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The Structure of Psychopathology in Adolescence: Replication of a General Psychopathology Factor in the TRAILS Study

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Abstract

This study aimed to replicate a study by Caspi and colleagues, which proposed that the structure of psychopathology is characterized by a general psychopathology factor, in addition to smaller internalizing and externalizing factors. Our study expanded the approach of the original by using continuous adolescent data and testing additional models, including both self- and parent-reported data, to bolster the robustness of the findings. Our findings indicate that the structure of psychopathology is best characterized by a model including a general factor, in addition to smaller internalizing and externalizing factors. These results emphasize the importance of this model for understanding the structure of psychopathology. Given the increasing emphasis on the importance of, and need for, replication, the overall evidence of a general factor seems rather robust.

Keywords

psychopathology, replication, confirmatory factor analysis, adolescence

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Traditional research on psychopathology has usually suggested a two-dimensional structure of mental disorders, with regard to both adult diagnostic data on mental disorders (Krueger, Caspi, Moffitt, & Silva, 1998; Krueger, Johnson, & Kling, 2006) and factor analytic studies of continuous measures of psychopathology in children, adolescents, and adults (Achenbach, 1966; Achenbach & Edelbrock, 1978). Over the decades, these findings have created the widely accepted and demonstrated distinction between two different, though positively correlated, factors underlying psychopathology: internalizing and externalizing problems/disorders (Forbush & Watson, 2013; Kendler, Prescott, Myers, & Neale, 2003; Slade & Watson, 2006; Vollebergh et al., 2001). More recent research has provided evidence for a third factor, labeled *thought problems* or *dysregulation*, which includes psychotic symptoms, for example. These symptoms have usually been excluded in studies on common mental disorders due to methodological and conceptual issues (Kotov, Gamez, Schmidt, & Watson, 2010; Wigman et al., 2011).

Recently, this two- (or three-) dimensional structure of mental disorders has been debated, with increasing emphasis placed on the existence of a general psychopathology factor instead of, or in addition to, the more specific dimensions. Specifically, a recent series of confirmatory factor analyses (CFAs) of longitudinal psychopathology data from the Dunedin study has challenged traditional models by providing strong support for a latent general overall severity factor, with high loadings in all individual symptom dimensions (Caspi et al., 2013). It is interesting that when taking into account this general psychopathology factor, the internalizing and

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externalizing factors remained, but loadings of symptoms on the internalizing and externalizing factors decreased substantially, whereas the usually positive correlation between internalizing and externalizing factors became a negative correlation. In addition, the thought-problem symptoms did not form a third latent factor in the general psychopathology model, but rather, due to their very high loadings on the general factor, appeared to be core symptoms of the general psychopathology factor instead. This general dimension was ingeniously coined the p-factor because it conceptually parallels a familiar dimension in psychology: the g-factor of general intelligence.

Caspi and colleagues are not the first to suggest an underlying general psychopathology factor. The notion of a general psychopathology factor is similar to the "index of definition" (ID; Wing, Mann, Leff, & Nixon, 1978). Also, the work of Gibbons and Hedeker and, more recently, Lahey and colleagues, for example, proposed a general psychopathology factor in addition to more domain-specific latent factors (Gibbons & Hedeker, 1992; Lahey et al., 2012; Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011). Similar to Caspi's, these studies did not model the general psychopathology factor as a higher-order factor, but rather at the same level as the domain-specific latent factors, with all specific symptoms loading directly on the general psychopathology factor (i.e., a "bifactor" model). Nonetheless, although the various models may not be incompatible, the proposed models differ from each other in various ways. For example, Caspi and colleagues allowed the internalizing and externalizing factors to correlate, whereas the study by Gibbons and Hedeker only included internalizing symptoms, and the model proposed by Lahey did not allow for correlations between the internalizing and externalizing latent factors in the general factor model. By allowing the factors to correlate, Caspi revealed that the positive correlation between the internalizing and externalizing factors turned negative in the general factor model, which suggests that internalizing and externalizing problems are clearly distinct *after* controlling for general vulnerability for psychopathology.

In the current article, we attempt to replicate and expand the findings by Caspi and colleagues. Caspi's model seems most elaborate with regard to the symptom dimensions included and matches best with our longitudinal cohort data, which maximizes comparability. Aiming at providing a close replication of Caspi's model, we compared the same models, including the same paths and using the same model specifications. Nonetheless, our study design differs from Caspi's approach in various ways. Whereas Caspi et al. focused on (young) adult samples, the current study uses data from a large sample of Dutch adolescents. Given that many mental disorders have their onset during early adolescence (Bernstein, Borchardt, & Perwien, 1996; Hankin et al., 1998), attempting to replicate Caspi's findings in an adolescent sample may provide insight into whether the model as proposed by Caspi also reflects the structure of psychopathology in adolescents. In addition, we added symptoms that are known to onset before the adolescent years (e.g., attention problems) or that are highly adolescence-specific (i.e., delinquency), and we used continuous questionnaire data instead of (semi)structured diagnostic interview data. Finally, we aimed to check the robustness of the best-fitting model by adding all parent-reported symptom data corresponding to the self-report measures included in the main analyses.

Method

Sample

Our study used data from the first, second, third, and fourth wave of the TRacking Adolescents' Individual Lives Survey (TRAILS; Ormel et al., 2012). TRAILS is a large prospective cohort study of 2,230 Dutch adolescents, followed bi- or triennially from age 11. Sample selection has been extensively described by de Winter and colleagues (2005).

