count, factor count and type/number of informants across studies. Several sensitivity analyses elucidated that to some degree all of these were associated with differences in ECV. A higher percent of uncontaminated correlations (PUC) was also positively associated with higher ECV. However, in this analysis, ECV did not substantially change across age. In sum, it is likely that differences in what goes into a model slightly change the meaning of ECV. The findings did not substantively change when examining only longitudinal studies that established longitudinal measurement invariance. Findings from this subset of studies

provide greater confidence that the strength of the general factor does not substantively change over the developmental span.

Fourth, we did not include studies with the mean age of over 18 years of age. This constraint limited the scope of the meta-analysis. Because we found that general psychopathology strength may change in preschool ages, it is possible that general factor strength might also change from adolescence to adulthood. Despite limitations, this was the first systematic review and meta-analysis to examine the change in general factor strength across childhood and adolescence and included robust multilevel meta-analysis methods that allowed estimating explained common variance at the population level, and its changes across development, while accounting for interdependence of data from shared data sources.

Future Directions and Reporting Guidelines

To better evaluate changes in general factor strength, longitudinal studies should test whether the findings from the present meta-analysis replicate after establishing longitudinal measurement invariance. Future studies should evaluate the degree to which questionnaires or interviews differ in their ability to detect general psychopathology. For ease of transparency for future meta-analyses, empirical studies should publish standardized factor loadings, variance-covariance matrices of included factor analysis indicators, and specific modeling methods in-text, in supplementary materials, or on an open-source pre-registration database (e.g., Open Science Framework; OSF). Future studies should also: clearly state the source of the participant sample pool; note whether sample has been included in prior studies; and provide details about data collection, methods, and demographic information about the data source. Furthermore, future research should be dedicated to developing approaches to assessing publication bias using nested data. Future intervention research should target transdiagnostic symptoms, such as difficulty or negative emotionality, starting in young children with the aim of preventing onset of more pronounced specific psychopathology in later years (Damme et al., 2020; Weissman et al., 2019). Future studies should also examine how best to interpret the general factor across developmental periods and the extent to which it involves method biases.

Conclusion

In conclusion, our meta-analytic findings suggest that general psychopathology makes up over half of the total reliable variance in ratings of psychopathology in children and adolescents. The strength of the general factor did not change across childhood to adolescence, suggesting that the strength of higher-order dimensional psychopathology is stable across childhood to adolescence at the population level, with a possible modest increase during preschool age. Research on the strength and stability of the general factor of psychopathology across childhood and adolescence will continue to accumulate, but our meta-analysis shows that the general psychopathology factor is meaningful and represents something that is robustly prominent at all stages of childhood and adolescent development.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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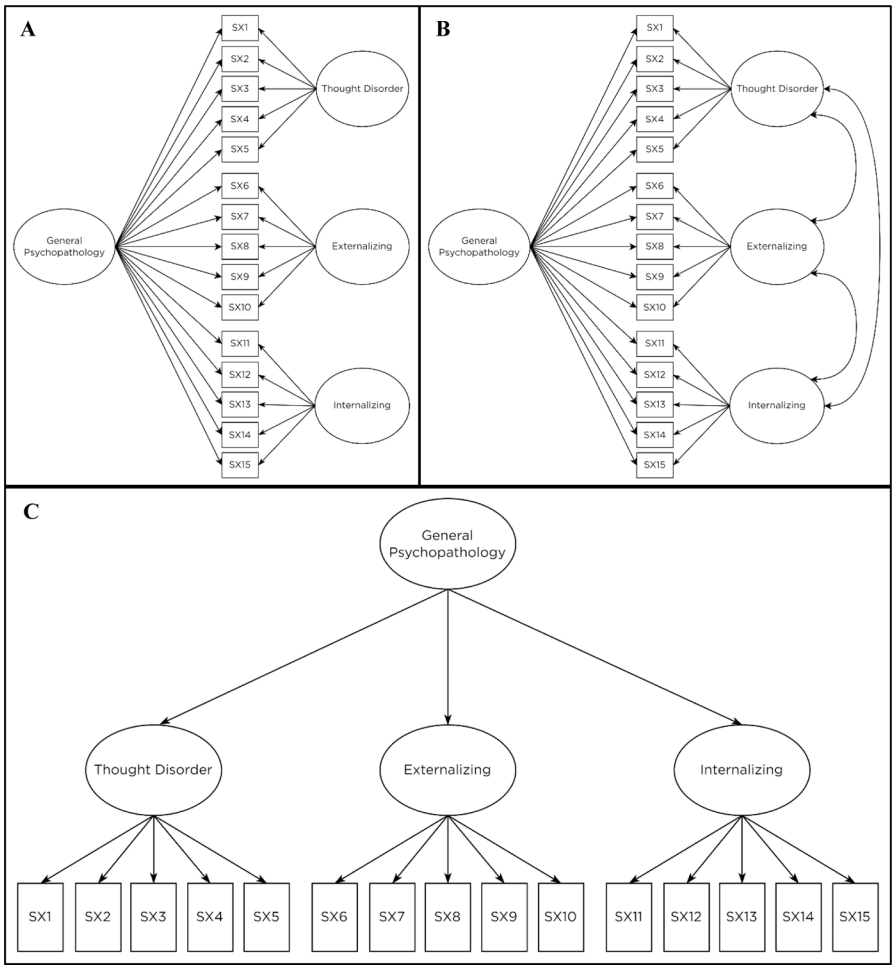

Figure 1.

Representation of Higher-Order, Bifactor, and Modified Bifactor Models

Note. Panel A = bifactor model. Panel B = modified bifactor model. Panel C = higher-order factor model.

