Journal of Abnormal Psychology
2017, Vol. 126, No. 1, 137–148

0021-843X/17/$12.00 http://dx.doi.org/10.1037/abn0000205

A General Psychopathology Factor (P Factor) in Children: Structural Model Analysis and External Validation Through Familial Risk and Child Global Executive Function

Michelle M. Martel
University of Kentucky

Pedro M. Pan
Universidade Federal de São Paulo

Maurício S. Hoffmann
Universidade Federal de Santa Maria

Ary Gadelha, Maria C. do Rosário, and Jair J. Mari
Universidade Federal de São Paulo

Gisele G. Manfro
Universidade Federal do Rio Grande do Sul

Eurípedes C. Miguel
Universidade de São Paulo

Tomás Paus
University of Toronto and Child Mind Institute,
New York, New York

Rodrigo A. Bressan
Universidade Federal de São Paulo

Luis A. Rohde and Giovanni A. Salum
Universidade Federal do Rio Grande do Sul

High rates of comorbidities and poor validity of disorder diagnostic criteria for mental disorders hamper advances in mental health research. Recent work has suggested the utility of continuous cross-cutting dimensions, including general psychopathology and specific factors of externalizing and internalizing (e.g., distress and fear) syndromes. The current study evaluated the reliability of competing structural models of psychopathology and examined external validity of the best fitting model on the basis of family risk and child global executive function (EF). A community sample of 8,012 families from Brazil with children ages 6–12 years completed structured interviews about the child and parental psychiatric syndromes, and a subsample of 2,395 children completed tasks assessing EF (i.e., working memory, inhibitory control, and time processing). Confirmatory factor analyses tested a series of structural models of psychopathology in both parents and children. The model with a general psychopathology factor ("P factor") with 3 specific factors (fear, distress, and externalizing) exhibited the best fit. The general P factor accounted for most of the variance in all models, with little residual variance explained by each of the 3 specific factors. In addition, associations between child and parental factors were mainly significant for the P factors and nonsignificant for the specific factors from the respective models. Likewise, the child P factor—but not the specific factors—was significantly associated with global child EF. Overall, our results provide support for a latent overarching P factor characterizing child psychopathology, supported by familial associations and child EF.

General Scientific Summary
An overarching general factor appears to best describe child psychopathology. This factor seems responsible for the familial aggregation of mental disorders, and it is consistently associated with global measures of executive function.

Keywords: P factor, children, structural model, executive function, familial risk

Supplemental materials: http://dx.doi.org/10.1037/abn0000205.supp

This article was published Online First October 17, 2016.

Michelle M. Martel, Psychology Department, University of Kentucky; Pedro M. Pan, Department of Psychiatry, Universidade Federal de São Paulo; Maurício S. Hoffmann, Department of Neuropsychiatry, Universidade Federal de Santa Maria; Ary Gadelha, Maria C. do Rosário, and Jair J. Mari, Department of Psychiatry, Universidade Federal de São Paulo; Gisele G. Manfro, Department of Psychiatry, Universidade Federal do Rio Grande do Sul; Eurípedes C. Miguel, Department of Psychiatry, Universidade de São Paulo; Tomás Paus, Departments of Psychology and Psychiatry, University of Toronto, and Child Mind Institute, New York, New York; Rodrigo A. Bressan, Department of Psychiatry, Universidade Federal de São Paulo; Luis A. Rohde and Giovanni A. Salum, Department of Psychiatry, Universidade Federal do Rio Grande do Sul.

This work is supported by the National Institute of Developmental Psychiatry for Children and Adolescents, a science and technology institute funded by Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq; National Council for Scientific and Technological Development; grant 573974/2008-0) and Fundação de Amparo a Pesquisa do Estado de São Paulo (FAPESP; Research Support Foundation of the State of São Paulo; grant 2008/57896-8). The authors thank the children and families for their participation, which made this research possible.

Correspondence concerning this article should be addressed to Giovanni A. Salum, Department of Psychiatry, Universidade Federal do Rio Grande do Sul, Rua Ramiro Barcelos 2350, CEP -90035-903, Porto Alegre, Brazil. E-mail: gsalumjr@gmail.com

The question of how to best conceptualize the structure of mental disorders has been of central importance to mental health research (Krueger, 1999; Meehl, 1992, 2004). The high rates of co-occurrence of mental disorders, as classified by the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*), has led researchers to investigate superordinate structures derived from loosely grouped polythetic categories in order to provide an empirically based framework for mental health research (Caspi et al., 2014; Kim & Eaton, 2015; Krueger & Markon, 2006; Lahey et al., 2008). Investigation of the reliability and external validity of such empirical classifications is central to advancing both research and clinical practice (Andrews et al., 2009).

Substantial empirical support exists for superordinate classification of psychopathology (e.g., Caspi et al., 2014; Krueger & Markon, 2006; Lahey et al., 2012). At this point, however, available evidence does not converge on a single structural model. Previous work utilizing structural equation modeling (SEM) has suggested that a large number of overarching categories can be effectively encompassed by a much smaller number of latent constructs. Existing evidence supports at least three superordinate models. First, there is the most widely utilized classification of internalizing (e.g., anxiety, depression) versus externalizing (e.g., attention-deficit hyperactivity, conduct) division (Achenbach & Edelbrock, 1978; Kendler, Prescott, Myers, & Neale, 2003; Kessler, Petukhova, & Zaslavsky, 2011; Lahey et al., 2008). Second, there is a finer grained classification splitting the internalizing factor into fear (e.g., phobias) and distress (e.g., generalized anxiety, depression) subdimensions (Krueger, 1999; Krueger et al., 2002; Krueger, Markon, Patrick, Benning, & Kramer, 2007; Simms, Grös, Watson, & O'Hara, 2008; Slade & Watson, 2006). Third, some have added a thought/psychotic dimension (Carragher, Krueger, Eaton, & Slade, 2015; Caspi et al., 2014; Kotov et al., 2011; Wright et al., 2013).

The factor structure of psychopathology may be conceptualized in several different ways. Psychopathology may be best represented as one simple factor (e.g., general psychopathology), which suggests that all of the variance in mental disorders is due to a single latent factor and that variations not captured by this factor are due to measurement error. Alternatively, there might be multiple correlated factors representing psychopathology (e.g., correlated internalizing and externalizing factors), which means that there are two somewhat separate sources of variance correlated with one another. Last, there are bifactor, or hierarchical, models in which a general psychopathology ("P") factor coexists with other, more specific factors, such as externalizing, internalizing, and thought/psychotic (Lahey et al., 2012; see also Caspi et al., 2014). As such, the latter model includes both communalities (unity) and specificities (diversity) within psychopathology.

Previous investigations studying these models have been limited in a number of important ways. First, the nature of the best supported structural model is inconclusive because comprehensive comparative tests among models of adult and child psychopathology structure have yet to be conducted (but see Blanco et al., 2015; Carragher et al., 2015; Caspi et al., 2014; Lahey et al., 2008, 2012, 2015). Second, few studies have investigated reliability indices for lower order dimensions (but see Murray, Eisner, & Ribeaud, 2016). Statistics such as omega hierarchical measure are able to investigate reliability for general and specific factors, being able to determine whether there is sufficient reliability left to specific factors after taking the general factor into account; this is a critical extension of prior work (Reise, 2012). Third, structural models have thus far been tested mostly in prominent Western cultures, particularly in the United States (but see Krueger, Chentsova-Dutton, Markon, Goldberg, & Ormel, 2003). Therefore, it is possible that the structure of psychopathology found in this culture may not replicate in other cultures such as southern, Latin cultures. Finally, although some work has tested empirically the structure of adult psychopathology using latent SEM, few studies have sought to validate structural models of psychopathology by examining associations of latent factors with external measures (but see Caspi et al., 2014; Lahey et al., 2015).

Examination of external validity is essential to clarifying the meaning of latent constructs. Among Feighner et al. (1972) criteria for external validity in psychiatry, two may be particularly informative in children, namely family history and "laboratory" tests. Psychopathology in young children is often associated with parental psychopathology (A. Goodman, Heiervang, Collishaw, & Goodman, 2011; S. H. Goodman & Gotlib, 1999), yet no previous study has investigated familial influences on structural models of psychopathology using similar, higher level structural models for both parents and offspring. This may be a critical issue because a failure to test the best models of parent and child psychopathology may obscure associations between the two.