Mean age was 10.5 years (SD = 0.58) at the first wave, 13.6 (SD = 0.59) at the second wave, 16.1 (SD = 0.59) at the third wave, and 19.1 (SD = 0.60) at the fourth wave. About 10% of our sample was identified as having a non-Western ethnicity. Having a non-Western ethnicity was defined as having at least one parent born in a non-Western country (N = 230 at Wave 1), of whom 20% had a parent or parents born in Surinam, 16% in the Dutch Antilles, 16% in Indonesia, 6.5% in Morocco, 5.5% in Turkey, and 36% in other non-Western countries such as Iraq, Iran, or Somalia (see also Vollebergh et al., 2005). Attrition during follow-ups was somewhat higher in males and in adolescents of non-Western ethnicity, and in those with divorced parents, low socioeconomic status (SES), low IQ and academic achievement, or poor physical health or with behavior and substance use problems (Nederhof et al., 2012). Each assessment wave was approved by the national ethical committee (CCMO, www.ccmo.nl).

Measures

Self-report. TRAILS assessed problem behaviors, as rated by adolescent subjects, using the following instruments: the Youth Self Report (YSR; T1, T2, T3), the Adult Self Report (ASR; T4), the Revised Child Anxiety and Depression Scale (RCADS; T1, T2, T3), and the Community Assessment of Psychic Experiences (CAPE; T3). The YSR has been widely used to assess self-report symptom

dimensions (Achenbach, 1991). Symptom dimensions covered by the YSR, and included in the current study, are anxious-depressed, withdrawn-depressed, aggressive behavior, delinquent behavior, attention-hyperactivity problems, and thought problems. At T4, when adolescents were 19 years old on average, the ASR replaced the YSR. Although the ASR contains slightly different items (more appropriate for older adolescents) the symptoms covered were identical to the symptoms covered by the YSR. Items were scored on a 3-point Likert-type scale (0 = *not true* to 2 = *very or often true*).

The RCADS consists of a list of items measuring anxiety, obsessive-compulsive, and depression symptoms (Chorpita, Yim, Moffitt, Umemoto, & Francis, 2000; Mathyssek et al., 2013). We focused on the anxiety and obsessive-compulsive scales because these were not fully covered by the YSR/ASR. The symptom dimensions covered were generalized anxiety disorder, social anxiety, separation anxiety, panic disorder, and obsessive-compulsive disorder. Items were scored on a 4-point Likert scale (0 = *never* to 3 = *always*).

TRAILS used the CAPE positive experiences scale (20 items) to assess psychotic experiences (Wigman et al., 2011). Each item assesses (a) frequency and (b) distress associated with an experience, both scored on a 4-point Likert-type scale (0 = *never/not distressed* to 3 = *nearly always/very distressed*). For the current study, we used both the frequency and the distress variables.

Parent report. To test the robustness of our models, we added all parent-reported data that corresponded to the self-report measures of our final models. The study assessed problem behaviors as rated by the parent using the Child Behavior Checklist (CBCL; T1, T2, T3), which is one of the most commonly used parent-report questionnaires in child and adolescent psychiatric research (Achenbach, 1991; Verhulst & Achenbach, 1995). The symptom dimensions covered by the CBCL are anxious-depressed, withdrawn-depressed, aggressive behavior, delinquent behavior, attention-hyperactivity problems, and thought problems. Items were scored on a 3-point Likert-type scale (0 = *not true* to 2 = *very or often true*).

Statistical analysis

Replicating Caspi's models in adolescent data. In line with Caspi and colleagues, we tested four models using CFA: a *three-correlated-factors model* including internalizing, externalizing, and thought problems (Model A; see Fig. 1); a *bifactor model* with internalizing, externalizing, and thought problems at the first level and a general psychopathology factor at the second level (Model B; see Fig. 1); a *revised-bifactor model* with internalizing and externalizing problems at the first level and the general psychopathology factor at the second level with the thought-problem factor removed from the model (Model B′; see Fig. 1); and a *one-factor model* (Model C; see Fig. 1). Notably, we statistically specified all models to be identical to Caspi's models to maximize comparability. Also identical to Caspi's models, we included an additional wave-factor into each model to expose age- and assessment-related variance.

All CFA analyses were performed using robust maximum likelihood estimation in Mplus version 7.1 (Muthén & Muthén, 2007). We assessed how well each model fit the data using the chi-square value, the comparative fit index (CFI), the Tucker–Lewis index (TLI), and the root mean square error of approximation (RMSEA). Nonsignificant chi-square values normally reflect good model fit. However, when using large sample sizes this test is strongly overpowered and the fit statistics are significant. CFI and TLI values greater than .95 indicate a good fit, as well as RMSEA scores less than .05 (Bollen & Curran, 2006). The critical CFI/TLI value for an acceptable model is .90, and for RMSEA the critical value for acceptable model fit is .08.