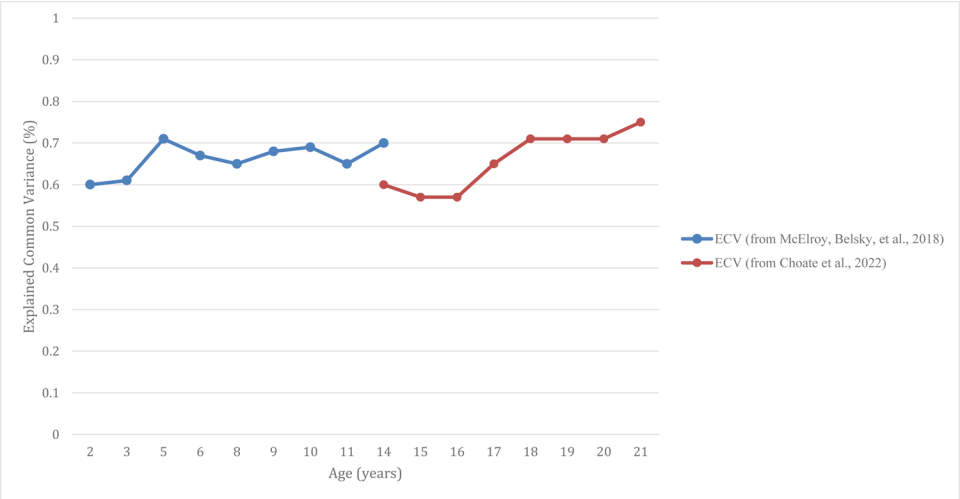

Figure 2.

Two Longitudinal Evaluations of ECV Change

Note. ECV = explained common variance. Data from McElroy, Belsky et al., 2018 included a longitudinal sample of 2- to 14-year-olds. Data from Choate et al., 2022 included a separate sample of 14- to 21-year-olds.

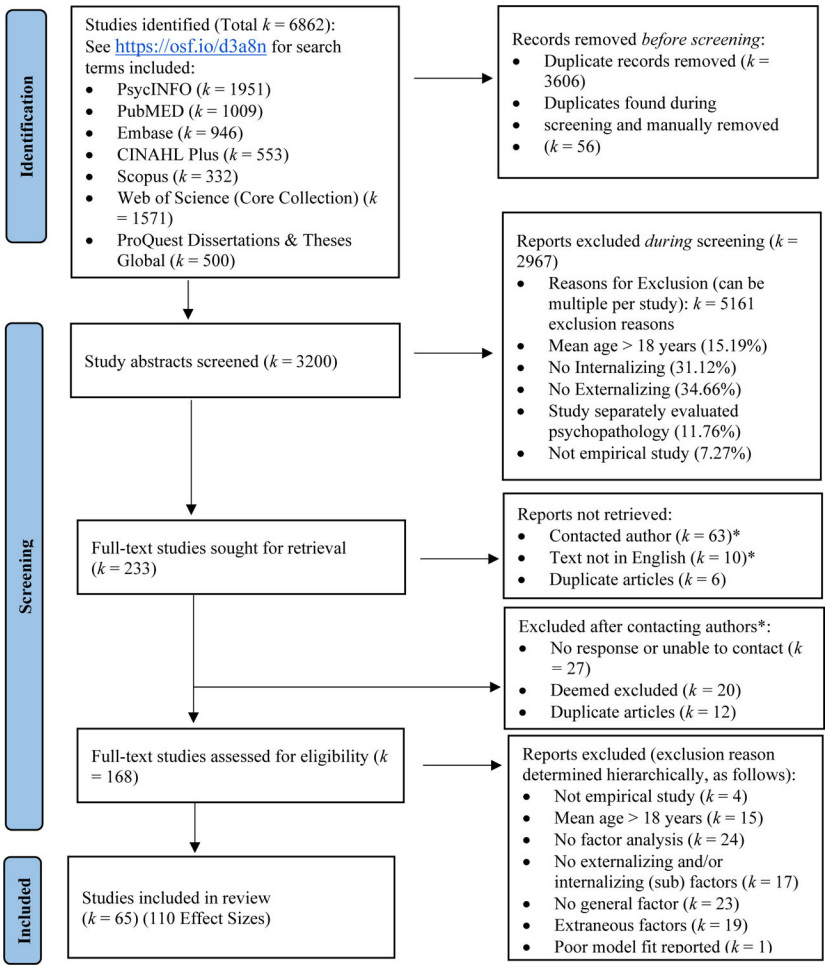

Figure 3.
PRISMA Diagram

Note. Rayyan was used for reviewing study abstracts. REDCap was used for full-text eligibility assessment and study data extraction. Diagram template from Page et al., (2021).

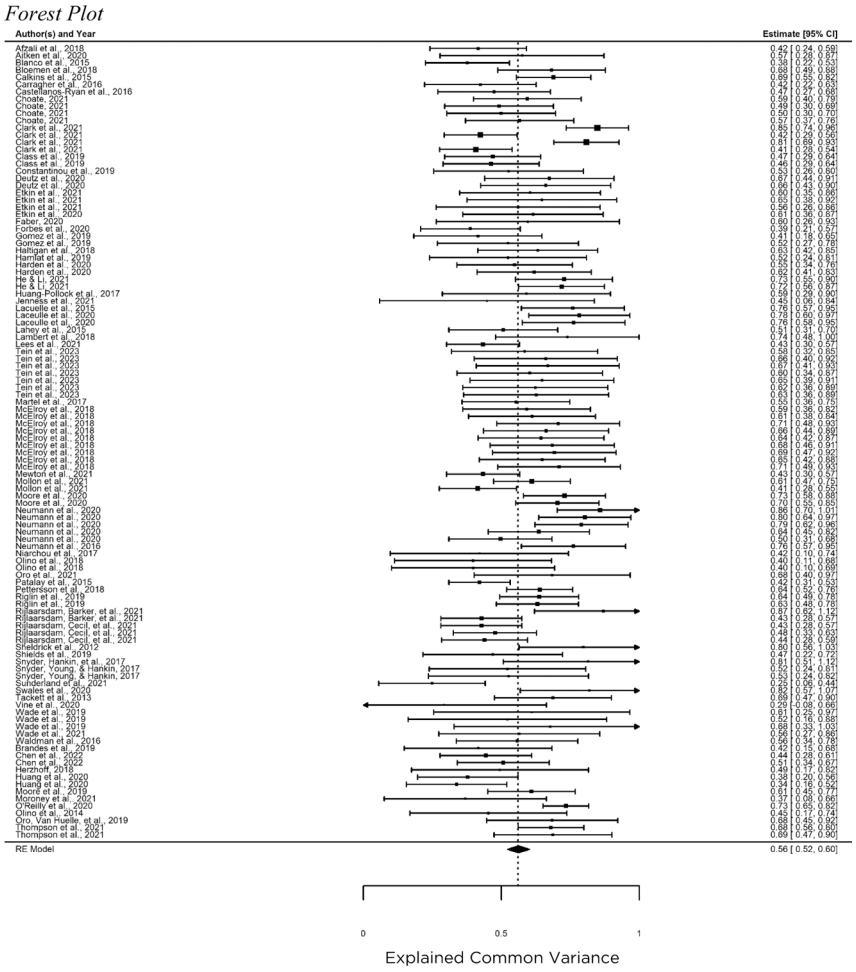

Figure 4.
Forest Plot

Note. Some lower- and upper-bound confidence intervals fell outside of 0–1 range and were cut off due to being improbable values for a proportion (shown with arrows). Every effect size is in the figure, including multiple effect sizes from a given study.