In addition to family history of psychopathology, measurement of executive function (EF) may be a particularly useful method for external validation of child psychopathology models because it relies on a different source of variance (other than questionnaires and interviews). EF, or the ability to plan and problem-solve to achieve a future goal, is commonly conceptualized as an overarching construct comprised of component operations such as inhibitory control and working memory (Miyake et al., 2000; Miyake & Friedman, 2012; Pennington & Ozonoff, 1996) and temporal processing (Barkley, 1997; Rao, Mayer, & Harrington, 2001); in other words, EF is often conceptualized as a second-order factor model. Further, EF exhibits strong associations with many forms of child psychopathology (reviewed by Morgan & Lilienfeld, 2000; Pennington & Ozonoff, 1996; Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Therefore, external validation of structural models of child psychopathology with family history and child EF may provide important new information about the validity of such models. Such ideas are certainly tenable given limited work conducted to date suggesting associations between child psychopathology factors and intelligence, neural function, neurocognition, and family history (Caspi et al., 2014; Lahey et al., 2015).

In the current study, we aim to address limitations of prior work by investigating the reliability and validity of competing models of psychopathology (unidimensional; internalizing and externalizing; fear, distress, and externalizing; and internalizing, externalizing, and thought) using different structural approaches (unidimensional, correlated, and bifactor structures) in a large community sample of families from a Latin American culture. We extend previous investigations by evaluating the external validity of the best fitting child psychopathology model by investigating factor associations with parental history of psychiatric disorders (evaluated using the same models) and child global (or second-order) EF. We predicted that a three-factor bifactor model of psychopathology would be best supported in children through exami-

nation of external validation indices of parental psychopathology and child EF.

Method

Participants and Study Design

The participants of this study came from the High Risk Cohort Study for Psychiatric Disorders, which investigated typical and atypical trajectories of brain and behavior in children and adolescents (Salum et al., 2015). The study was performed in multiple steps. First, families were screened at schools on the registry day (N = 8,012). Next, high risk and random selection sampling was conducted (n = 2,512). Selected families completed a household parent interview, conducted by a lay interviewer, as well as a child evaluation at schools, conducted by a psychologist and a speech therapist. This report focuses on investigations from the screening phase (n = 8,012 families), parent interview (n = 2,512), and child evaluations (n = 2,395).

Sample demographics on the selected sample (n = 2,512) are as follows: The mean age of parents was 35.6 years (SD = 7.36). The mean age of children was 9.65 years (SD = 1.93); 46.2% were girls. Most (67.5%) of the families were from the middle class socioeconomic strata, with 27% from the upper middle class and 5% from the low and very low class. The median family income (per month) was R$2,900 (US$1,610; 25th percentile: R$2,000 [US$1,110]; 75th percentile: R$4,200 [US$2,333]).

Procedure

In the *screening phase,* a total of 57 schools (22 in Porto Alegre and 35 in São Paulo) were included in the study. The enrollment for the screening phase was conducted at public schools during the early registry days. Attendance in schools is compulsory in Brazil for all children, and, by law, at least one caregiver must be present to register the child. Eligible children were (a) those being registered by a biological parent who was a primary caregiver and could provide sufficient information about the children's behavior and (b) those 6–12 years old at enrollment. All parents present at the selected schools on school registration days were invited to participate. Parents who agreed to participate were interviewed in person or, soon afterward, by telephone with a modified version of the Family History Screen (FHS; Weissman et al., 2000), administered by a lay interviewer. The majority of the interviews were conducted with mothers (87.3%), with the remainder completed by fathers. Among the 9,937 eligible children (from 8,012 families in the screening phase), we recruited two subgroups: one randomly selected (n = 958) and one constituting a high-risk sample (n = 1,554). Briefly, selection for the high-risk sample involved a risk-prioritization procedure that was conducted to identify individuals with current symptoms and/or a family history of specific disorders. Detailed information about the selection procedure can be found in Salum et al. (2015).

After screening, the detailed assessment phase was performed in multiple visits. Data analyzed in this report were collected during the household interview with parents (parent interview) and a child assessment at home or in school (child evaluation). In the household interview parents completed the Development and Well-Being Assessment (DAWBA; A. Goodman et al., 2011). The interview was completed mostly by mothers (94.5%), with the remainder completed by fathers. The child evaluation was completed with a trained clinical psychologist and with a trained speech therapist at the school or home and included several EF tasks, namely: (a) the *digit span* subtest of Spanish version of the Wechsler Intelligence Scale for Children (3rd ed.; WISC–III; Wechsler, 2002); (b) the *Corsi blocks task* (Vandierendonck, Kemps, Fastame, & Szmalec, 2004); (c) the *conflict control task* (CCT; Hogan, Vargha-Khadem, Kirkham, & Baldeweg, 2005); (d) *go/no-go* (GNG; Bitsakou, Psychogiou, Thompson, & Sonuga-Barke, 2008); and (e) the 400-ms and 2,000-ms *time anticipation tasks* (TAs; Toplak & Tannock, 2005). These measures are detailed in the next section.

Measures

Family psychiatric risk. During the screening phase, we conducted the Family History Screen (FHS; Weissman et al., 2000), an interview used to screen all members of a family for mental disorder symptoms according to the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; *DSM–IV;* American Psychiatric Association, 1994) on the basis of the information provided by one family member. The FHS is completely structured, with a mean time of administration of approximately 50 min; it was conducted by trained interviewers using an electronic data-collection system. At the beginning of the interview, the interviewer asked the informant to make a complete list of all biological first-degree family members of each eligible child. Subsequently, the informant was asked about the presence of *DSM–IV* screening symptoms for each diagnosis (e.g., "Did anyone on the list feel sad, blue, or depressed for most of the time for 2 days or more? If yes, who was that?"). The screening question serves as a gateway to ask about impairment, duration or frequency, and/or exclusion questions, asked for only those individuals who screened positively (i.e., these were conditional questions). The instrument was adapted for the purposes of this study. The version used had 48 items: 29 main questions accompanied by 19 conditional questions. It had questions about the main psychiatric syndromes: depression, mania, specific phobia, social phobia, generalized anxiety disorder, panic disorder, agoraphobia, obsessive–compulsive disorder, psychotic experiences, alcohol use and problems due to alcohol use, drug use and problems due to drug use, attention deficit/hyperactivity, separation anxiety, oppositional defiant disorder, and conduct disorder. All estimates were rated as present or absent, coded as 1 or 0, respectively. The instrument has shown acceptable test–retest reliability (median κ = .56) and acceptable accuracy with single informants for most diagnoses (median sensitivity = 35%, median specificity = 93%; Weissman et al., 2000). Maternal and paternal psychopathology indices as rated by the primary informant (mostly mothers) were the focus in the current study. Prevalence rates for each category in mothers and fathers can be seen in the online supplemental materials (see supplemental Table S1).

Child psychopathology. During the parent interview, parents completed the Development and Well-Being Assessment (DAWBA; A. Goodman et al., 2011). The DAWBA is a structured interview in which all questions are closely related to *DSM–IV* diagnostic criteria and focus on current problems causing significant distress or social impairment. Child probabilistic diagnoses

were established using diagnostic probabilistic bands (A. Goodman et al., 2011), which represent computer-generated categories based on answers to the DAWBA questions that inform the rater about the probability of a positive diagnosis (<.1%, ~.5%, ~3%, ~15%, ~50%, and higher than 70%; coded as 0 [<.1%] to 5 [higher than 70%]). All DAWBA bands were considered in the present study, including panic, agoraphobia, social anxiety, separation anxiety, eating, depression, posttraumatic stress, obsessive–compulsive, conduct, oppositional defiant and attention-deficit/hyperactivity, and autism spectrum disorders. DAWBA bands showed very good concordance with clinician-rated diagnosis, with kappas ranging between .4 and .7, sensitivities .4 and .8, and specificities .98 and .99 (A. Goodman et al., 2011). In the current study, DAWBA bands were used as the measure of child psychopathology. Unlike clinical ratings (yes/no), DAWBA bands offer a degree of dimensionality to the diagnostic assessment, which is desirable especially for disorders with low prevalence. Child prevalence rates for each DAWBA band can be seen in the online supplemental materials (see supplemental Table S2).

Assessment of executive function (EF). Testing of EF testing took place at school over four sessions with trained clinicians. All dependent measures from all EF tasks were adjusted for age and transformed into *t* scores before data analysis. Higher scores represent better EF.