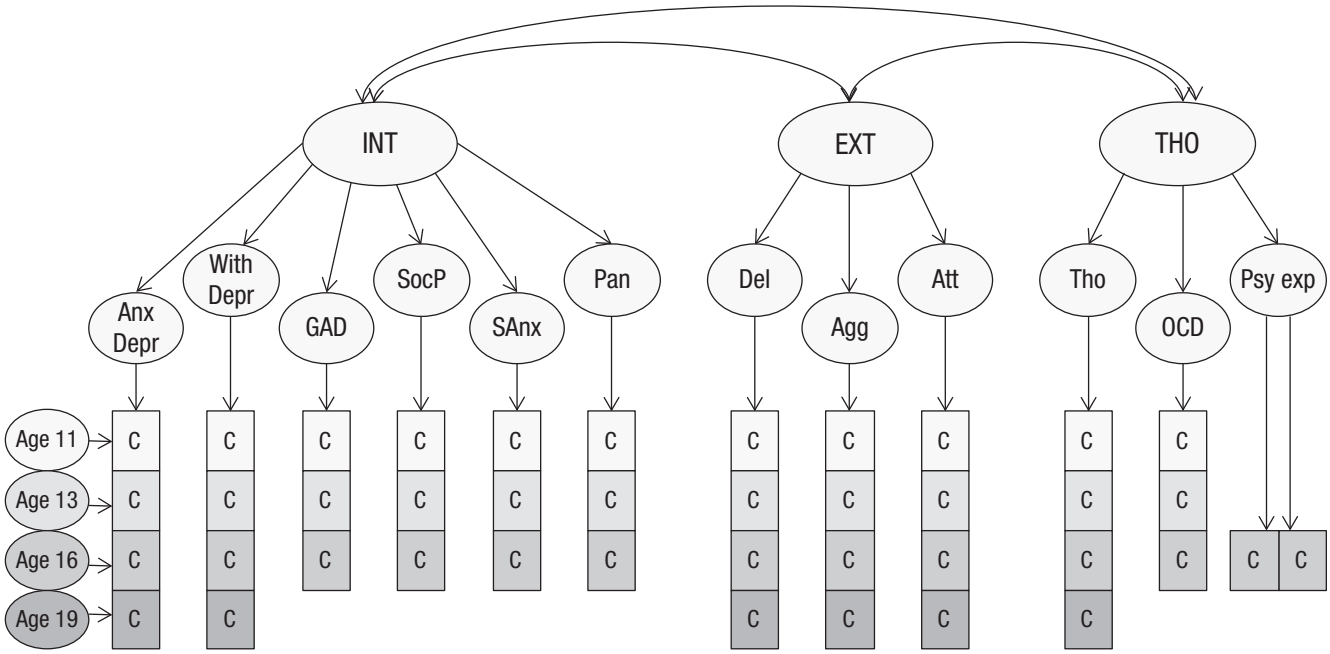
Checking the robustness of the best-fitting models by adding parent-reported symptoms. For our best fitting models we added all parent-reported symptom data that corresponded to the self-report measures included in the main analyses (Fig. 2). For these models, we also included a reporter factor to pull out informant-related variance.