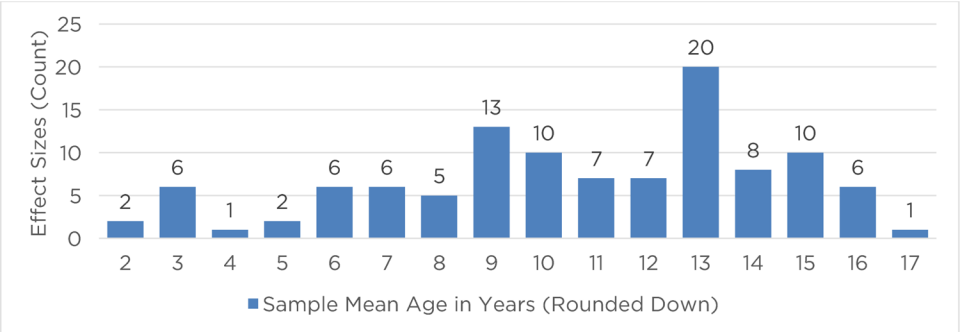

Figure 5.

Distribution of Effect Sizes Across Included Ages

Note. The present sample of papers did not have any studies with a mean age of 1 or 18 years of age.

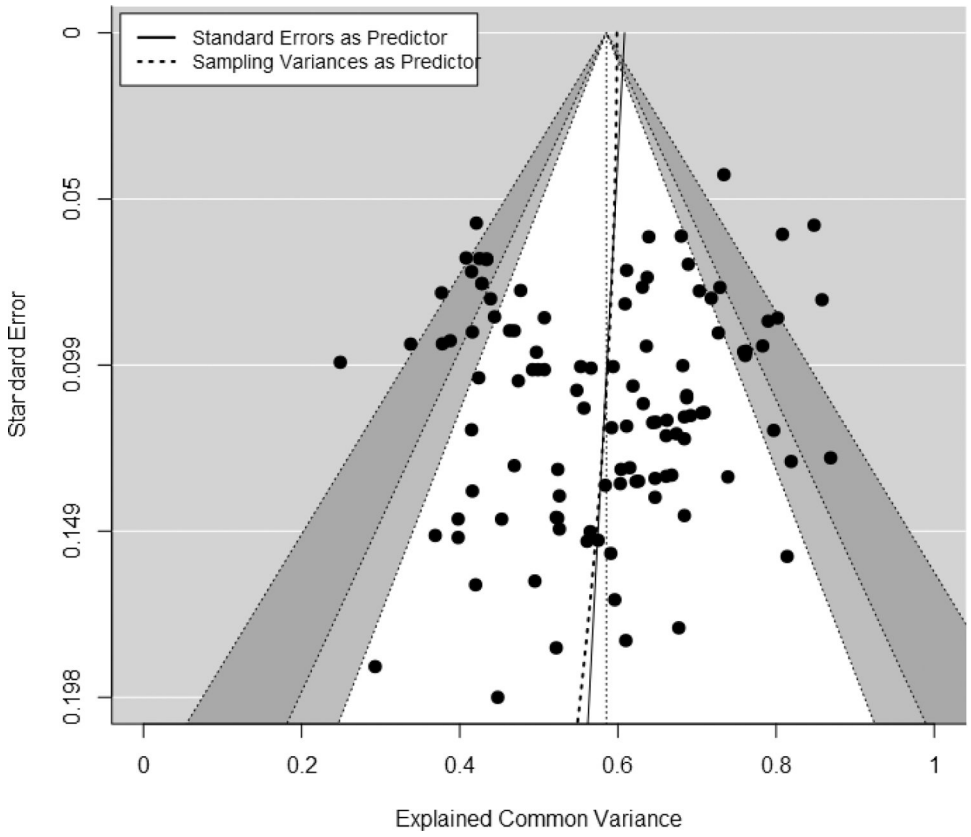

Figure 6.

Contour-Enhanced Funnel Plot with Standard Error and Sampling Variance Predictors

Note. *Plot generated using non-nested data*. Areas with light gray show effects between $p = .05–.1$, and dark gray showing $p = .01–.05$. Solid line represents standard error as a predictor in the association between standard error and explained common variance. Dashed line represents sampling variance as a predictor in the association between standard error and explained common variance.