Working memory.

Digit span. This is a subtest of the WISC–III (Wechsler, 2002) in which children hear sequences of numbers (increasing in difficulty) and are asked to repeat them, either as heard (forward) or in reverse order (backward). The level at which the child failed to repeat correctly the numbers on two consecutive trials at one level of difficulty was the outcome measure.

Corsi blocks task (Vandierendonck et al., 2004). This task involves repeating a spatial sequence tapped by a researcher on up to nine identical spatially separated blocks, with sequences that increase in length. The level at which the child failed to correctly repeat the sequence of blocks of a given length on two consecutive trials was the outcome measure.

Inhibitory control.

Conflict control task (CCT; Hogan et al., 2005). In this task, participants are instructed to press the button indicating the direction or the opposite direction of arrows appearing on the screen. Seventy-five trials were presented with green arrows in which participants were instructed to press the button indicating the direction of the arrow (congruent trials). The remaining 25 trials were presented with red arrows in which participants had to respond in the opposite direction to that indicated by the arrows (incongruent trials). Intertrial interval was 1,500 ms, and the stimulus duration was 100 ms. This task includes an inhibitory executive component requiring participants to occasionally suppress a dominant tendency to respond to the actual direction of an arrow and to initiate a response indicating the opposite direction, a "conflict" effect. Accuracy and speed were equally emphasized in task instructions. The percentage of correct responses in the incongruent trials was the outcome measure.

Go/no-go (GNG; Bitsakou et al., 2008). This task includes a different inhibitory component that requires participants to suppress completely and withhold a dominant tendency to press the buttons indicating the direction of the green arrows (*go* stimuli; $n = 75$) when a double-headed green arrow (*no-go* stimuli; $n = 25$) appears on the screen. This task consisted of 100 trials. Intertrial interval was 1,500 ms, and the stimulus duration was 100 ms. Accuracy and speed were equally emphasized in task instructions. The percentage of failed inhibitions in the *no-go* trials (i.e., commission errors) was the dependent measure.

Time processing.

Time anticipation (TA) tasks: 400 ms and 2,000 ms (Toplak & Tannock, 2005). These are gamelike tasks in which participants had to anticipate when a visual stimulus would reappear. The task entails an allied spaceship running out of oxygen, and the participant has to beam oxygen to save the allied crew. In each task, the allied spaceship was visible for the first 10 trials; for the remaining 16 trials, participants were asked to press a button to anticipate when it would arrive because an invisible shield was activated. The participant was given feedback after every trial. Participants had a 750-ms window of time to respond correctly. The child beamed oxygen over to a spaceship to save the crew. In Task 1, the anticipation interval was 400 ms, and in Task 2 it was 2,000 ms. The 2,000-ms task was always administered after the 400-ms task. The mean percentage of total hits (i.e., button pressed in the correct time window interval) was the outcome measure for the tasks.

Data Analysis

All analyses were performed in Mplus software package, Version 7.4 (Muthén & Muthén, 1998–2014). We used complex modeling that allowed for inclusion of sampling weights (see Table S3 and Figure S1 in the online supplemental materials) and for taking the clustered (by school) data into account. Missing data were handled using full-information maximum likelihood, which considers all available data points. There were no missing data for the clinical data. EF assessment was performed over four sessions; therefore, the number of participants who completed each task differs between tasks (ranging from 2,158 to 2,243 valid assessments; with 2,395 completing at least one task). There were no significant differences on demographics or levels of psychopathology among those who completed and did not complete EF assessment (all $ps > .05$). A description of the measures used in this study with the valid sample size for each task can be found in the online supplemental materials (see Table S3).

Model fit. In order to test and compare competing models of psychopathology structure for children and their parents, we conducted confirmatory factor analyses. Specifically, unidimensional, correlated, and bifactor models of psychopathology per the unidimensional; internalizing and externalizing; fear, distress, and externalizing; and internalizing, externalizing, and thought frameworks were tested. For bifactor models, we tested both orthogonal (i.e., correlations among factors are set to zero) and nonorthogonal (i.e., fear and distress factors are allowed to correlate; internalizing and thought factors are allowed to correlate) models. This procedure was utilized across child (DAWBA) and parent (FHS) model tests. For the child data, we did not test the internalizing, externalizing, and thought model, given that there are no available DAWBA band measures for psychosis and mania (rare in children).

Model goodness of fit was evaluated using chi-square fit statistics, root-mean-square error of approximation (RMSEA), comparative fit index (CFI), and Tucker–Lewis index (TLI), as recommended (Hu & Bentler, 1999; Kline, 2005). Smaller chi-square

and RMSEA values and larger CFI values indicate better fit. A nonsignificant chi-square, RMSEA equal to or below .06, and CFI and TLI above .95 indicate a good fit, according to the literature (Hu & Bentler, 1999; Kline, 2005). Comparison of model fit was conducted using chi-square for difference testing.

Reliability. In order to assess the reliability of the factors, we considered the following indices: (a) the percentage of explained common variance (an index of unidimensionality) that was attributable to the general factor and to the specific factors, defined as the ratio of variance explained by the general factor divided by the variance explained by the general plus the specific factors (Bentler, 2009; Reise, 2012), which is interpreted in conjunction with the percentage of uncontaminated correlations; (b) Lucke's omega (ω; Lucke, 2005), a model-based reliability estimate, being analogous to alpha coefficient but appropriate for congeneric tests (varying factor loadings); (c) the hierarchical omega coefficient (ω*H*; McDonald, 1999; Zinbarg, Revelle, Yovel, & Li, 2005), which judges the degree to which composite scale scores are interpretable as a measure of a single common factor; and (d) the omega subscale (ω*S*) reliability estimate for a residualized subscale, an index that controls for that part of the reliability due to the general factor (i.e., indicating the reliability of subscale score remaining once the effects of the general factor are removed; Reise, 2012). Values of ω, ω*H*, and ω*S* coefficients may vary between 0 and 1, where higher scores indicate greater reliability; a value of 1 indicates that the instrument's sum score measures the target construct with perfect accuracy. We compared fit and reliability indices among models and selected the best fitting and most reliable model for external validation analysis.

Data reduction for EF model. In order to reduce the EF data, we fitted to the data a second-order model with one higher order factor (EF) and three lower order factors (i.e., working memory, inhibitory control, and temporal processing), which were fully encompassed by the EF factor (see Table S4 in the online supplemental materials). As expected, the lower order factors exhibited high loadings on the higher order factor (λ ranging from .4 to .8). This model exhibited excellent fit to the data (RMSEA = .004, 90% confidence interval [CI: <001, .027], CFI > .999, TLI = .999; see Table S5 in the online supplemental materials).

External validation. In order to validate the best fitting and most reliable child psychopathology model, we conducted an integrative SEM. This model contains all correlations among latent variables from best fitting models from children (DAWBA) and parents (FHS) to allow for examination of associations between the child and parental psychiatric risk. In order to control for reporting effects, we included the respondent as a covariate in the SEM model. Next, the best fitting and most reliable child model was also externally validated via examination of associations between child latent factors and (a) parental latent factors and (b) child global EF via regression coefficients in SEM.

Results

Child Psychopathology Models

Unidimensional, correlated factor, and bifactor models from confirmatory factor analysis (with and without orthogonal specific factors) were tested using unidimensional; internalizing and externalizing; and fear, distress, and externalizing frameworks. The bifactor model with a general P psychopathology factor and specific fear, distress, and externalizing factors exhibited the best fit to the data compared with other models (see Table 1). Whereas the P factor is a general factor accounting for shared variance across all items, the specific factors represent the common variance among specific items after controlling for the variance shared by all items. This best fitting model allowed fear and distress specific factors to be correlated (ϕ = .861). The P factor exhibited high reliability (ω = .914, ω*H* = .733), indicating that 73% of the variance in the unit-weighted total score can be attributed to individual differences in the P factor. Comparing reliability indices (ω and ω*H*), 80% (.733 divided by .914) of the reliable variance can be attributed to the P factor, assumed to reflect individual differences in overall psychopathology. Only 18% (.914 minus .733) of the reliable variance can be attributed to the specific factors (fear, distress, and externalizing). This is reflected in the reliabilities of the specific factors, before (ω_{fear} = .904; $\omega_{distress}$ = .903; $\omega_{externalizing}$ = .9) and after (ωS_{fear} = .089; $\omega S_{distress}$ = .072; $\omega S_{externalizing}$ = .044) controlling for the P factor. Thus, the apparent reliability of the specific factors is attributable to individual differences in the P factor.