Results

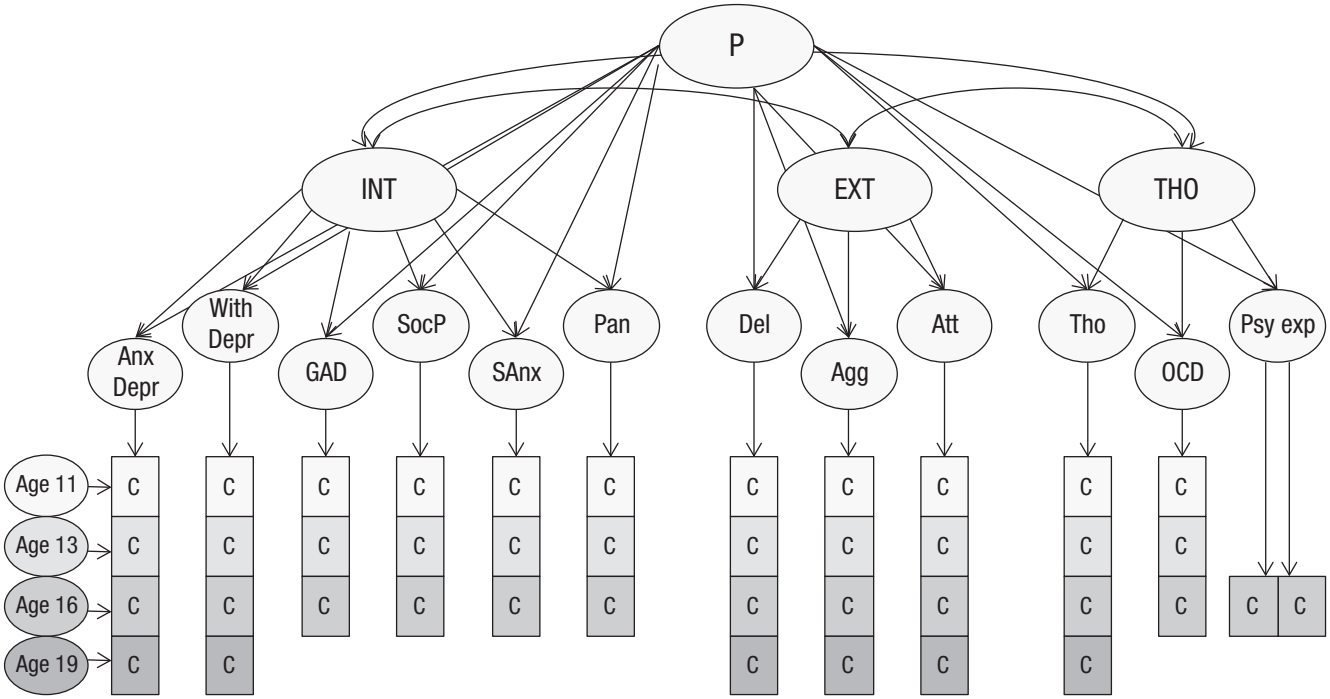
The structure of psychopathology

Replicating Caspi's models in adolescent data. We aimed at replicating the four models tested by Caspi and colleagues in as much detail as possible. In the first model (A), we tested the hypothesis that the observed symptom dimensions are characterized by three correlated factors: internalizing (including anxious-depressed, withdrawn-depressed, GAD, social anxiety, separation anxiety, and panic disorder), externalizing (including delinquency, aggression, and attention problems), and thought problems (including thought problem symptoms, OCD, and psychotic experiences; see Fig. 1). Model fit statistics showed that the data did not fit the model adequately: $\chi^2(723) = 5148.82$, CFI = .890, TLI = .875, RMSEA = .052, 90% confidence interval (CI) = .051–.054. In addition, the standardized factor loading of aggression on the latent externalizing factor was larger than 1 and the residual variance of aggression was negative. This indicated that the three-factor model was not optimally identified and thus did not explain the structure of the symptom

Model A: Three-correlated factor



Model B: Bi-factor model



(continued)

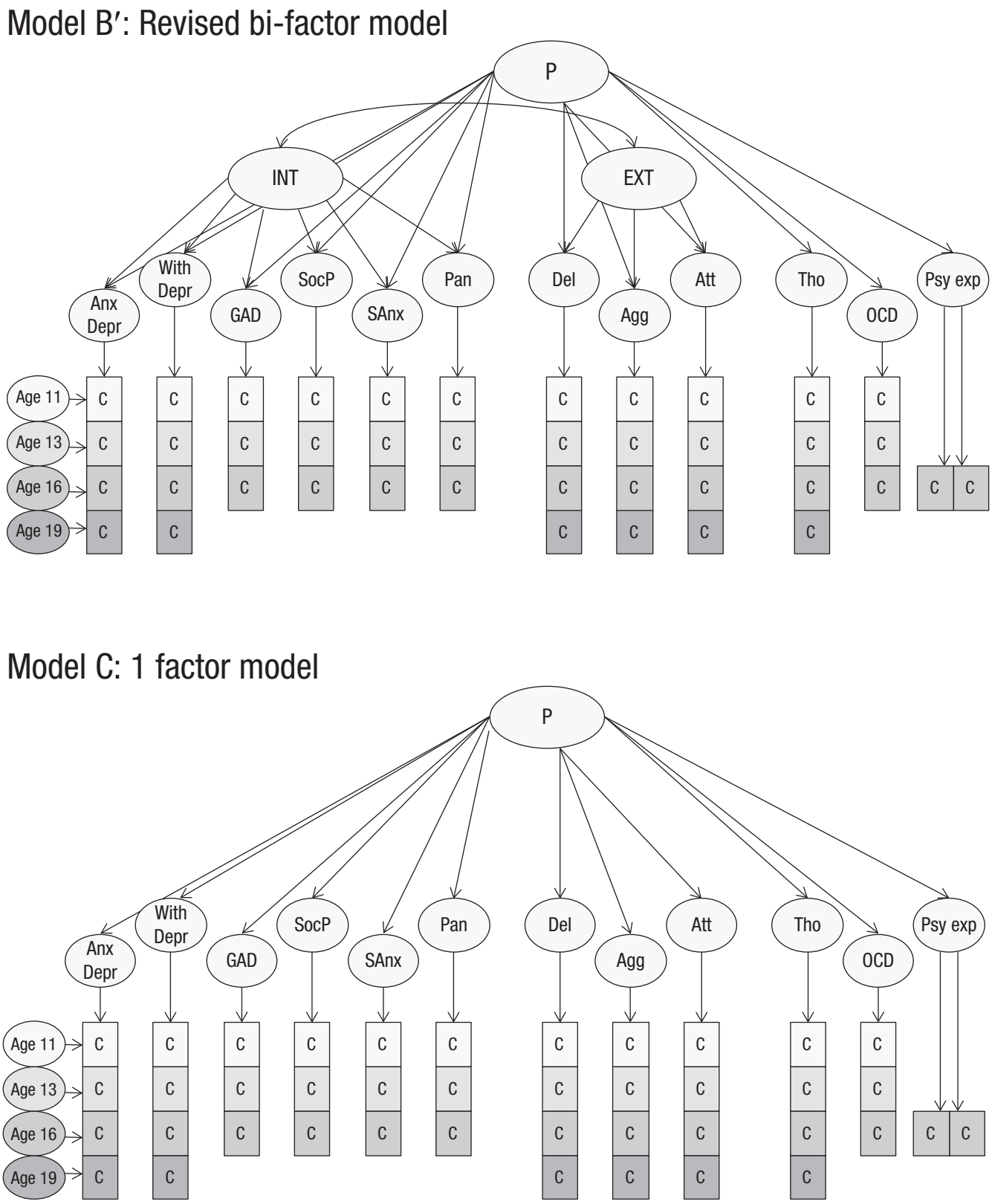


Fig. 1. The structure of psychopathology. Three models were tested using confirmatory factor analysis: a correlated-factors model (Model A), a bifactor model (Model B; i.e., Model A plus all symptom dimensions loading on a general factor), and a one-factor model (Model C). Model B′ shows the revised-bifactor model (i.e., Model B but without a separate thought-problem factor).

