

The structure of adolescent psychopathology: a symptom-level analysis

N. Carragher^{1,2*}, M. Teesson¹, M. Sunderland¹, N. C. Newton¹, R. F. Krueger³, P. J. Conrod⁴,
E. L. Barrett⁵, K. E. Champion⁵, N. K. Nair⁵ and T. Slade⁵

¹National Drug and Alcohol Research Centre, University of New South Wales, Sydney, Australia

²Office of Medical Education, University of New South Wales, Sydney, Australia

³Department of Psychology, University of Minnesota, MN, USA

⁴Department of Psychiatry, Université de Montréal, Montréal, Canada

⁵National Drug and Alcohol Research Centre, University of New South Wales, Sydney, Australia

Background. Most empirical studies into the covariance structure of psychopathology have been confined to adults. This work is not developmentally informed as the meaning, age-of-onset, persistence and expression of disorders differ across the lifespan. This study investigates the underlying structure of adolescent psychopathology and associations between the psychopathological dimensions and sex and personality risk profiles for substance misuse and mental health problems.

Method. This study analyzed data from 2175 adolescents aged 13.3 years. Five dimensional models were tested using confirmatory factor analysis and the external validity was examined using a multiple-indicators multiple-causes model.

Results. A modified bifactor model, with three correlated specific factors (internalizing, externalizing, thought disorder) and one general psychopathology factor, provided the best fit to the data. Females reported higher mean levels of internalizing, and males reported higher mean levels of externalizing. No significant sex differences emerged in liability to thought disorder or general psychopathology. Liability to internalizing, externalizing, thought disorder and general psychopathology was characterized by a number of differences in personality profiles.

Conclusions. This study is the first to identify a bifactor model including a specific thought disorder factor. The findings highlight the utility of transdiagnostic treatment approaches and the importance of restructuring psychopathology in an empirically based manner.

Received 21 June 2015; Revised 19 October 2015; Accepted 26 October 2015; First published online 1 December 2015

Key words: Adolescents, externalizing, general psychopathology, internalizing, meta-structure, thought disorder.

Introduction

Over the past decade, an extensive literature has amassed elucidating the natural classification of mental disorders. This research, examining the underlying covariance (or correlational) structure of psychopathology, focuses on understanding inter-individual differences and the latent variables underlying these differences. A seminal paper by Krueger *et al.* (1998) – partly inspired by Achenbach & Edelbrock (1984) – suggested that relationships between disorders reflect two underlying dimensions – internalizing and externalizing – that are subject to genetic and environmental influences. This grouping of mental disorders, or meta-structure, has received robust support from

community and clinical samples worldwide, demonstrating invariance across cultures (Krueger *et al.* 1998, 2003; Vollebergh *et al.* 2001; Slade & Watson, 2006; Røysamb *et al.* 2011), gender (Eaton *et al.* 2012), ethnicity (Eaton *et al.* 2013), age (Eaton *et al.* 2011), and time (Krueger *et al.* 1998; Vollebergh *et al.* 2001; Measelle *et al.* 2006; Eaton *et al.* 2011). However, important gaps remain.

First, although the terms ‘internalizing’ and ‘externalizing’ originate from the youth literature (Achenbach & Edelbrock, 1984) much of the research into covariance structure has been confined to adults, with few studies comparing alternative models of adolescent psychopathology. Wittchen *et al.* (2009) suggest that different underlying structures of co-morbidity may manifest at different developmental stages. For example, anxiety disorders typically present during childhood whereas depressive and substance use disorders emerge during adolescence or adulthood (Kessler *et al.* 2005; Beesdo-Baum *et al.* 2009).

* Address for correspondence: Dr N. Carragher, National Drug and Alcohol Research Centre, University of New South Wales, NSW 2052, Australia.
(Email: n.carragher@unsw.edu.au)

Second, research into covariance structure has been largely confined to common anxiety and depressive disorders. To capture the meta-structure of psychopathology and facilitate the emergence of nuanced factor structures and novel dimensions, it is necessary to take into account additional and severe types of mental illness, including psychotic symptoms. Psychotic symptoms are rarely included in research into covariance structure, partly due to their omission from most large-scale psychiatric surveys. Methodological issues notwithstanding, the absence of psychosis from the meta-structure is noteworthy due to the higher prevalence of psychotic symptoms in the community than previously assumed, high rates of comorbidity and economic burden (Caspi *et al.* 2014).