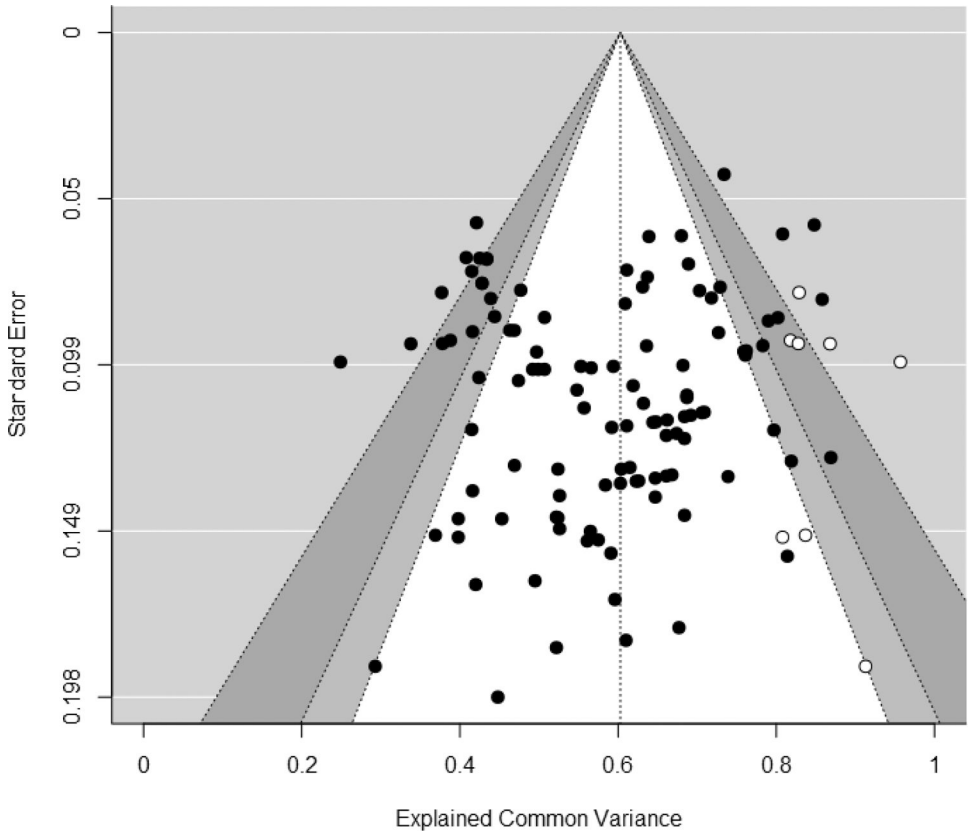

Figure 7.
Trim and Fill Plot

Note. *Plot generated using non-nested data*. Areas with light gray show effects between p = .05–.1, and dark gray showing p = .01–.05. White dots are imputed effect sizes. A total of 8, SE = 6.61 imputed values would need to render the current findings symmetrical.

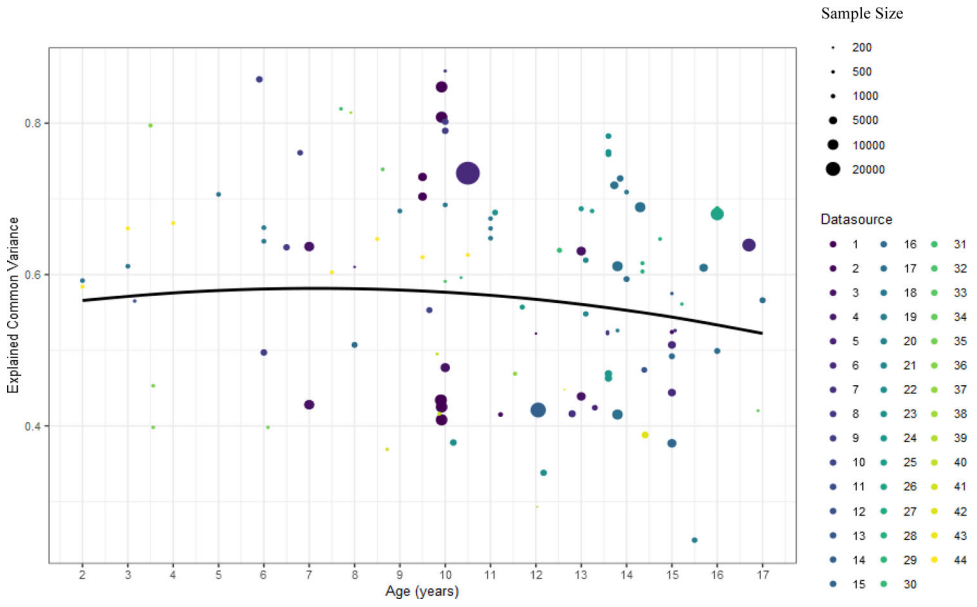

Figure 8.

Bubble Plot of Effect Sizes as a Function of Age Overlaid with Model-Implied Nonlinear ECV Curve

Note. Each bubble represents a single effect size. The bubble size corresponds to the sample size (larger bubbles representing larger samples). 44 uniquely colored data sources are represented numerically. The nonlinear trendline of ECV as a function of age was calculated using the model-implied quadratic trajectory of ECV.

Table 1.

Included Study Characteristics

Study	Data Source	Sample Size (N)	Mean Age (SD)	% Female	Country	Model Type	Informant	Measure	# EXT	# INT	# TD	ECV (Sampling Variance)	Study Quality (Mean| Sum)
Afzali et al., 2018	Co-Venture	3826	12.8 (0.4)	49.2	CA	Mod. Bifactor	Self	SDQ, BSI, APSS	7	20	9	0.42 (0.008)	0.85|11
Aitken et al., 2020	IMPACT	465	15 (NA)	75	GB	Bifactor	Self, Parent(s)	MFQ, RCMAS, LOI, BC	7	30	NA	0.58 (0.023)	1|16
Blanco et al., 2015	NCS-A	6483	15 (NA)	51.1	US	Hierarch	Parent(s), Teacher	CIDI	6	10	NA	0.38 (0.006)	0.73|11
Bloemen et al., 2018	TRAILS	2230	11.1 (0.6)	51	NL	Bifactor	Parent(s)	CBCL, CSBQ	6	2	6	0.68 (0.01)	0.94|15
Brandes et al., 2019	From Tackett, 2011	695	9.87 (NA)	52	US	Bifactor	Parent(s)	CBCL, C-DISC IV	5	3	NA	0.42 (0.019)	0.94|15
Calkins et al., 2015	PNC	9421	14.3 (3.67)	51.7	US	Bifactor	Parent(s)	GOASSESS	4	11	NA	0.69 (0.005)	0.94|15
Carragher et al., 2016	CAP	2175	13.3 (0.48)	42.6	AU, NZ	Mod. Bifactor	Self	SDQ, BSI, RAPI	15	20	9	0.42 (0.011)	0.93|14
Castellanos-Ryan et al., 2016	IMAGEN	2144	14.39 (0.77)	51	GB, IE, FR, DE	Bifactor	Self, Parent(s)	DAWBA, AUDIT	4	6	NA	0.47 (0.011)	0.94|15
Chen et al., 2022	CATSS	4786	15 (NA)	51	SE	Bifactor	Self	SDQ	10	5	NA	0.44 (0.007)	0.87|13
Chen et al., 2022	CATSS	4786	15 (NA)	51	SE	Bifactor	Parent(s)	SDQ	10	4	NA	0.51 (0.007)	0.87|13
Chen et al., 2022	PGS	2450	14 (NA)	99.99	US	Bifactor	Self	SCARED, ASI-4, SCAS	4	2	NA	0.59 (0.01)	1|16
Choate et al., 2022	PGS	2450	15 (NA)	99.99	US	Bifactor	Self	SCARED, ASI-4, SCAS	4	2	NA	0.49 (0.01)	1|16
Choate et al., 2022	PGS	2450	16 (NA)	99.99	US	Bifactor	Self	SCARED, ASI-4, SCAS	4	2	NA	0.5 (0.01)	1|16
Choate et al., 2022	PGS	2450	17 (NA)	99.99	US	Bifactor	Self	SCARED, ASI-4, SCAS	4	2	NA	0.57 (0.01)	1|16
Clark et al., 2021	ABCD	11875	9.92 (0.62)	48.4	US	Bifactor	Parent(s)	CBCL	29	31	NA	0.85 (0.003)	0.93|13
Clark et al., 2021	ABCD	11875	9.92 (0.62)	48.4	US	Hierarch	Parent(s)	CBCL	35	31	NA	0.42 (0.005)	0.93|13
Clark et al., 2021	ABCD	11875	9.92 (0.62)	48.4	US	Bifactor	Parent(s)	CBCL	2	3	NA	0.81 (0.004)	0.93|13