Parental Psychopathology Models

The same models were also fitted to mother (see Table 2) and father (see Table 3) FHS data. The bifactor model with a general P psychopathology factor and specific fear, distress, and externalizing factors exhibited the best fit to the data for mothers and fathers. In both maternal and paternal models, fear and distress factors were allowed to correlate (ϕ = .73 for mothers; ϕ = .73 for fathers).

The maternal P factor exhibited high reliability (ω = .898, ω*H* = .730), indicating that 73% of the variance in the unit-weighted total scores can be attributed to the individual differences in the P factor from the mother. Comparing reliability indices (ω and ω*H*), one can see that 81% (.730 divided by .898) of the reliable variance can be attributed to the P factor. Only 17% (.898 minus .730) of the reliable variance can be attributed to all specific factors, reflected in very low reliabilities after accounting for the P factor (ωS_{fear} = .044; $\omega S_{distress}$ = .057; $\omega S_{externalizing}$ = .088).

The paternal P factor also exhibited high reliability (ω = .882, ω*H* = .696), indicating that about 70% of the variance in the unit-weighted total scores can be attributed to individual differences in the P factor. Comparing reliability indices (ω and ω*H*), 79% (.696 divided by .882) of the reliable variance can be attributed to the P factor. Only 18% (.898 minus .730) of the reliable variance can be attributed to all specific factors, reflected in very low reliabilities after accounting for the P factor (ωS_{fear} = .087; $\omega S_{distress}$ = .027; $\omega S_{externalizing}$ = .094).

Thus, the bifactor model with a general P psychopathology factor and specific fear, distress, and externalizing factors exhibited best fit in both children and parents; correlations between fear and distress models substantially improved model fit. In all models, the general P factor accounted for most of the variance in both child and adult models; after taking the general factor into account, reliability for specific models was very low.

Table 1
Confirmatory Factor Analysis Models for DAWBA Data From the Child (n = 2,512) in the Weighted Sample (Given as λ)

		Correlated models					Bifactor models										
		Two factors		Krueger & Watson[a]			Two-group factors			Bifactor Krueger & Watson[a]: Orthogonal				Bifactor Krueger & Watson[a]: Nonorthogonal (Fea with Dis)			
Variable	Uni	Int	Ext	Fea	Dis	Ext	P	Int	Ext	P	Fea	Dis	Ext	P	Fea	Dis	Ext
Items											No convergence						
Panic	.731	.754		.778			.568	.498						.575	.506		
Agoraphobia	.744	.786		.808			.515	.643						.510	.672		
Social anxiety	.558	.603		.624			.385	.503						.382	.529		
Specific anxiety	.546	.610		.635			.437	.444						.449	.442		
Separation anxiety	.579	.640		.661			.469	.445						.482	.436		
Depression	.700	.779			.781		.695	.325						.694		.339	
Generalized anxiety	.626	.685			.684		.442	.582						.438		.619	
Obsessive–compulsive	.460	.511			.510		.390	.332						.391		.344	
Tic	.542	.571			.574		.552	.185						.560		.178	
Posttraumatic stress	.518	.568			.568		.431	.376						.432		.384	
Eating disorder	.460	.498			.497		.320	.421						.317		.439	
Conduct disorder	.780		.871			.870	.596		.626					.593			.629
Oppositional defiant	.830		.879			.881	.585		.763					.583			.764
ADHD	.689		.762			.762	.649		.379					.646			.383
Autism spectrum	.644		.741			.738	.781		[b]					.779			[b]
Model fit											No convergence						
FP	69	70			72			83							84		
Model χ^2	679.8	279.9			270.5			178.8							174.729		
χ^2 test for difference testing		$\chi^2(1) = 199.1$, $p < .001$[c]			$\chi^2(2) = 18.9$, $p < .001$[d]			$\chi^2(13) = 105.2$, $p < .001$[d]							$\chi^2(1) = 9.1$, $p = .003$[e]		
RMSEA	.051	.029			.029			.023							.023		
RMSEA 90% CI	[.048, .055]	[.025, .033]			[.025, .033]			[.019, .028]							[.019, .027]		
CFI	.882	.962			.963			.979							.980		
TLI	.862	.955			.956			.972							.972		
WRMR	2.485	1.484			1.449			.994							.977		
Reliability																	
ECV (%)							56.3							55.3			
PUC (%)							44.8							73.3			
ω (Lucke)	.909	.884	.888	.830	.776	.887	.922	.919	.897					.914	.904	.903	.900
ω*H*							.649							.733			
ω*S*								.248	.044						.089	.072	.044

Note. Factor correlations (Φ): correlated two-factor model (Int with Ext $r = .598$); correlated Krueger & Watson model (Fea with Ext $r = .533$, Fea with Dis $r = .939$; Ext with Dis $r = .626$); bifactor Krueger & Watson nonorthogonal model (Fea with Dis $r = .861$). DAWBA = Development and Well-Being Assessment; λ = factor loadings; Uni = unidimensional; Int = internalizing; Ext = externalizing; Fea = fear; Dis = distress; P = general psychopathology; ADHD = attention-deficit/hyperactivity disorder; FP = free parameters; RMSEA = root-mean-square error of approximation; CI = confidence interval; CFI = comparative fit index; TLI = Tucker–Lewis index; WRMR = weighted root-mean-square residual; ECV = explained common variance; PUC = percentage of uncontaminated correlations; ω = omega coefficient; ω*H* = omega hierarchical; ω*S* = omega subscale.
[a] Krueger & Markon, 2006; Watson, 2005. [b] Did not significantly load in the specific externalizing factor. [c] Reference (uni). [d] Reference (two factors). [e] Reference (bifactor two-specific).

Table 2
Confirmatory Factor Analysis Models for Family History Screen Data (Given as λ) From the Biological Mother (n = 8,012)

		Correlated models								Bifactor models																
		Two factors		Krueger & Watson[a]			Caspi			Two-group factors			Bifactor Krueger & Watson[a]: Orthogonal				Bifactor Krueger & Watson[a]: Nonorthogonal (Fea with Dis)				Bifactor Caspi: Orthogonal				Bifactor Caspi: Nonorthogonal (Int with Tho)			
Variable	Uni	Int	Ext	Fea	Dis	Ext	Int	Tho	Ext	P	Int	Ext	P	Fea	Dis	Ext	P	Fea	Dis	Ext	P	Int	Tho	Ext	P	Int	Tho	Ext
Items										No convergence																		
Panic	.959	.714		.734			.720			.449	.676						.471	.680			.616	.602			.442	.676		
Agoraphobia	.956	.707		.730			.713			.455	.638						.486	.626			.628	.433			.446	.645		
Social anxiety	.748	.616		.629			.620			.562	.255						.580	.239			.619	.054			.558	.266		
Specific anxiety	.552	.497		.513			.502			.450	.205						.463	.201			.493	.048			.447	.215		
Separation anxiety	.590	.521		.531			.524			.593	.003						.599	−.006			.563	−.114			.595	.010		
Depression	.698	.591			.604		.595			.460	.369						.413		.499		.546	.219			.456	.374		
Generalized anxiety	.910	.690			.704		.697			.575	.377						.528		.509		.664	.174			.570	.388		
Obsessive–compulsive	.857	.666			.681			.692		.617	.262						.600		.313		.684		−.247		.599		.337	
Mania	.660	.568			.576			.580		.460	.340						.450		.372		.621		.654		.463		.362	
Psychosis	.883	.668			.685			.694		.624	.263						.620		.295		.688		−.098		.604		.338	
Conduct disorder	.582		.664			.664			.662	.421		.584					.422			.586	.363			.619	.426			.584
Oppositional defiant	.716		.751			.752			.753	.518		.568					.519			.567	.451			.623	.525			.563
ADHD	.754		.746			.746			.746	.545		.463					.549			.460	.477			.528	.551			.456
Alcohol	.587		.638			.638			.637	.468		.369					.472			.363	.421			.415	.476			.357
Drugs	.572		.654			.653			.652	.410		.549					.416			.541	.356			.591	.421			.533
Model fit										No convergence																		
FP	30	31		33			33			45							46				45				46			
Model χ^2	794.9	384.402		369.9			376.7			284.0							262.6				293.0				285.3			
χ^2 test for difference testing		$\chi^2(1) = 287.8$, $p < .001$[b]		$\chi^2(2) = 25.9$, $p < .001$[c]			$\chi^2(2) = 15.7$, $p < .001$[c]			$\chi^2(14) = 137.5$, $p < .001$[c]							$\chi^2(1) = 36.6$, $p < .001$[d]				$\chi^2(1) = 32.4$, $p < .001$[e]				$\chi^2(1) = 1.9$, $p = .163$[d]			
RMSEA	.031	.020		.020			.020			.019							.018				.019				.019			
RMSEA 90% CI	[.029, .033]	[.018, .022]		[.018, .022]			[.018, .023]			[.016, .021]							[.016, .020]				[.017, .021]				[.017, .021]			
CFI	.917	.965		.967			.966			.975							.978				.974				.975			
TLI	.903	.959		.960			.959			.966							.968				.964				.965			
WRMR	3.112	2.088		2.034			2.059			1.656							1.581				1.736				1.650			
Reliability																												
ECV (%)										58.2							55.9				47.2				42.6			
PUC (%)										47.6							71.4				67.6				67.6			
ω (Lucke)	.887	.865	.821	.767	.786	.821	.819	.694	.821	.902	.894	.886					.898	.883	.885	.889	.910	.901	.898	.907	.897	.887	.878	.886
ω*H*										.688							.730				.794				.723			
ω*S*											.149	.088						.044	.057	.088		.026	.001	.094		.091	.016	.087