Third, research into covariance structure has largely used a diagnostic-level approach. Symptom-level analyses are warranted to unpack disorders and better capture the underlying structure of psychopathology (Markon, 2010). It is well established that disorders in our leading psychiatric nosologies are based on arbitrary thresholds (Carragher *et al.* 2015) with considerable diagnostic overlap. This underscores the importance of building models of psychopathology structure from the bottom up. Finally, research into covariance structure has provided evidence of an overarching, general psychopathology factor (p factor) (Lahey *et al.* 2012, 2015; Tackett *et al.* 2013; Blanco *et al.* 2015; Noordhof *et al.* 2015). Similar to the g factor of general intelligence, the p factor reflects an underlying liability to experience all forms of psychopathology and emerged from observations that mental disorders and the underlying spectra are positively and substantially correlated. While a bifactor model (general factor and specific factors) has a long history in psychometrics and proven useful in intelligence and personality research, it has only recently been applied to psychopathology. Further research in different settings is warranted to establish whether the p factor observed in adult samples is present in adolescent samples.

These gaps have given rise to a nascent, developmental literature, most notably with the work of Caspi *et al.* (2014). These authors conducted confirmatory factor analyses (CFA) among adults at ages 18, 21, 26, 32, and 38 years. They found evidence for a bifactor model, comprising a general psychopathology factor and internalizing and externalizing specific factors. In this framework, individual symptoms are organized into distinct diagnoses. These diagnoses can be interpreted as representing a normally distributed general psychopathology dimension. Additionally, the residual variance of specific groups of diagnoses can be explained by internalizing and externalizing dimensions. The authors failed to find support for a distinct thought disorder factor. This may be due, partly, to a

small number of thought disorder indicators, reducing capacity to adequately model this dimension, and a diagnostic-level approach. As Krueger & Markon (2011) note, symptom-level analysis can help 'unpack' low-prevalence conditions and facilitate the emergence of new dimensions.

Extending Caspi *et al.*'s (2014) research, Laceulle *et al.* (in press) found support for general psychopathology, internalizing and externalizing factors using data from Dutch adolescents aged 11, 13, 16 and 19 years and their parents. They also failed to find a thought disorder factor, perhaps due to restricting psychotic assessment to one time-point. Further, utilizing a symptom-based approach and data from 23 477 adolescents at age 11 and 10 270 adolescents at 13.5 years, Patalay *et al.* (2015) identified general psychopathology, internalizing and externalizing dimensions. They did not measure psychotic symptoms and so were unable to model a thought disorder factor.

Validation of the meta-structure

There is a relative paucity of research examining the external validity of the meta-structure, particularly in adolescents. Information about how factors are differentially related to external correlates could help identify important treatment targets. Caspi *et al.* (2014) highlighted the importance of gender, finding evidence that liability to general psychopathology, externalizing and internalizing are highly gendered styles in adults. Relatedly, personality has been associated with mental health and substance use disorders (Krank *et al.* 2011; Links & Eynan, 2013). Accordingly, we examined the influence of sex and personality risk traits for adolescent substance use and mental health problems on the structure of adolescent psychopathology. To our knowledge, research into covariance structure has not examined these correlates among adolescents. External validity was examined using structural equation modeling (SEM), overcoming limitations associated with Patalay *et al.* (2015) who utilized factor scores. Factor scores are numerical values which indicate an individual's relative location on a latent factor. They do not have the same distribution as true factors nor do they share the same relationships to other variables (Muthén, 2007).