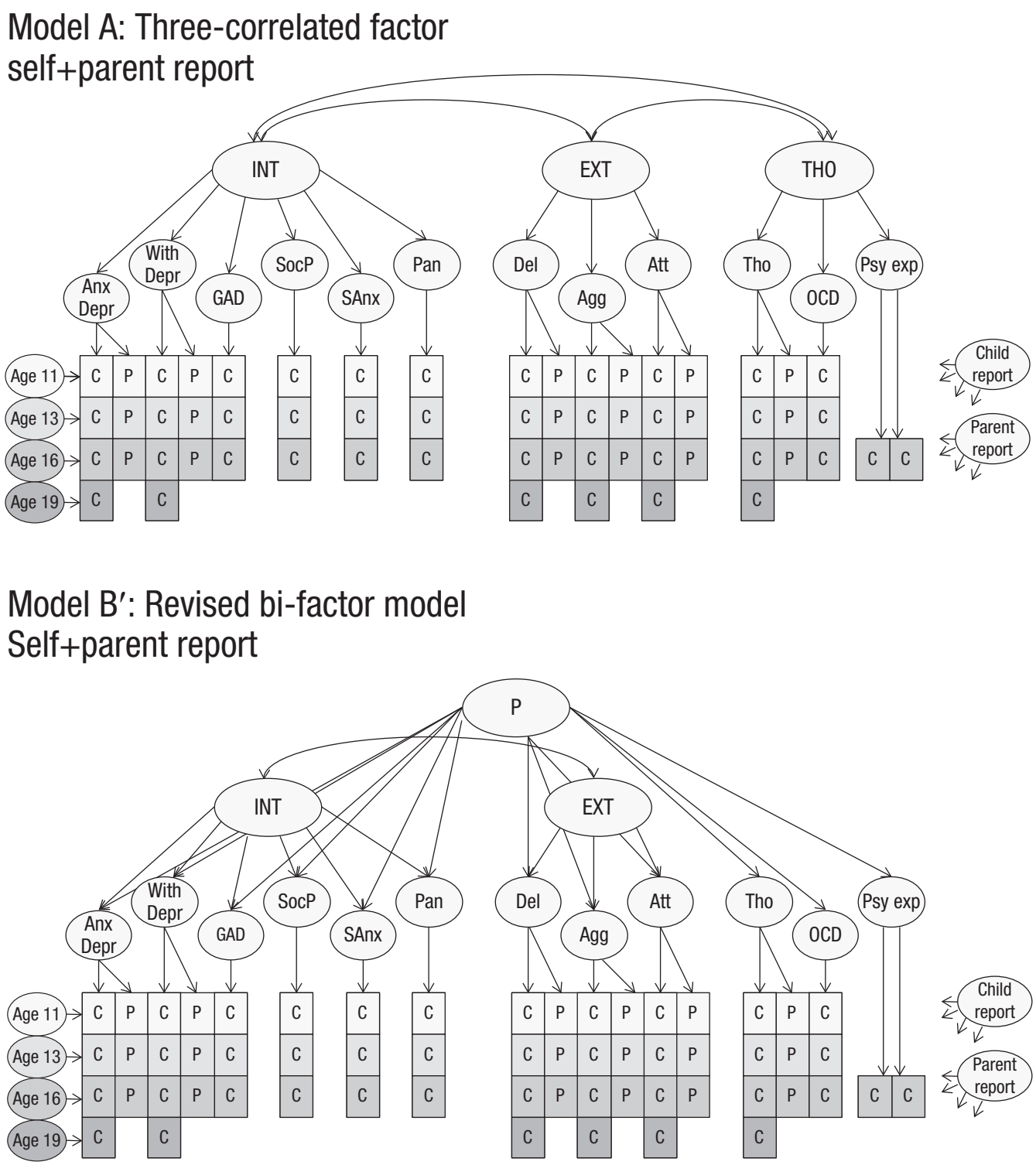


Fig. 2. Two additional models were tested: Model $A_{\text{Self+parent report}}$ (Model A, but with all available parent-report data added to the model) and Model $B'_{\text{self+parent report}}$ (Model B′, but with all available parent-report data added to the model).

dimensions across adolescence well. Table 1 shows this model with standardized factor loadings and the correlations between the three latent variables: internalizing, externalizing, and thought problems. Note that caution is needed in interpreting these loadings and correlations because of the model identification problems.

Table 1. Standardized Factor Loadings and Correlations of the Three-Factor Model (A) and the Revised-General-Factor Model (B′)

Statistics, Loadings, and Correlations	Model A			Model B′		
	INT	EXT	Thought	P	INT	EXT
Standardized factor loadings						
Anxious-depressed	0.932			0.856	0.388	
Withdrawn-depressed	0.711			0.736	0.139	
GAD	0.900			0.822	0.368	
Social anxiety	0.880			0.730	0.592	
Separation anxiety	0.844			0.719	0.485	
Panic disorder	0.845			0.835	0.209	
Delinquency		0.847		0.413		0.849
Aggression		1.016		0.655		0.714
Attention problems		0.783		0.726		0.401
Thought problems			0.855	0.869		
OCD			0.907	0.894		
Psychotic experiences			0.970	0.968		
Factor correlations						
Internalizing		0.440	0.883			–0.438
Externalizing			0.612			