Note. Factor correlations (Φ): Correlated two-factor model (Int with Ext $r = .586$); correlated Krueger & Watson model (Fea with Ext $r = .559$; Fea with Dis $r = .913$; Ext with Dis $r = .585$); correlated Caspi model (Int with Ext $r = .565$; Int with Tho $r = .932$; Ext with Tho $r = .606$); bifactor Krueger & Watson nonorthogonal model (Fea with Dis $r = .729$); bifactor Caspi nonorthogonal model (Int with Tho $r = .832$). λ = factor loadings; Uni = unidimensional; Int = internalizing; Ext = externalizing; Fea = fear; Dis = distress; Tho = thought/psychotic; P = general psychopathology; ADHD = attention-deficit/hyperactivity disorder; FP = free parameters; RMSEA = root-mean-square error of approximation; CI = confidence interval; CFI = comparative fit index; TLI = Tucker–Lewis index; WRMR = weighted root-mean-square residual; ECV = explained common variance; PUC = percentage of uncontaminated correlations; ω = omega coefficient; ω*H* = omega hierarchical; ω*S* = omega subscale.

[a] Krueger & Markon, 2006; Watson, 2005. [b] Reference (uni). [c] Reference (two factors). [d] Reference (bifactor two-specific). [e] Reference (bifactor Caspi non-orthogonal).

Table 3
Confirmatory Factor Analysis Models for Family History Screen Data (Given as λ) From the Biological Father (n = 8,012)

		Correlated models								Bifactor models																			
		Two factors[a]		Krueger & Watson[b]			Caspi[a]			Two-group factors[a]			Bifactor Krueger & Watson[b]: Orthogonal[a]				Bifactor Krueger & Watson[b]: Nonorthogonal (Fea with Dis)				Bifactor Caspi: Orthogonal				Bifactor Caspi: Nonorthogonal (Int with Tho)			
Variable	Uni[a]	Int	Ext	Fea	Dis	Ext	Int	Tho	Ext	P	Int	Ext	P	Fea	Dis	Ext	P	Fea	Dis	Ext	P	Int	Tho	Ext	P	Int	Tho	Ext
Items							No convergence														No convergence				No convergence			
Panic	.659	.711		.745						.630	.315		.688	.144			.606	.371										
Agoraphobia	.709	.769		.817						.632	.465		.710	.302			.590	.539										
Social anxiety	.563	.620		.663						.488	.453		.535	.579			.454	.505										
Specific anxiety	.408	.447		.474						.301	.446		.384	.339			.263	.491										
Separation anxiety	.487	.514		.538						.424	.307		.455	.286			.415	.320										
Depression	.596	.637			.650					.616	.176		.644		.092		.596		.261									
Generalized anxiety	.594	.652			.657					.556	.345		.665		.300		.520		.451									
Obsessive–compulsive	.496	.553			.558					.435	.395		.553		.224		.387		.526									
Mania–psychosis[c]	.661	.712			.729					.905	−.182		.785		−.322		.851		−.031									
Conduct disorder	.583		.704			.702				.350		.683	.335			.690				.671								
Oppositional defiant	.673		.779			.781				.421		.716	.396			.731	.375			.697								
ADHD	.674		.738			.734				.480		.583	.461			.597	.449			.562								
Alcohol	.425		.535			.538				.342		.109	.326			.126	.507			.082								
Drugs	.455		.557			.560				.277		.336	.246			.356	.365			.321								
Model fit							No convergence														No convergence				No convergence			
FP	28	29		31						43			43				44											
Model χ^2	809.4	443.9		430.4						218.1			260.0				211.4											
χ^2 test for difference testing		$\chi^2(1) = 191.6$, $p < .001$[d]		$\chi^2(2) = 22.5$, $p < .001$[e]						$\chi^2(14) = 257.9$, $p < .001$[e]			$\chi^2(1) = 62.6$, $p < .001$[f]				$\chi^2(1) = 13.7$, $p < .001$[g]										
RMSEA	.034	.025		.025						.018			.020				.018											
RMSEA 90% CI	[.032, .037]	[.022, .027]		[.022, .027]						[.015, .020]			[.017, .023]				[.015, .020]											
CFI	.805	.902		.905						.958			.947				.960											
TLI	.770	.883		.883						.939			.923				.940											
WRMR	3.299	2.361		2.291						1.479			1.643				1.442											
Reliability																												
ECV (%)										58.9			62.7				54.3											
PUC (%)										49.5			71.4				71.4											
ω (Lucke)	.869	.853	.770	.788	.745	.771				.887	.876	.873	.889	.878	.873	.885	.882	.867	.859	.868								
ω*H*										.692			.757				.696											
ω*S*											.119	.097		.044	.001	.096		.087	.027	.094								

Note. Factor correlations (Φ): correlated two-factor model (Int with Ext $r = .528$); correlated Krueger & Watson model (Fea with Ext $r = .438$; Fea with Dis $r = .870$; Ext with Dis $r = .567$); bifactor Krueger & Watson nonorthogonal model (Fea with Dis $r = .725$). λ = factor loadings; Uni = unidimensional; Int = internalizing; Ext = externalizing; Fea = fear; Dis = distress; Tho = thought/psychotic; P = general psychopathology; ADHD = attention-deficit/hyperactivity disorder; FP = free parameters; RMSEA = root-mean-square error of approximation; CI = confidence interval; CFI = comparative fit index; TLI = Tucker–Lewis index; WRMR = weighted root-mean-square residual; ECV = explained common variance; PUC = percentage of uncontaminated correlations; ω = omega coefficient; ω*H* = omega hierarchical; ω*S* = omega subscale.
[a] Errors of alcohol and drugs items were correlated in this model. [b] Krueger & Markon, 2006; Watson, 2005. [c] Mania and psychosis were summed in order to produce model convergence. [d] Reference (unidimensional). [e] Reference (two factors). [f] Reference (bifactor Krueger & Watson nonorthogonal). [g] Reference (bifactor two-specific).

External Validation of Child Psychopathology Model

As shown in Figure 1, standardized regression coefficients among factors from the best fitting child and parental models with child factors regressed on parental factors were significant mostly for the child and parental P factors (vs. specific factors), in 60% of instances. There were no significant associations for the mother and father specific fear and distress factors. The maternal P factor was significantly associated with the child P factor ($\beta = .33$, 95% CI [.21, .45], $p < .01$), as well as the child distress ($\beta = .31$, 95% CI [.19, .43], $p < .01$) and child fear ($\beta = .23$, 95% CI [.096, .36], $p < .01$) factors. The paternal P factor was significantly associated with the child P factor ($\beta = .18$, 95% CI [.09, .28], $p < .01$), as well as the child distress ($\beta = .12$, 95% CI [.01, .23], $p < .01$) and child fear ($\beta = .23$, 95% CI [.14, .32], $p < .01$) factors. The maternal externalizing factor was significantly associated with the child P factor ($\beta = .17$, 95% CI [.05, .28], $p < .01$), as well as with the child specific externalizing factor ($\beta = .17$, 95% CI [.06, .28], $p < .01$). The paternal externalizing factor was also significantly associated with child specific externalizing factor ($\beta = .19$, 95% CI [.07, .30], $p < .01$) and inversely significantly associated with the child fear factor ($\beta = -.15$, 95% CI [−.28, −.014], $p < .01$; see Table S6 in the online supplemental materials).