The current study

Based on the above gaps and recent findings warranting further exploration, this study aimed to inform research on the structure of adolescent psychopathology. Our first goal was to evaluate the existence of internalizing, externalizing, thought disorder, and general psychopathology dimensions in a large sample of Australian adolescents. This involved testing alternative dimensional models using symptoms. Our second

goal involved assessing the external validity of the best-fitting model. This is the first study of its kind in Australia and overcomes limitations with previous studies. Our study focuses on early adolescence, when many mental disorders emerge (Merikangas *et al.* 2010).

Method

Participants and procedure

This study is part of a larger project, the CAP study, a cluster randomized control trial to prevent substance misuse and related harms in adolescents. The trial was conducted in 26[†] secondary schools in Sydney and Melbourne, Australia. The research protocol (see Newton *et al.* 2012) and informed consent procedures were approved by the University of New South Wales Human Ethics Committee, Sydney Catholic Education Office, and New South Wales Department of Education and Communities. The trial is registered with the Australian and New Zealand Clinical Trials Registry (ACTRN 12612000026820). This study utilizes baseline data. Of the 2190 students who participated at baseline, 15 had missing data across all variables of interest. Accordingly, this study focuses on 2175 students [males = 57.4%, mean age = 13.3 (s.e. = 0.48) years].

Measures

The measures used to assess the past 6 months internalizing, externalizing and psychotic symptoms are described below and an item summary is provided in Supplementary Table S1. Our community-based sample of adolescents displayed a fairly limited range of severity. To reduce the number of sparse cells, improve statistical power and yield stable estimates, all Likert-type items were recoded into dichotomous variables².

As described below, internalizing and externalizing symptoms were partly assessed using the Strengths and Difficulties Questionnaire (SDQ; Goodman, 2001). The SDQ is a brief, 25-item questionnaire which assesses emotional and behavioral problems in children and adolescents. Selection of items to load onto the internalizing and externalizing dimensions was informed by recent analyses by the instrument's authors (Goodman *et al.* 2010). Based on documented problems with the reverse-coded SDQ items (van de Looij-Jansen *et al.* 2011), we removed these items.

Internalizing symptoms

Internalizing was assessed using eight items from the SDQ emotional and peer scales, and 12 items from the depression and anxiety scales of the Brief Symptom Inventory (BSI; Derogatis, 1993). The SDQ items were rated on a three-point scale (0 = not true, 1 = sometimes true, 2 = certainly true). The SDQ is one of the most commonly used instruments for screening psychopathology in children and adolescents. It has been widely validated in clinical practice, community and epidemiological settings across different countries (see Giannakopoulos *et al.* 2013). The BSI is a widely used, 53-item psychological distress scale, comprising nine symptom scales. It was rated on a five-point scale (0 = not at all, 1 = a little bit, 2 = moderately, 3 = quite a bit, 4 = often) and has high test-retest reliability and high validity (Derogatis & Melisaratos, 1983).

Externalizing symptoms

Externalizing was assessed using seven items from the SDQ behavioral and hyperactivity scales. Additionally, we used eight alcohol-related harm items from an abbreviated version of the Rutgers Alcohol Problem Index (RAPI; White & Labouvie, 1989) based on the most frequently endorsed items by adolescents in previous work (Conrod *et al.* 2008). Items were rated on a five-point scale (0 = never, 1 = 1–2 times, 2 = 3–4 times, 3 = 5–6 times, 4 = more than 6 times). The RAPI is one of the most commonly used measures of alcohol problems and demonstrates good psychometric properties (Neal *et al.* 2006).