Study	Data Source	Sample Size (N)	Mean Age (SD)	% Female	Country	Model Type	Informant	Measure	# EXT	# INT	# TD	ECV (Sampling Variance)	Study Quality (Mean|Sum)
Clark et al., 2021	ABCD	11875	9.92 (0.62)	48.4	US	Hierarch	Parent(s)	CBCL	2	3	NA	0.41 (0.005)	0.93|13
Class et al., 2019	TTS	3990	13.6 (2.5)	52.1	US	Bifactor	Parent(s)	CADS	6	7	NA	0.47 (0.008)	1|16
Class et al., 2019	TTS	3990	13.6 (2.5)	52.1	US	Bifactor	Self	CADS	6	7	NA	0.46 (0.008)	1|16
Constantinou et al., 2019	START	684	13.8 (1.4)	18	GB	Bifactor	Self	SDQ, MFQ	11	13	NA	0.53 (0.019)	0.86|12
Deutz et al., 2020	SECCYD	1073	11 (NA)	49.8	US	Bifactor	Mother	CBCL	26	13	NA	0.67 (0.014)	0.8|12
Deutz et al., 2020	SECCYD	1073	11 (NA)	49.8	US	Bifactor	Mother	CBCL	26	31	NA	0.66 (0.014)	0.8|12
Etkin et al., 2020	Ind. Sample	835	14.35 (1.58)	49	ES, RO, MA	Bifactor	Self	SENA	3	3	NA	0.62 (0.017)	0.69|9
Etkin et al., 2021	From Etkin et al., 2020	831	14.35 (1.58)	50.6	ES	Bifactor	Self	SENA	3	5	NA	0.6 (0.017)	0.81|13
Etkin et al., 2021	From Etkin et al., 2020	619	14.74 (1.22)	50.8	ES	Bifactor	Self	SENA	3	5	NA	0.65 (0.019)	0.81|13
Etkin et al., 2021	From Etkin et al., 2020	465	15.22 (1)	49.9	ES	Bifactor	Self	SENA	3	5	NA	0.56 (0.023)	0.81|13
Faber & Chaplin, 2020	Ind. Sample	294	10.35 (3.6)	47.85	US	Bifactor	Parent(s)	Y-OQ	11	12	NA	0.6 (0.029)	0.83|10
Forbes et al., 2020	LSAC	3335	14.41 (0.49)	49.1	AU	Hierarch	Self	SDQ, SMFQ	3	2	NA	0.39 (0.008)	0.94|15
Gomez et al., 2019	ABCD	1233	11.22 (3.1)	28.2	AU	Bifactor	Mother, Father	CBCL, ADISC	3	10	NA	0.42 (0.014)	0.93|14
Gomez et al., 2019	ABCD	866	15 (NA)	28.2	AU	Bifactor	Mother, Father	CBCL, ADISC	3	10	NA	0.52 (0.017)	0.93|14
Haltigan et al., 2018	Ind. Sample	1552	12.52 (3.91)	23.2	CA	Bifactor	Parent(s)	CBCL	36	30	12	0.63 (0.012)	1|13
Hamlat et al., 2019	GEM	567	13.58 (2.37)	55.5	US	Bifactor	Self, Parent(s)	CBCL, YSR, MASC, CDI, EATQ-R, SNAP-IV	3	4	NA	0.52 (0.021)	0.92|12
Harden et al., 2020	Texas Twin Project	1913	13.1 (NA)	49	US	Bifactor	Self	CBCL, Conner's 3, BFI	8	3	NA	0.55 (0.011)	1|15
Harden et al., 2020	Texas Twin Project	1913	13.1 (NA)	49	US	Bifactor	Parent(s)	CBCL, Conner's 3, BFI	8	3	NA	0.62 (0.011)	1|15