Next, we tested the bifactor model (B), hypothesizing that symptom dimensions reflect the three latent factors—internalizing, externalizing, and thought problems—as well as a latent general psychopathology factor (Fig. 1). The model assumed this general factor to directly influence all symptom dimensions. In line with Caspi and colleagues, we constrained the general factor to be uncorrelated with the three (correlated) first-level latent variables. We could not identify this model, which seems to be in line with Caspi and colleagues who also reported that this model was not valid for their data. Subsequently we tested a revised-bifactor model as suggested by Caspi and colleagues (Model B′; see Fig. 1 and Table 1). That is, we removed the latent thought-problem factor from the model, assuming that our identification problems with Model B were mostly due to the variance of the thought-problem factor (i.e., thought-problem symptoms, OCD, and psychotic experiences) being subsumed into the general psychopathology factor. Model fit statistics suggested that the data fit the revised model acceptably: $\chi^2(716) = 4665.65$, CFI = .902, TLI = .887, RMSEA = .050, 90% CI = .048–.051. As in Caspi's study, we could not directly compare the three-factor and revised-bifactor models because the models are not fully nested. But based on the CFI, TLI, and RMSEA, we can conclude that the revised-bifactor model offers a slightly better and more parsimonious solution. It is important that revising the model solved the model identification problems we encountered when fitting the three-factor model to the data (i.e., negative error variance and factor loadings > 1). This suggests that the general-factor model could explain the structure of symptom dimensions across adolescence better than the three-factor model.

Comparing the factor loadings in the three-correlated-factors model versus the revised-bifactor model revealed similar information to that reported by Caspi and colleagues. We found that some symptom dimensions were more indicative of general psychopathology (the general factor) than other symptom dimensions. Overall, internalizing symptom dimensions were more indicative of the general factor than externalizing symptom dimensions. Of the externalizing symptom dimensions, attention problems (not included by Caspi) were more indicative of the general factor than aggression and delinquency; the factor loading of attention problems on the latent externalizing factor reduced by about half after taking the general factor into account. Seemingly, much of the variance initially ascribed to the latent externalizing factor was now subsumed by the general factor. In the revised-bifactor model, all factor loadings of internalizing symptom dimensions on the latent internalizing factor were substantially reduced or even disappeared compared with the three-correlated-factors model, with the largest changes found for withdrawn-depressed, and panic disorder. In addition, and identical to Caspi, we found a significant positive correlation ($r = .440$) between externalizing and internalizing factors in the correlated-factors model, but a negative correlation ($r = -.438$) between externalizing and internalizing factors in the revised-bifactor model. Finally, and like Caspi, we tested the one-factor model in which all symptom dimensions were assigned only to the general factor (Model C; see Fig. 1).

This model could not be identified, which is in line with Caspi and colleagues who also reported that this model was not valid for their data.

Checking the robustness of the best-fitting models by adding parent-reported symptoms. Based on the findings presented earlier, we consider Caspi's revised-bifactor model as the final model. For both this model and the correlated-factors model, we added all parent-reported symptom data that corresponded to the self-report measures included in the main analyses (Fig. 2). The model fitting results were as follows: results for Model $A_{\text{self+parent report}}$: $\chi^2(1521) = 10935.87$, CFI = .848, TLI = .829, RMSEA = .053, 90% CI = .052–.054; results for Model $B'_{\text{self+parent report}}$: $\chi^2(1514) = 10348.30$, CFI = .857, TLI = .838, RMSEA = .051, 90% CI = .050–.052. Thus, testing and comparing Models $A_{\text{self+parent report}}$ and $B'_{\text{self+parent report}}$ revealed similar information as for the self-report-only models (overall model fit was even somewhat better than for the self-report-only models). Path estimates are available on request.

Discussion

In the current study we replicated the findings of Caspi and colleagues, who proposed that the structure of psychopathology is reflected by a general psychopathology factor in addition to a smaller internalizing and externalizing factor. Overall, despite methodological differences (i.e., adolescent sample, continuous measures of psychopathology, and additional symptom dimensions not included by Caspi), our findings clearly and consistently replicated their findings. Our results confirmed the existence of a general psychopathology factor underlying all symptoms of psychopathology. Taking into account this general factor, the internalizing factors in particular, but also the externalizing factors, were clearly attenuated. The robustness of these findings was confirmed by additional model testing in which we added all available parent-report data to the model, thus revealing that the model remains valid when multiple informants are used to measure psychopathology.

The extent to which the specific dimensions loaded on the general factor differed both between and within the broader domains of internalizing, externalizing, and thought problems. In line with Caspi, thought problem symptoms did not form a latent thought problem factor independent from the general factor. This suggests the relative severity of thought problems, and may indicate that the general psychopathology factor should be interpreted as characterizing the overall severity of psychopathology, for which thought problems appear to be core symptoms. In contrast, with the addition of the general factor, enough variance was left to form the internalizing and externalizing factors, indicating that these factors are necessary to provide a full picture of the underlying structure of psychopathology, although the symptom loadings were very strongly attenuated. This raises the question of how the two dimensions should be interpreted once the more general psychopathological characteristics are taken into account (Caspi et al., 2013; Martel, 2013; Oldehinkel, Hartman, de Winter, Veenstra, & Ormel, 2004). Our finding that the usual positive correlation found between these two dimensions turned negative when adding the general factor seems to further complicate interpretation.

We found that, when adding the general factor, the decreases in factor loadings on the latent internalizing/externalizing factors were substantially larger for the internalizing symptom dimensions than for the externalizing. This may either reflect that internalizing problems are more pathological in general or it may just reflect that we included some adolescent specific externalizing symptoms that may naturally decrease when adolescents get older and therefore may not characterize an individual's vulnerability for severe psychopathology. Indeed, when looking at within-domain differences, it seemed that adolescent-specific problems, such as delinquency, were not very indicative of the general factor. Adding more chronic or life-course-persistent externalizing problems (e.g., antisocial personality disorder) might further clarify whether externalizing symptom dimensions are indeed less indicative of psychopathology.