Psychotic symptoms

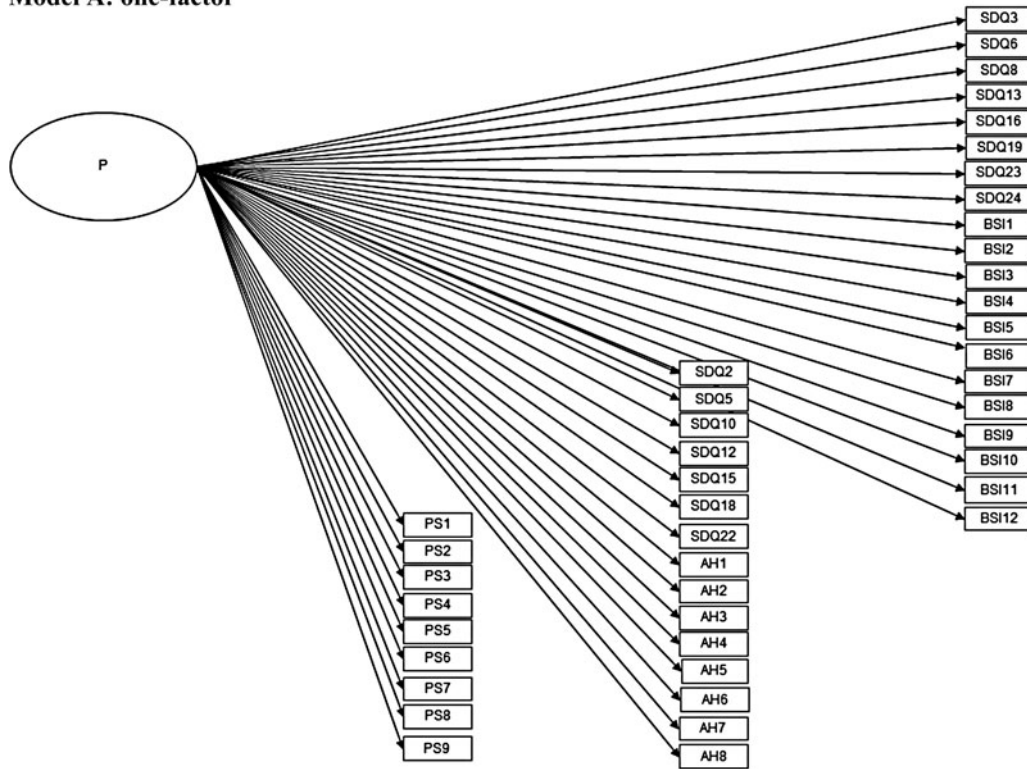
Nine questions assessed hallucinatory experiences and delusional beliefs (Laurens *et al.* 2012) – including five items adapted from the psychosis section of the Diagnostic Interview Schedule for Children (Costello *et al.* 1982) – using a three-point scale (0 = not true, 1 = sometimes true, 2 = certainly true). This measure has been validated using item response theory (Laurens *et al.* 2012).

Statistical analysis

Analyses involved two steps. First, alternative dimensional models tested by Caspi *et al.* (2014), and replicated more recently by others, were estimated using CFA (see Fig. 1). *Model A* estimates a one-factor model whereby all symptoms are subsumed under a general psychopathology factor (Caspi *et al.* 2014; Del Giudice, *in press*; Laceulle *et al.* *in press*). *Model B* comprises three correlated factors (internalizing, externalizing, thought disorder) (Caspi *et al.* 2014; Del Giudice, *in press*; Laceulle *et al.* *in press*). The internalizing

[†] The notes appear after the main text.