We observed, in regard to associations with child global EF, significant associations between the child P factor and the global EF factor ($\beta = -.24$, 95% CI [−.35, −.12], $p < .01$), but no significant associations emerged for the fear ($\beta = -.019$, 95% CI [−.51, .48], $p > .94$), distress ($\beta = .23$, 95% CI [−.29, .76], $p > .39$), or externalizing ($\beta = -.10$, 95% CI [−.22, .02], $p > .09$) factors.

Discussion

The goal of the current study was to test the reliability of competing latent structures of child and adult psychopathology and externally validate the best fitting models using familial risk of psychopathology and child global EF in a large community sample of families from Brazil. For children, a bifactor model with a general psychopathology factor (P factor) and the three specific factors of distress, fear, and externalizing provided the best fit to the data. Yet, only the P factor was characterized by substantial reliable variance on the basis of reliability indices. Parental psychopathology model results were similar. Associations between children and parental factors were mostly significant for the P factors from the respective models. A similar pattern of association was found between child psychopathology factors and a global

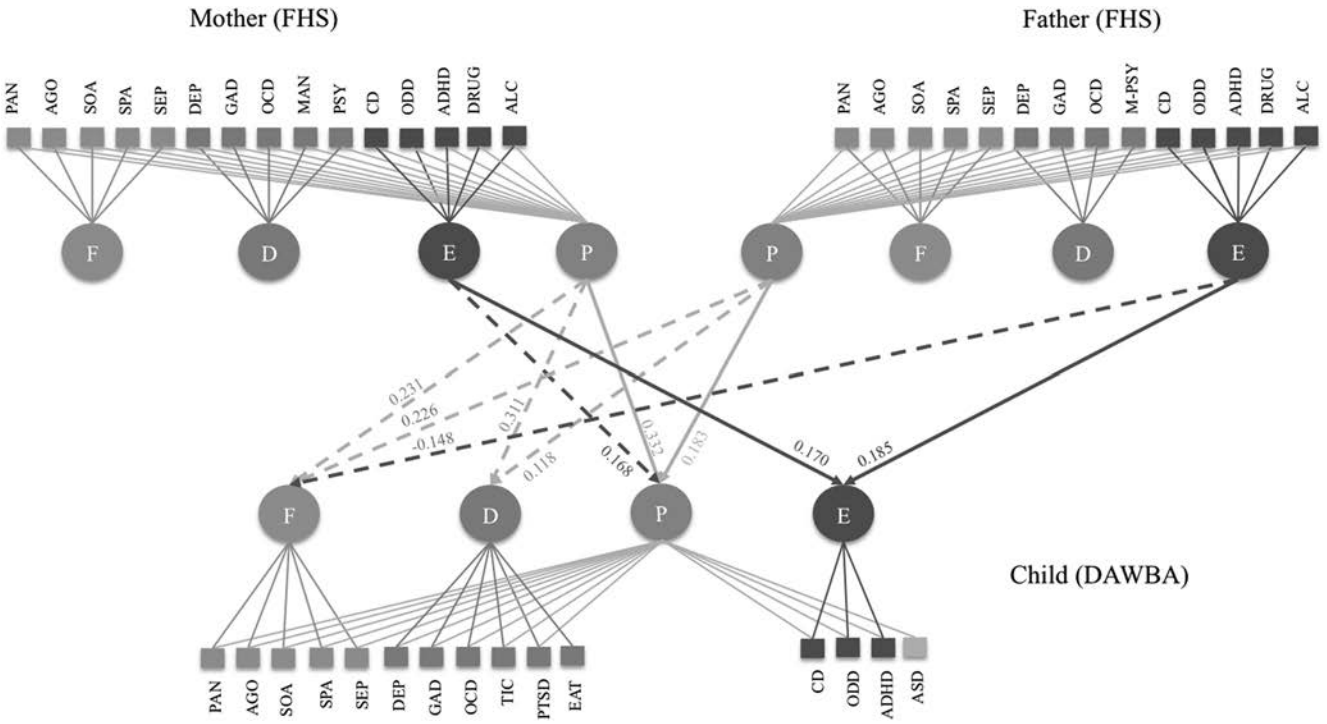

Figure 1. Structural equation model showing associations between father and mother bifactor models of psychopathology and child bifactor models. Values represent regression coefficients. Dashed arrows represent associations between nonhomonymous latent dimensions. FHS = Family History Screen; DAWBA = Development and Well-Being Assessment; D = distress; E = externalizing; P = general psychopathology; F = fear; PAN = panic; AGO = agoraphobia; SOA = social anxiety; SPA = specific anxiety; SEP = separation anxiety; DEP = depression; GAD = generalized anxiety disorder; OCD = obsessive–compulsive disorder; MAN = mania; PSY = psychosis; CD = conduct disorder; ODD = oppositional defiant disorder; ADHD = attention-deficit/hyperactivity disorder; DRUG = drug abuse or dependence; ALC = alcohol abuse or dependence; M-PSY = mania–psychosis (collapsed); TIC = tic; PTSD = posttraumatic stress disorder; EAT = eating disorders; ASD = autism spectrum disorder. See the online article for the color version of this figure.

measure of child EF, such that higher P factor values were associated with lower global child EF.

In this community sample of children and parents from Brazil, Watson and colleagues' bifactor model of psychopathology was the best fitting model (Simms et al., 2008; Slade & Watson, 2006). Yet, the distress and fear factors were not orthogonal to one another, suggesting incomplete distinctions between the two, somewhat in line with traditional ideas of an internalizing–externalizing dichotomy (Kendler et al., 2003; Kessler et al., 2011; Lahey et al., 2008). This is in line with the hierarchical models of psychopathology suggesting that, at the highest level, there is a P factor, with internalizing–externalizing just below, followed by distress, fear, and externalizing below that (Markon, 2010; Watson, 2005). Although our results are most consistent with Watson (2005) bifactor model, it is possible that other models adequately describe the structure of psychopathology at different levels of analyses, in different types of samples, and in relation to different predictors (see Markon, 2010; Watson, 2005).

Our results also provide support for the P factor hypothesis. Specific factors in both child and parent models accounted for little variance and exhibited low reliability on the basis of omega hierarchical statistics. Therefore, at least in this community sample, there seems to be little variance left after accounting for the general P factor for both child and parent models using two different instruments, the DAWBA and FHS. This is in accordance with results in a recent study (Rodriguez, Reise, & Haviland, 2016), which calculated omega hierarchical statistics for 50 common instruments used in psychopathology and showed that—after accounting for the general factor—there was no reliable variance left for specific factors from each instrument. Murray and colleagues' (2016) work further indicated that P factor reliability, as measured with omega hierarchical statistics, was stable over the course of development. Nevertheless, it is important to note that bifactor models fit better than do one-dimensional models, which suggests that the specific factors are meaningful to the correlational structure of psychopathology. Therefore, the low reliability estimates for specific factors do not suggest they do not exist but rather seem to indicate that the current instruments might be limited in their ability to capture specific information above and beyond general information. Perhaps efforts directed toward creating instruments focusing on specific aspects of psychopathology might be justified.

Our results suggest, in addition to the reliability data, high external validity of the P factor, at least in this population sample. First, familial associations were mostly statistically significant for maternal and paternal P factors, which were significantly associated with both the child's P factor and specific fear, distress, and externalizing dimensions. This is consistent with prior work suggesting that familial transmission occurs at higher order levels (Hicks, Krueger, Iacono, McGue, & Patrick, 2004). Therefore, it is possible that co-occurrence between mental disorders occurs predominantly because they are influenced by the same set of genetic and environmental factors (Lahey et al., 2012; Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011). Second, correlations with child global EF were significant for only the P factor, and no significant associations were found for any of the child specific factors, which is also in accordance with results of other studies (Caspi et al., 2014). These results underscore the possibility of shared etiological influences between mental health and cognition, given EF deficits are common in several psychiatric conditions. This pattern of associations is consistent with results from behavioral genetics studies, which suggest the model of generalist genes and specialist environments, which are posited for both psychopathology (Lahey et al., 2011) and cognition (Kovas & Plomin, 2007).