Study	Data Source	Sample Size (N)	Mean Age (SD)	% Female	Country	Model Type	Informant	Measure	# EXT	# INT	# TD	ECV (Sampling Variance)	Study Quality (Mean| Sum)
He & Li, 2021	PNC	3088	13.86 (0.07)	54.05	US	Bifactor	Self, Parent(s)	GOASSESS	3	9	3	0.73 (0.008)	0.85|11
He & Li, 2021	PNC	5147	13.73 (0.05)	49.64	US	Bifactor	Self, Parent(s)	GOASSESS	3	9	3	0.72 (0.006)	0.85|11
Herzhoff & Tackett, 2018	From Tackett, 2011	349	9.82 (0.66)	53	US	Bifactor	Mother	CBCL, C-DISC IV	5	5	NA	0.5 (0.027)	1|14
Huang-Pollock et al., 2017	Ind. Sample	415	10 (NA)	40.96	US	Bifactor	Mother, Father	BASC-2	4	3	NA	0.59 (0.024)	1|15
Huang et al., 2020	TTC	3171	10.18 (0.28)	46.9	JP	Bifactor	Self, Mother, Father	CBCL, SDQ, APSS, SMFQ	3	8	3	0.38 (0.009)	0.81|13
Huang et al., 2020	TTC	3007	12.17 (0.31)	47.2	JP	Bifactor	Self, Mother, Father	CBCL, SDQ, APSS, SMFQ	3	8	3	0.34 (0.009)	0.81|13
Jenness et al., 2021	Ind. Sample	160	12.63 (2.68)	52	US	Bifactor	Self, Parent(s)	K-SADS	3	3	NA	0.45 (0.039)	1|14
Laceulle et al., 2020	TRAILS	2230	13.6 (0.59)	50.8	NL	Mod. Bifactor	Parent(s)	CBCL	3	2	NA	0.76 (0.009)	1|16
Laceulle et al., 2020	TRAILS	2230	13.6 (0.59)	50.8	NL	Mod. Bifactor	Self	YSR, CADS, CAPE	3	6	NA	0.78 (0.009)	1|16
Laceulle et al., 2015	TRAILS	2230	13.6 (0.59)	NA	NL	Mod. Bifactor	Self, Parent(s)	CBCL, YSR, CADS, CAPE	3	6	NA	0.76 (0.009)	0.75|12
Lahey et al., 2015	PGS	2450	8 (NA)	99.99	US	Bifactor	Parent(s)	CSI-4, SCARED	5	6	NA	0.51 (0.01)	1|16
Lambert et al., 2018	Ind. Sample	626	8.62 (1.74)	51	US	Bifactor	Teacher	EBS	6	3	NA	0.74 (0.018)	0.77|10
Lees et al., 2021	ABCD	11875	9.9 (0.6)	47.9	US	Hierarch	Parent(s)	K-SADS	3	7	4	0.43 (0.005)	0.62|10
Martel et al., 2017	HRC	2512	9.65 (1.93)	46.2	BR	Mod. Bifactor	Parent(s)	DAWBA	3	11	NA	0.55 (0.01)	1|15
McElroy, Belsky, et al., 2018	SECCYD	1253	2 (NA)	51	US	Bifactor	Mother	CBCL	24	36	NA	0.59 (0.014)	0.69|11
McElroy, Belsky, et al., 2018	SECCYD	1253	3 (NA)	51	US	Bifactor	Mother	CBCL	24	36	NA	0.61 (0.014)	0.69|11
McElroy, Belsky, et al., 2018	SECCYD	1253	5 (NA)	51	US	Bifactor	Mother	CBCL	37	30	NA	0.71 (0.013)	0.69|11
McElroy, Belsky, et al., 2018	SECCYD	1253	6 (NA)	51	US	Bifactor	Mother	CBCL	35	31	NA	0.66 (0.013)	0.69|11

Study	Data Source	Sample Size (N)	Mean Age (SD)	% Female	Country	Model Type	Informant	Measure	# EXT	# INT	# TD	ECV (Sampling Variance)	Study Quality (Mean| Sum)
McElroy, Belsky, et al., 2018	SECCYD	1253	6 (NA)	51	US	Bifactor	Mother	CBCL	35	31	NA	0.64 (0.014)	0.69|11
McElroy, Belsky, et al., 2018	SECCYD	1253	9 (NA)	51	US	Bifactor	Mother	CBCL	34	31	NA	0.68 (0.013)	0.69|11
McElroy, Belsky, et al., 2018	SECCYD	1253	10 (NA)	51	US	Bifactor	Mother	CBCL	35	31	NA	0.69 (0.013)	0.69|11
McElroy, Belsky, et al., 2018	SECCYD	1253	11 (NA)	51	US	Bifactor	Mother	CBCL	37	31	NA	0.65 (0.013)	0.69|11
McElroy, Belsky, et al., 2018	SECCYD	1253	14 (NA)	51	US	Bifactor	Mother	CBCL	39	31	NA	0.71 (0.013)	0.69|11
Mewton et al., 2021	ABCD	11721	9.91 (0.62)	47.8	US	Hierarch	Parent(s)	K-SADS	3	7	4	0.43 (0.005)	1|16
Mollon et al., 2021	PNC	9421	13.8 (3.6)	49.7	US	Bifactor	Self, Parent(s)	GOASSESS	25	60	27	0.61 (0.005)	0.92|12
Mollon et al., 2021	PNC	9421	13.8 (3.6)	49.7	US	Hierarch	Self, Parent(s)	GOASSESS	25	60	27	0.42 (0.005)	0.92|12
Moore et al., 2019	PNC	5563	15.7 (2.6)	51.7	US	Bifactor	Self	GOASSESS	24	62	24	0.61 (0.007)	0.86|12
Moore et al., 2020	ABCD	5934	9.5 (NA)	47.9	US	Bifactor	Parent(s)	CBCL	41	26	NA	0.73 (0.006)	0.93|13
Moore et al., 2020	ABCD	5934	9.5 (NA)	47.9	US	Bifactor	Parent(s)	CBCL	47	19	NA	0.7 (0.006)	0.93|13
Moroney & Lee, 2021	From Hinshaw, 2002; Shemmassian & Lee, 2012	460	8.72 (1.67)	65.65	US	Bifactor	Parent(s)	CBCL, C-TRF, C-DISC IV	4	7	NA	0.37 (0.022)	1|14
Neumann et al., 2016	Generation R	2115	6.8 (1.3)	NA	NL, TR, SR, MA	Bifactor	Self, Parent(s), Teacher	CBCL, TRF, SSRS/SRS, BPI, CPRS-R	11	10	NA	0.76 (0.009)	1|15
Neumann et al., 2020	Generation R	3030	5.9 (0.3)	50.4	NL	Bifactor	Mother	CBCL	2	4	NA	0.86 (0.006)	1|15
Neumann et al., 2020	Generation R	3030	10 (0.3)	50.4	NL	Bifactor	Mother	CBCL	3	3	NA	0.8 (0.007)	1|15
Neumann et al., 2020	Generation R	3030	10 (0.3)	50.4	NL	Bifactor	Father	CBCL	3	3	NA	0.79 (0.007)	1|15
Neumann et al., 2020	Generation R	3030	6.5 (1.1)	50.4	NL	Bifactor	Teacher	TRF	3	3	NA	0.64 (0.009)	1|15
Neumann et al., 2020	Generation R	3030	6 (0.4)	50.4	NL	Bifactor	Self	BPI	3	3	NA	0.5 (0.009)	1|15