Model A: one-factor



Model B: Correlated three-factors

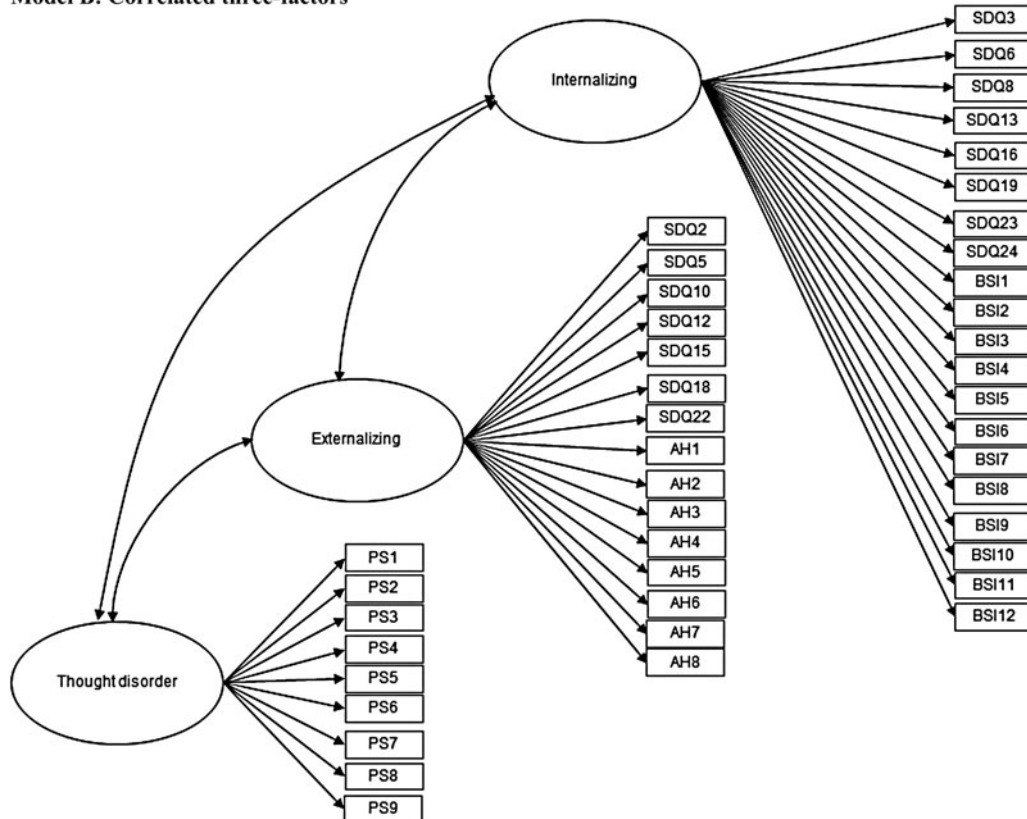
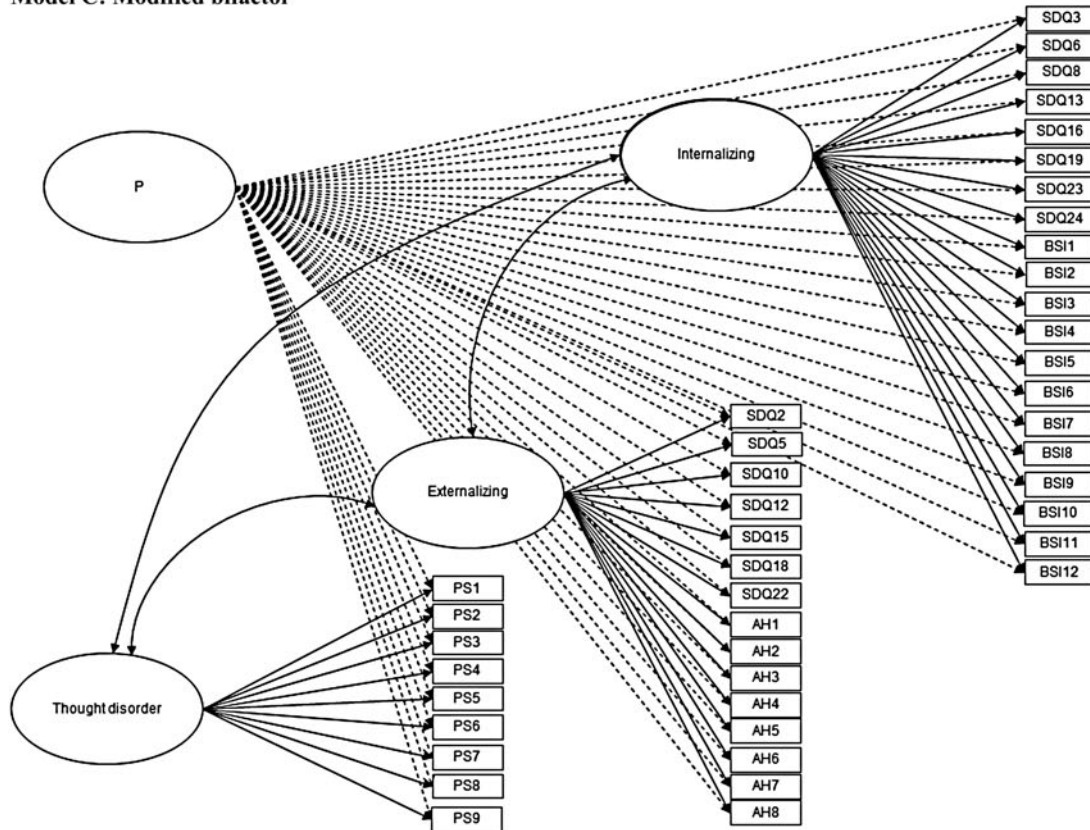