Taken together, our findings clearly and consistently confirmed that the structure of psychopathology is best reflected by a model that includes a general factor of psychopathology. According to Caspi and colleagues, higher scores on the general factor may not only indicate an increased risk for developing a mental disorder, but also that the general factor correlates more strongly with personality, life impairment, developmental history, and brain integrity, than do the internalizing and externalizing factors. In addition to specific consequences, the general factor may also have specific shared determinants such as self-control, neuroticism, and low SES, although conclusive experimental data is still lacking (Brunner, 1997; Moffitt et al., 2011; Neeleman, Bijl, & Ormel, 2004; Neeleman, Ormel, & Bijl, 2001).

Also, as extensively described by Lahey and colleagues (2011), the general factor may have a substantial genetic component. That is, genetic factors may set individuals at risk for all symptom dimensions, albeit in different degrees. This notion seems to fit well with recent genetic-evolutionary ideas about the relationship between mutational load and mental disorders as elaborated especially by Keller and Miller (2006). They argued that mental disorders reflect the inevitable mutational load on the thousands of genes underlying human behavior. The mutations

involved may influence characteristics that affect the risk for any and all mental disorders. This would explain why the genes involved in the etiology of psychopathology seem to operate in a pleiotropic fashion, enhancing risk for any and all disorders rather than breeding true to any single disorder (Smoller, 2013). Keller and Miller's (2006) watershed model of the pathways connecting upstream genes to downstream phenotypes provides an excellent model of how genes may act in a pleiotropic fashion and "contribute" to a general factor.

It is important not to *reify* the general factor. The general factor represents the shared variance among all psychopathology measures, and this can have different causes. One possibility is that all forms of psychopathology, to some extent, share a common etiology. Another alternative is that the factor represents dynamic processes in which one dimension raises the risk of developing another disorder or dimension, rather than a single unitary cause. Current network approaches to psychopathology assume such dynamic processes (Cramer, Waldorp, van der Maas, & Borsboom, 2010). Another alternative is that the general factor reflects distress, which is a concomitant/feature of virtually all mental disorders. Nonetheless, the findings showing a rather strong general factor suggest that there may be multiple characteristics that account for meaningful covariance amongst major forms of psychopathology. In their article, Caspi and colleagues put forward the question of whether the general factor is merely a statistical reductio ad absurdum, or rather is real and meaningful. In response, we like to answer: The general factor certainly seems useful, but how it is best interpreted remains unclear given the many possible interpretations (mutation load, early persistent adversity, dynamic processes, distress, etc.). The same is true for interpreting the internalizing and externalizing factors that remain after the general factor is taken into account. Do these factors truly represent different underlying dimensions of *other* forms of psychopathology, or do they reflect only different behavioral problems in coping with environmental stressors? In the absence of an answer to these questions, the current findings are best interpreted as a step toward a more parsimonious view of the structure of psychopathology than that implied by traditional differentiations between (a) categorically distinct disorders and (b) spectra or domains of disorders that include internalizing, externalizing, and (in some recent studies) thought problems.

Author Contributions

O. M. Laceulle and J. Ormel developed the study concept aiming at replicating the study by Caspi et al. (2013). O. M. Laceulle, W. A. M. Vollebergh, and J. Ormel contributed to the study design. O. M. Laceulle performed the data analysis and drafted the manuscript. All authors provided revisions and approved the final version of the manuscript for submission.

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References

Achenbach, T. M. (1966). The classification of children's psychiatric symptoms: A factor-analytic study. *Psychological Monographs*, *80*, 1–37.

Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 profile*. Burlington: University of Vermont.

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

Barnett, R., & Hunter, M. (2012). Adjustment of siblings of children with mental health problems: Behaviour, self-concept, quality of life and family functioning. *Journal of Child and Family Studies*, *21*, 262–272.

Bernstein, G. A., Borchardt, C. M., & Perwien, A. R. (1996). Anxiety disorders in children and adolescents: A review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, *35*, 1110–1119. doi:10.1097/00004583-199609000-00008

Bollen, K. A., & Curran, P. J. (2006). *Latent curve models: A structural equation perspective*. New York, NY: John Wiley.

Brunner, E. (1997). Stress and the biology of inequality. *BMJ*, *314*, 1472–1476.

Caspi, A., Houts, R. M., Belsky, D. W., Goldman-Mellor, S. J., Harrington, H., Israel, S., . . . Moffitt, T. E. (2013). The p factor: One general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science*, *2*, 119–137. doi:10.1177/2167702613497473

Chorpita, B. F., Yim, L., Moffitt, C., Umemoto, L. A., & Francis, S. E. (2000). Assessment of symptoms of DSM-IV anxiety and depression in children: A Revised Child Anxiety and Depression Scale. *Behaviour Research and Therapy*, *38*, 835–855.

Cramer, A. O., Waldorp, L. J., van der Maas, H. L. J., & Borsboom, D. (2010). Comorbidity: A network perspective. *Behavioral and Brain Sciences*, *33*, 137–150.

de Winter, A. F., Oldehinkel, A. J., Veenstra, R., Brunnekreef, J. A., Verhulst, F. C., & Ormel, J. (2005). Evaluation of non-response bias in mental health determinants and outcomes in a large sample of pre-adolescents. *European Journal of Epidemiology*, *20*, 173–181. doi:10.1007/s10654-004-4948-6

Forbush, K. T., & Watson, D. (2013). The structure of common and uncommon mental disorders. *Psychological Medicine*, *43*, 97–108. doi:10.1017/S0033291712001092

Gibbons, R. D., & Hedeker, D. R. (1992). Full-information item bi-factor analysis. *Psychometrika*, *57*, 423–436.