Although our results speak in favor of a general factor of psychopathology, it is important to consider alternative explanations for the present findings. An overarching possibility is that results are explained by common method variance. That is, the P factor could merely represent shared variance among items due to a common perspective, biased symptom reporting (i.e., tendencies to evaluate in generally negative or positive terms; Lahey et al., 2012), or overall impairment due to symptomatic dimensions (Laceulle, Vollebergh, & Ormel, 2015). Yet, associations between the P factor and objective measures of child function, such as global measure of EF, suggest the P factor is likely measuring something over and above shared source variance. In addition, diagnostic interviews might provide only a static picture of very dynamic processes of symptomatology, in which symptoms are causally related to others in distinct processes but only captured poorly by imperfect diagnostic systems (Laceulle et al., 2015). Given that, research that uses experience sampling methods and frequent assessment of behaviors, emotions, and thoughts might be particularly helpful in capturing the time course of symptomatic relationships and reveal additional methods for reliably capturing specificities in symptomatic presentations.

Results of the current study suggest the importance of the general P factor for understanding child and parental psychopathology, transmission of familial risk, and development of global EF. Such general liability might best be targeted with early intervention efforts directed broadly across the childhood spectrum of psychopathology, including, for example, parenting training and emotion regulation skills.

There are some limitations of the current work that should be noted. First, although validated, the FHS focused mainly on diagnostic estimates (for screening purposes) rather than diagnostic assessment. This might have limited our power to examine specific factors in parents. Nevertheless, fine-grained analysis from DAWBA in children also supported FHS results in adults, reinforcing the overarching role of the P factor. Second, we investigated associations with only child global EF, and therefore we cannot exclude more specific associations with other domains of cognitive functioning. Third, assessment of child psychopathology was performed using only parental reports, and FHS data were based mainly on maternal reports on fathers. Finally, although the study extended prior work on models of psychopathology to a Brazilian sample, the current results may not generalize to other countries or populations (e.g., clinical samples), and our sample and perhaps at times unrepresentative prevalence rates likely influenced study results (e.g., the model supported, the prominence of the P factor). Some strengths should also be noted. This is the first study to provide a comprehensive test of both validity and reliability of competing models of psychopathology with strong support in the literature. It is one of the few in a southern, Latin American culture and country, making use of a large sample size and an external validator (cognition) that did not share source variance with diagnostic data.

Results of the current study support a bifactor model of child psychopathology with a general P factor and distress, fear, and externalizing specific factors. Yet, the specific factors accounted for little variance and exhibited few specific associations with external validation indices such as parental psychopathology familial risk and child global EF. Thus, the P factor merits special consideration in current research on the structure of child psychopathology and, more speculatively, may suggest important early intervention avenues.

References

Achenbach, T. M., & Edelbrock, C. S. (1978). The classification of child psychopathology: A review and analysis of empirical efforts. *Psychological Bulletin, 85,* 1275–1301. http://dx.doi.org/10.1037/0033-2909.85.6.1275

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Andrews, G., Goldberg, D. P., Krueger, R. F., Carpenter, W. T., Hyman, S. E., Sachdev, P., & Pine, D. S. (2009). Exploring the feasibility of a meta-structure for *DSM–V* and ICD-11: Could it improve utility and validity? *Psychological Medicine, 39,* 1993–2000. http://dx.doi.org/10.1017/S0033291709990250

Barkley, R. A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin, 121,* 65–94. http://dx.doi.org/10.1037/0033-2909.121.1.65

Bentler, P. M. (2009). Alpha, dimension-free, and model-based internal consistency reliability. *Psychometrika, 74,* 137–143. http://dx.doi.org/10.1007/s11336-008-9100-1

Bitsakou, P., Psychogiou, L., Thompson, M., & Sonuga-Barke, E. J. (2008). Inhibitory deficits in attention-deficit/hyperactivity disorder are independent of basic processing efficiency and IQ. *Journal of Neural Transmission, 115,* 261–268. http://dx.doi.org/10.1007/s00702-007-0828-z

Blanco, C., Wall, M. M., He, J. P., Krueger, R. F., Olfson, M., Jin, C. J., . . . Merikangas, K. R. (2015). The space of common psychiatric disorders in adolescents: Comorbidity structure and individual latent liabilities. *Journal of the American Academy of Child & Adolescent Psychiatry, 54,* 45–52. http://dx.doi.org/10.1016/j.jaac.2014.10.007

Carragher, N., Krueger, R. F., Eaton, N. R., & Slade, T. (2015). Disorders without borders: Current and future directions in the meta-structure of mental disorders. *Social Psychiatry and Psychiatric Epidemiology, 50,* 339–350. http://dx.doi.org/10.1007/s00127-014-1004-z

Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., . . . Moffitt, T. E. (2014). The p factor: One general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science, 2,* 119–137. http://dx.doi.org/10.1177/2167702613497473

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A., Jr., Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry, 26,* 57–63. http://dx.doi.org/10.1001/archpsyc.1972.01750190059011

Goodman, A., Heiervang, E., Collishaw, S., & Goodman, R. (2011). The "DAWBA bands" as an ordered-categorical measure of child mental health: Description and validation in British and Norwegian samples. *Social Psychiatry and Psychiatric Epidemiology, 46,* 521–532. http://dx.doi.org/10.1007/s00127-010-0219-x

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review, 106,* 458–490. http://dx.doi.org/10.1037/0033-295X.106.3.458

Hicks, B. M., Krueger, R. F., Iacono, W. G., McGue, M., & Patrick, C. J. (2004). Family transmission and heritability of externalizing disorders: A twin-family study. *Archives of General Psychiatry, 61,* 922–928. http://dx.doi.org/10.1001/archpsyc.61.9.922

Hogan, A. M., Vargha-Khadem, F., Kirkham, F. J., & Baldeweg, T. (2005). Maturation of action monitoring from adolescence to adulthood: An ERP study. *Developmental Science, 8,* 525–534. http://dx.doi.org/10.1111/j.1467-7687.2005.00444.x

Hu, L., & Bentler, P. M. (1999). Cutoff criteria for fit indices in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6,* 1–55. http://dx.doi.org/10.1080/10705519909540118

Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry, 60,* 929–937. http://dx.doi.org/10.1001/archpsyc.60.9.929

Kessler, R. C., Petukhova, M., & Zaslavsky, A. M. (2011). The role of latent internalizing and externalizing predispositions in accounting for the development of comorbidity among common mental disorders. *Current Opinion in Psychiatry, 24,* 307–312. http://dx.doi.org/10.1097/YCO.0b013e3283477b22

Kim, H., & Eaton, N. R. (2015). The hierarchical structure of common mental disorders: Connecting multiple levels of comorbidity, bifactor models, and predictive validity. *Journal of Abnormal Psychology, 124,* 1064–1078. http://dx.doi.org/10.1037/abn0000113

Kline, R. B. (2005). *Principles and practice of structural equation modeling* (2nd ed.). New York, NY: Guilford Press.