Study	Data Source	Sample Size (N)	Mean Age (SD)	% Female	Country	Model Type	Informant	Measure	# EXT	# INT	# TD	ECV (Sampling Variance)	Study Quality (Mean| Sum)
Niarchou et al., 2017	From Gur et al., 2014; Tang, Yi, Calkins, et al., 2014; Tang, Yi, Moore, et al., 2014	331	16.9 (8.7)	49	US	Bifactor	Self, Parent(s)	K-SADS, SIPS, SCID	19	26	15	0.42 (0.027)	0.93|13
O'Reilly et al., 2020	CATSS	60888	10.5 (NA)	NA	SE	Bifactor	Mother, Father	A-TAC	31	12	NA	0.73 (0.002)	0.94|15
Olino et al., 2014	From Olino et al., 2010	559	3.56 (0.27)	46.1	US	Bifactor	Parent(s)	PAPA, CBQ, Unknown/ Other	4	5	NA	0.45 (0.021)	0.86|12
Olino et al., 2018	From Olino et al., 2010	541	3.56 (0.27)	46.1	US	Bifactor	Parent(s)	PAPA	4	5	NA	0.4 (0.021)	0.75|12
Olino et al., 2018	From Olino et al., 2010	466	6.09 (0.44)	46.1	US	Bifactor	Parent(s)	PAPA	4	5	NA	0.4 (0.023)	0.75|12
Oro et al., 2021	WTP	502	13.24 (1.52)	NA	US	Bifactor	Self, Parent(s)	CIDI, C-DISC IV	3	7	NA	0.68 (0.021)	0.81|13
Oro et al., 2019	WTP	1004	13.24 (1.52)	52.5	US	Bifactor	Self	C-DISC IV	3	7	NA	0.68 (0.015)	0.93|13
Patalay et al., 2015	Me and My School	23477	12.05 (0.56)	50.4	GB	Bifactor	Self	SDQ, Me & My School	11	14	NA	0.42 (0.003)	0.8|12
Pettersson et al., 2018	CATSS	16806	16.7 (NA)	48.25	SE	Mod. Bifactor	Parent(s)	A-TAC	31	12	NA	0.64 (0.004)	0.81|13
Riglin et al., 2019	ALSPAC	8161	7 (NA)	48	GB	Bifactor	Parent(s)	DAWBA, SCDC	7	5	NA	0.64 (0.005)	0.94|15
Riglin et al., 2019	ALSPAC	7017	13 (NA)	48	GB	Bifactor	Parent(s)	DAWBA, SCDC	7	5	NA	0.63 (0.006)	0.94|15
Rijlaarsdam et al., 2021	Generation R	440	10 (NA)	50.7	NL	Mod. Bifactor	Mother	CBCL	3	3	NA	0.87 (0.016)	1|14
Rijlaarsdam et al., 2021	ALSPAC	7814	7 (NA)	50	GB	Mod. Bifactor	Parent(s)	DAWBA	3	4	NA	0.43 (0.006)	1|14
Rijlaarsdam, Cecil, et al., 2021	ALSPAC	7814	7 (NA)	51	GB	Bifactor	Parent(s)	DAWBA	3	4	NA	0.43 (0.006)	0.94|15
Rijlaarsdam, Cecil, et al., 2021	ALSPAC	7145	10 (NA)	51	GB	Bifactor	Parent(s)	DAWBA	3	5	NA	0.48 (0.006)	0.94|15
Rijlaarsdam, Cecil, et al., 2021	ALSPAC	6210	13 (NA)	51	GB	Bifactor	Parent(s)	DAWBA	3	5	NA	0.44 (0.006)	0.94|15
Sheldrick et al., 2012	Ind. Sample	817	3.5 (NA)	45	US	Bifactor	Parent(s)	PPSC	7	6	NA	0.8 (0.014)	0.94|15

Study	Data Source	Sample Size (N)	Mean Age (SD)	% Female	Country	Model Type	Informant	Measure	# EXT	# INT	# TD	ECV (Sampling Variance)	Study Quality (Mean| Sum)
Shields et al., 2019	Pooled Samples from Caspi et al., 2014; Harden et al., 2020; Huang-Pollock et al., 2017; Martel et al., 2017	895	11.54 (NA)	52	US, CA	Bifactor	Parent(s)	CBCL, C-DISC IV	5	6	NA	0.47 (0.017)	0.93|13
Snyder, Hankin, et al., 2017	Ind. Sample	254	7.92 (1.38)	46	US	Bifactor	Parent(s)	CBCL, CBQ	3	4	NA	0.81 (0.024)	0.91|10
Snyder, Young, et al., 2017	GEM	571	13.58 (2.37)	55.5	US	Bifactor	Self, Parent(s)	CBCL, YSR, MASC, CDI, EATQ-R, SNAP-IV	3	4	NA	0.52 (0.021)	0.81|13
Snyder, Young, et al., 2017	GEM	519	15.07 (2.36)	55.5	US	Bifactor	Self, Parent(s)	CBCL, YSR, MASC, CDI, EATQ-R, SNAP-IV	3	4	NA	0.53 (0.022)	0.81|13
Sunderland et al., 2021	YMM	2003	15.5 (0.03)	48.6	AU	Hierarch	Parent(s)	C-DISC IV	5	5	5	0.25 (0.01)	0.71110
Swales et al., 2020	From Hankin et al., 2017	554	7.7 (1.35)	49.8	US	Bifactor	Mother	CBCL, CBQ	3	4	NA	0.82 (0.016)	1|14
Tackett et al., 2013	TTS	1569	13 (NA)	51	US	Bifactor	Self, Parent(s)	CAPS, CADS	4	5	NA	0.69 (0.012)	0.93|13
Thompson et al., 2021	1958 NCDS	16091	16 (NA)	50.7	GB	Bifactor	Parent(s)	Rutter Scales	11	5	NA	0.68 (0.004)	0.88|14
Thompson et al., 2021	1970 BCS	1528	16 (NA)	52.2	GB	Bifactor	Parent(s)	Rutter Scales	11	5	NA	0.69 (0.012)	0.88|14
Tein et al., 2023	Early Steps	731	2 (NA)	49	US	Bifactor	Parent(s)	CBCL	24	22	NA	0.58 (0.018)	1|15
Tein et al., 2023	Early Steps	731	3 (NA)	49	US	Bifactor	Parent(s)	CBCL	24	22	NA	0.66 (0.018)	1|15
Tein et al., 2023	Early Steps	731	4 (NA)	49	US	Bifactor	Parent(s)	CBCL	24	22	NA	0.67 (0.017)	1|15
Tein et al., 2023	Early Steps	731	7.5 (NA)	49	US	Bifactor	Parent(s)	CBCL	38	24	NA	0.6 (0.018)	1|15
Tein et al., 2023	Early Steps	731	8.5 (NA)	49	US	Bifactor	Parent(s)	CBCL	38	24	NA	0.65 (0.018)	1|15
Tein et al., 2023	Early Steps	731	9.5 (NA)	49	US	Bifactor	Parent(s)	CBCL	38	24	NA	0.62 (0.018)	1|15
Tein et al., 2023	Early Steps	731	10.5 (NA)	49	US	Bifactor	Parent(s)	CBCL	38	24	NA	0.63 (0.018)	1|15
Vine et al., 2020	Ind. Sample	162	12.03 (0.92)	47	US	Bifactor	Self, Parent(s)	K-SADS	5	5	NA	0.29 (0.036)	0.79|11