Hankin, B. L., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., & Angell, K. E. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, *107*, 128–140. doi:10.1037/0021-843X.107.1.128

Keller, M. C., & Miller, G. (2006). Resolving the paradox of common, harmful, heritable mental disorders: Which evolutionary genetic models work best? *Behavioral and Brain Sciences*, *29*, 385–404.

Kendler, K. S., Prescott, C. A., Myers, J. M., & 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. doi:10.1001/archpsyc.60.9.929

Kotov, R., Gamez, W., Schmidt, F., & Watson, D. (2010). Linking "big" personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin*, *136*, 768–821. doi:10.1037/a0020327

Krueger, R. F., Caspi, A., Moffitt, T. E., & Silva, P. A. (1998). The structure and stability of common mental disorders (*DSM-III-R*): A longitudinal-epidemiological study. *Journal of Abnormal Psychology*, *107*, 216–227. doi:10.1037/0021-843X.107.2.216

Krueger, R. F., Johnson, W., & Kling, K. C. (2006). Behavior genetics and personality development. In D. Mroczek & T. Little (Eds.), *Handbook of personality development* (pp. 81–108). Mahwah, NJ: Erlbaum.

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. doi:10.1037/a0028355

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.

Martel, M. M. (2013). Sexual selection and sex differences in the prevalence of childhood externalizing and adolescent internalizing disorders. *Psychological Bulletin*, *139*, 1221–1259.

Mathyssek, C. M., Olino, T. M., Hartman, C. A., Ormel, J., Verhulst, F. C., & Van Oort, F. V. A. (2013). Does the Revised Child Anxiety and Depression Scale (RCADS) measure anxiety symptoms consistently across adolescence? The TRAILS study. *International Journal of Methods in Psychiatric Research*, *22*, 27–35. doi:10.1002/mpr.1380

Moffitt, T. E., Arseneault, L., Belsky, D., Dickson, N., Hancox, R. J., Harrington, H., . . . Caspi, A. (2011). A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences USA*, *108*, 2693–2698. doi:10.1073/pnas.1010076108

Muthén, L. K., & Muthén, B. O. (2007). *Mplus: Statistical analysis with latent variables: User's guide*. Los Angeles, CA: Muthén & Muthén.

Neeleman, J., Bijl, R., & Ormel, J. (2004). Neuroticism, a central link between somatic and psychiatric morbidity: Path analysis of prospective data. *Psychological Medicine*, *34*, 521–531. doi:http://dx.doi.org.proxy-ub.rug.nl/10.1017/S0033291703001193

Neeleman, J., Ormel, J., & Bijl, R. V. (2001). The distribution of psychiatric and somatic III health: Associations with personality and socioeconomic status. *Psychosomatic Medicine*, *63*, 239–247.

Oldehinkel, A. J., Hartman, C. H., de Winter, A. F., Veenstra, R., & Ormel, J. (2004). Temperament profiles associated with internalizing and externalizing problems in preadolescence. *Development and Psychopathology*, *16*, 421–440. doi:10.1017S0954579404044591

Ormel, J., Oldehinkel, A. J., Sijtsema, J., van Oort, F., Raven, D., Veenstra, R., . . . Verhulst, F. C. (2012). The TRacking Adolescents' Individual Lives Survey (TRAILS): Design, current status, and selected findings. *Journal of the American Academy of Child and Adolescent Psychiatry*, *51*, 1020–1036. doi:10.1016/j.jaac.2012.08.004

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. doi:10.1017/S0033291706008452

Smoller, J. (2013). Cross disorder group of the psychiatric genomics consortium. Identification of risk loci with shared effects on five major psychiatric disorders: A genome-wide analysis. *Lancet*, *381*, 1360–1360.

van den Akker, M., Buntinx, F., Metsemakers, J. F., Roos, S., & Knottnerus, J. A. (1998). Multimorbidity in general practice: Prevalence, incidence, and determinants of co-occurring chronic and recurrent diseases. *Journal of Clinical Epidemiology*, *51*, 367–375.

Verhulst, F. C., & Achenbach, T. M. (1995). Empirically based assessment and taxonomy of psychopathology:

Cross-cultural applications. A review. *European Child and Adolescent Psychiatry*, *4*, 61–76.

Vollebergh, W. A. M., Iedema, J., Bijl, R. V., de Graaf, R., Smit, F., & Ormel, J. (2001). The structure and stability of common mental disorders: The NEMESIS study. *Archives of General Psychiatry*, *58*, 597–603. doi:10.1001/archpsyc.58.6.597

Vollebergh, W. A. M., ten Have, M., Decovic, M., Oosterwegel, A., Pels, T., Veenstra, R., . . . Verhulst, F. (2005). Mental health in immigrant children in the Netherlands. *Social Psychiatry and Psychiatric Epidemiology*, *40*, 489–496.

Wigman, J. T., Vollebergh, W. A. M., Raaijmakers, Q. A., Iedema, J., van Dorsselaer, S., Ormel, J., . . . van Os, J. (2011). The structure of the extended psychosis phenotype in early adolescence—A cross-sample replication. *Schizophrenia Bulletin*, *37*, 850–860.

Wing, J. K., Mann, S. A., Leff, J. P., & Nixon, J. M. (1978). The concept of a case in psychiatric population surveys. *Psychological Medicine*, *8*, 203–217.