Kotov, R., Chang, S. W., Fochtmann, L. J., Mojtabai, R., Carlson, G. A., Sedler, M. J., & Bromet, E. J. (2011). Schizophrenia in the internalizing-externalizing framework: A third dimension? *Schizophrenia Bulletin, 37,* 1168–1178. http://dx.doi.org/10.1093/schbul/sbq024

Kovas, Y., & Plomin, R. (2007). Learning abilities and disabilities: Generalist genes, specialist environments. *Current Directions in Psychological Science, 16,* 284–288. http://dx.doi.org/10.1111/j.1467-8721.2007.00521.x

Krueger, R. F. (1999). The structure of common mental disorders. *Archives of General Psychiatry, 56,* 921–926. http://dx.doi.org/10.1001/archpsyc.56.10.921

Krueger, R. F., Chentsova-Dutton, Y. E., Markon, K. E., Goldberg, D., & Ormel, J. (2003). A cross-cultural study of the structure of comorbidity among common psychopathological syndromes in the general health care setting. *Journal of Abnormal Psychology, 112,* 437–447. http://dx.doi.org/10.1037/0021-843X.112.3.437

Krueger, R. F., Hicks, B. M., Patrick, C. J., Carlson, S. R., Iacono, W. G., & McGue, M. (2002). Etiologic connections among substance dependence, antisocial behavior, and personality: Modeling the externalizing spectrum. *Journal of Abnormal Psychology, 111,* 411–424. http://dx.doi.org/10.1037/0021-843X.111.3.411

Krueger, R. F., & Markon, K. E. (2006). Reinterpreting comorbidity: A model-based approach to understanding and classifying psychopathology. *Annual Review of Clinical Psychology, 2,* 111–133. http://dx.doi.org/10.1146/annurev.clinpsy.2.022305.095213

Krueger, R. F., Markon, K. E., Patrick, C. J., Benning, S. D., & Kramer, M. D. (2007). Linking antisocial behavior, substance use, and personality: An integrative quantitative model of the adult externalizing spectrum. *Journal of Abnormal Psychology, 116,* 645–666. http://dx.doi.org/10.1037/0021-843X.116.4.645

Lahey, B. B., Applegate, B., Hakes, J. K., Zald, D. H., Hariri, A. R., & Rathouz, P. J. (2012). Is there a general factor of prevalent psychopathology during adulthood? *Journal of Abnormal Psychology, 121,* 971–977. http://dx.doi.org/10.1037/a0028355

Lahey, B. B., Rathouz, P. J., Keenan, K., Stepp, S. D., Loeber, R., & Hipwell, A. E. (2015). Criterion validity of the general factor of psychopathology in a prospective study of girls. *Journal of Child Psychology and Psychiatry, 56,* 415–422. http://dx.doi.org/10.1111/jcpp.12300

Lahey, B. B., Rathouz, P. J., Van Hulle, C., Urbano, R. C., Krueger, R. F., Applegate, B., . . . Waldman, I. D. (2008). Testing structural models of *DSM–IV* symptoms of common forms of child and adolescent psychopathology. *Journal of Abnormal Child Psychology, 36,* 187–206. http://dx.doi.org/10.1007/s10802-007-9169-5

Lahey, B. B., Van Hulle, C. A., Singh, A. L., Waldman, I. D., & Rathouz, P. J. (2011). Higher-order genetic and environmental structure of prevalent forms of child and adolescent psychopathology. *Archives of General Psychiatry, 68,* 181–189. http://dx.doi.org/10.1001/archgenpsychiatry.2010.192

Lucke, J. F. (2005). The alfa and the omega of congeneric test theory: An extension of reliability and internal consistency to heterogeneous tests. *Applied Psychological Measurement, 29,* 65–81. http://dx.doi.org/10.1177/0146621604270882

Markon, K. E. (2010). Modeling psychopathology structure: A symptom-level analysis of Axis I and II disorders. *Psychological Medicine, 40,* 273–288. http://dx.doi.org/10.1017/S0033291709990183

McDonald, R. (1999). *Test theory: A unified approach*. Mahwah, NJ: Erlbaum.

Meehl, P. E. (1992). Factors and taxa, traits and types, differences of degree and differences in kind. *Journal of Personality, 60,* 117–174. http://dx.doi.org/10.1111/j.1467-6494.1992.tb00269.x

Meehl, P. E. (2004). What's in a taxon? *Journal of Abnormal Psychology, 113,* 39–43. http://dx.doi.org/10.1037/0021-843X.113.1.39

Miyake, A., & Friedman, N. P. (2012). The nature and organization of individual differences in executive functions four general conclusions. *Current Directions in Psychological Science, 21,* 8–14. http://dx.doi.org/10.1177/0963721411429458

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000) The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41,* 49–100. http://dx.doi.org/10.1006/cogp.1999.0734

Morgan, A. B., & Lilienfeld, S. O. (2000). A meta-analytic review of the relation between antisocial behavior and neuropsychological measures of executive function. *Clinical Psychology Review, 20,* 113–136. http://dx.doi.org/10.1016/S0272-7358(98)00096-8

Murray, A. L., Eisner, M., & Ribeaud, D. (2016). The development of the general factor of psychopathology "p factor" through childhood and adolescence. *Journal of Abnormal Child Psychology*. Advance online publication. http://dx.doi.org/10.1007/s10802-016-0132-*1*

Muthén, L. K., & Muthén, B. O. (1998–2014). *Mplus user's guide, fourth edition*. Los Angeles, CA: Author.

Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 37,* 51–87. http://dx.doi.org/10.1111/j.1469-7610.1996.tb01380.x

Rao, S. M., Mayer, A. R., & Harrington, D. L. (2001). The evolution of brain activation during temporal processing. *Nature Neuroscience, 4,* 317–323. http://dx.doi.org/10.1038/85191

Reise, S. P. (2012). Invited paper: The rediscovery of bifactor measurement models. *Multivariate Behavioral Research, 47,* 667–696. http://dx.doi.org/10.1080/00273171.2012.715555

Rodriguez, A., Reise, S. P., & Haviland, M. G. (2016). Applying bifactor statistical indices in the evaluation of psychological measures. *Journal of Personality Assessment, 98,* 223–237.

Salum, G. A., Gadelha, A., Pan, P. M., Moriyama, T. S., Graeff-Martins, A. S., Tamanaha, A. C., . . . Rohde, L. A. (2015). High risk cohort study for psychiatric disorders in childhood: Rationale, design, methods and preliminary results. *International Journal of Methods in Psychiatric Research, 24,* 58–73. http://dx.doi.org/10.1002/mpr.1459

Simms, L. J., Grös, D. F., Watson, D., & O'Hara, M. W. (2008). Parsing the general and specific components of depression and anxiety with bifactor modeling. *Depression and Anxiety, 25*(7), E34–E46. http://dx.doi.org/10.1002/da.20432

Slade, T., & Watson, D. (2006). The structure of common *DSM–IV* and ICD-10 mental disorders in the Australian general population. *Psychological Medicine, 36,* 1593–1600. http://dx.doi.org/10.1017/S0033291706008452

Toplak, M. E., & Tannock, R. (2005). Tapping and anticipation performance in attention deficit hyperactivity disorder. *Perceptual and Motor Skills, 100,* 659–675. http://dx.doi.org/10.2466/PMS.100.3.659-675

Vandierendonck, A., Kemps, E., Fastame, M. C., & Szmalec, A. (2004). Working memory components of the Corsi blocks task. *British Journal of Psychology, 95,* 57–79. http://dx.doi.org/10.1348/000712604322779460

Watson, D. (2005). Rethinking the mood and anxiety disorders: A quantitative hierarchical model for *DSM–V*. *Journal of Abnormal Psychology, 114,* 522–536. http://dx.doi.org/10.1037/0021-843X.114.4.522

Wechsler, D. (2002). *WISC-III: Escala de Inteligência Wechsler para Crianças: Manual* [Wechsler Intelligence Scale for Children-Third Edition]. São Paulo, Brazil: Casa do Psicólogo.

Weissman, M. M., Wickramaratne, P., Adams, P., Wolk, S., Verdeli, H., & Olfson, M. (2000). Brief screening for family psychiatric history: The Family History Screen. *Archives of General Psychiatry, 57,* 675–682. http://dx.doi.org/10.1001/archpsyc.57.7.675

Willcutt, E. G., Doyle, A. E., Nigg, J. T., Faraone, S. V., & Pennington, B. F. (2005). Validity of the executive function theory of attention-deficit/hyperactivity disorder: A meta-analytic review. *Biological Psychiatry, 57,* 1336–1346. http://dx.doi.org/10.1016/j.biopsych.2005.02.006

Wright, A. G., Krueger, R. F., Hobbs, M. J., Markon, K. E., Eaton, N. R., & Slade, T. (2013). The structure of psychopathology: Toward an expanded quantitative empirical model. *Journal of Abnormal Psychology, 122,* 281–294. http://dx.doi.org/10.1037/a0030133

Zinbarg, R. E., Revelle, W., Yovel, I., & Li, W. (2005). Cronbach's α, Revelle's β, and McDonald's ω_H: Their relations with each other and two alternative conceptualizations of reliability. *Psychometrika, 70,* 123. http://dx.doi.org/10.1007/s11336-003-0974-7

Received October 20, 2015
Revision received July 11, 2016
Accepted August 9, 2016 ■