Study	Data Source	Sample Size (N)	Mean Age (SD)	% Female	Country	Model Type	Informant	Measure	# EXT	# INT	# TD	ECV (Sampling Variance)	Study Quality (Mean|Sum)
Wade et al., 2019	BEIP	220	8 (NA)	50	RO	Bifactor	Parent(s)	MacArthur HBQ	5	3	NA	0.61 (0.033)	0.75|9
Wade et al., 2019	BEIP	220	12 (NA)	50	RO	Bifactor	Parent(s)	MacArthur HBQ	5	3	NA	0.52 (0.034)	0.75|9
Wade et al., 2019	BEIP	220	16 (NA)	50	RO	Bifactor	Parent(s)	MacArthur HBQ	5	3	NA	0.68 (0.032)	0.75|9
Wade et al., 2021	iKFP	501	3.15 (0.27)	49.3	CA	Bifactor	Mother, Father	BITSEA, OCHS	6	4	NA	0.56 (0.022)	0.94|15
Waldman et al., 2016	TTS	1568	11.7 (3.3)	51	US	Bifactor	Parent(s)	CAPS	4	7	NA	0.56 (0.013)	0.86|12

Note. NA: Data not available in-text. *Data Source*. ABCD: Adolescent Brain Cognitive Development; ALSPAC: Avon Longitudinal Study of Parents and Children; BEIP: Bucharest Early Intervention Project; CAP: Climate Schools and Preventure; CATSS: Child and Adolescent Twin Study in Sweden; Co-Venture: Co-Venture Project; Early Steps: Early Steps Multisite Project; GEM: Gene, Environment, Mood Study; Generation R: Generation R Study; HRC: High Risk Cohort Study for Psychiatric Disorders; iKFP: intensive sample of the Kids, Families Places study; IMAGEN: IMAGEN Consortium Study; IMPACT: IMPACT Trial; LSAC: Longitudinal Study of Australian Children; Me and My School: Me and My School Study; NCS-A: National Comorbidity Survey Adolescent Supplement; PGS: Pittsburgh Girls Study; PNC: Philadelphia Neurodevelopmental Cohort; SECCYD: NICHD Study of Early Child Care and Youth Development; YMM: Second Australian Child and Adolescent Survey of Mental Health and Wellbeing (Young Minds Matter); START: Systemic Therapy for At Risk Teens Trial; TTC: Tokyo Teen Cohort; TRAILS: Tracking Adolescents' Individual Lives Survey; TTS: Tennessee Twin Study; WTP: Wisconsin Twin Project; 1958 NCDS: 1958 National Child Development Study; 1970 BCS: 1970 British Cohort Study.

Measures. ADISC: Anxiety Disorders Interview Schedule for Children; APSS: Adolescent Psychotic-Like Symptom Screener; ASI-4: The Adolescent Symptom Inventory-4; A-TAC: Autism-Tics, ADHD and other Comorbidities; AUDIT: Alcohol Use Disorders Identification Test; BASC-2: Behavior Assessment System for Children, 2nd Ed.; BC: Antisocial Behavior Checklist; BFI: Big Five Inventory; BITSEA: Brief Infant-Toddler Social and Emotional Assessment; BPI: Berkeley Puppet Interview; BSI: Brief Symptom Inventory; CADS: Revised Child Anxiety and Depression Scale; CAPE: Community Assessment of Psychic Experiences; CAPS: Child and Adolescent Psychopathology Scale; CBCL: Child Behavior Checklist; CBQ: Children's Behavior Questionnaire; CDI: Children's Depression Inventory; C-DISC IV: Diagnostic Interview Schedule for Children, version IV; CIDI: Composite International Diagnostic Interview; Conner's 3: Conner's 3 rating scales; CPRS-R: Conners' Parent Rating Scale−Revised; CSBQ: Children's Social Behavior Questionnaire; CSI-4: Child Symptom Inventory, 4th ed.; C-TRF: Caregiver-Teacher Report Form; DAWBA: Development and Well-Being Assessment; EATQ-R: Early Adolescent Temperament Questionnaire Revised; EBS: Emotional and Behavioral Screener; GOASSESS: Computerized, structured interview; K-SADS: Kiddie Schedule for Affective Disorders and Schizophrenia; LOI: Short Leyton Obsessional Inventory; MacArthur HBQ: The MacArthur Health and Behavior Questionnaire; MASC: Manifest Anxiety Scale for Children; M&MS: Me and My School questionnaire; MFQ: Mood and Feelings Questionnaire; OCHS: Ontario Child Health Study Scales; PAPA: Preschool Age Psychiatric Assessment; PPSC: Preschool Pediatric Symptom Checklist; RAPI: Rutgers Alcohol Problem Index; RCMAS: Revised Children's Manifest Anxiety Scale; SCAS: The Spence Children's Anxiety Scale; SCARED: Screen for Child Anxiety Related Disorders; SCDC: Social and Communication Disorders Checklist; SCID: Structured Clinical Interview for DSM Disorders; SDQ: Strengths and Difficulties Questionnaire; SENA: Assessment System for Children and Adolescents; SIPS: Structured Interview for Prodromal Syndromes; SMFQ: Short Mood and Feeling Questionnaire; SNAP-IV: Swanson, Nolan, and Pelham scale, 4th ed.; SRS: Social Responsiveness Scale; TRF: Teacher's Rating Form; Y-OQ: Youth Outcome Questionnaire; YSR: Youth Self Report.

Country: Country codes were the two-letter ISO 3166 Alpha-2 codes from the International Organization of Standardization (ISO) found at https://www.iso.org/obp/ui/#home.

Studies derived from multiple samples were comma separated.