Fig. 1. Model A (one-factor) and model B (correlated three-factors). For legend see model E.

Model C: Modified bifactor



Model D: Classic bifactor

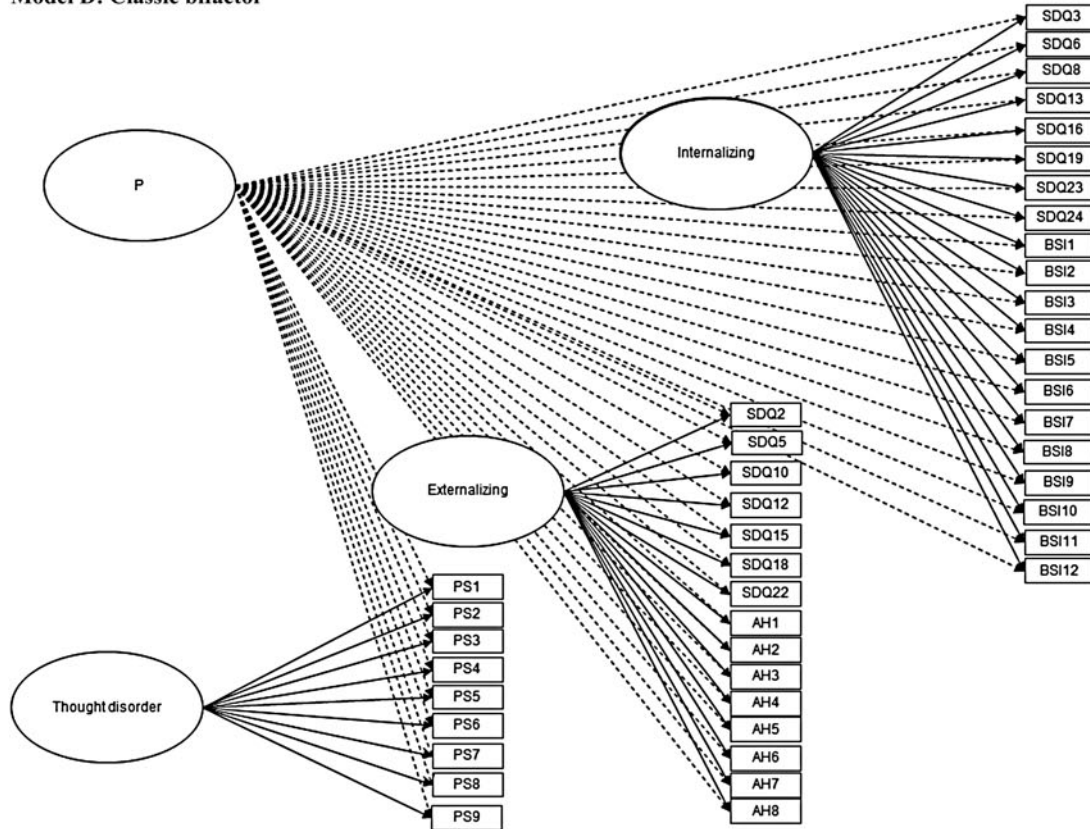


Fig. 1. Model C (modified bifactor) and model D (classic bifactor). For legend see model E.

Model E: Higher-order

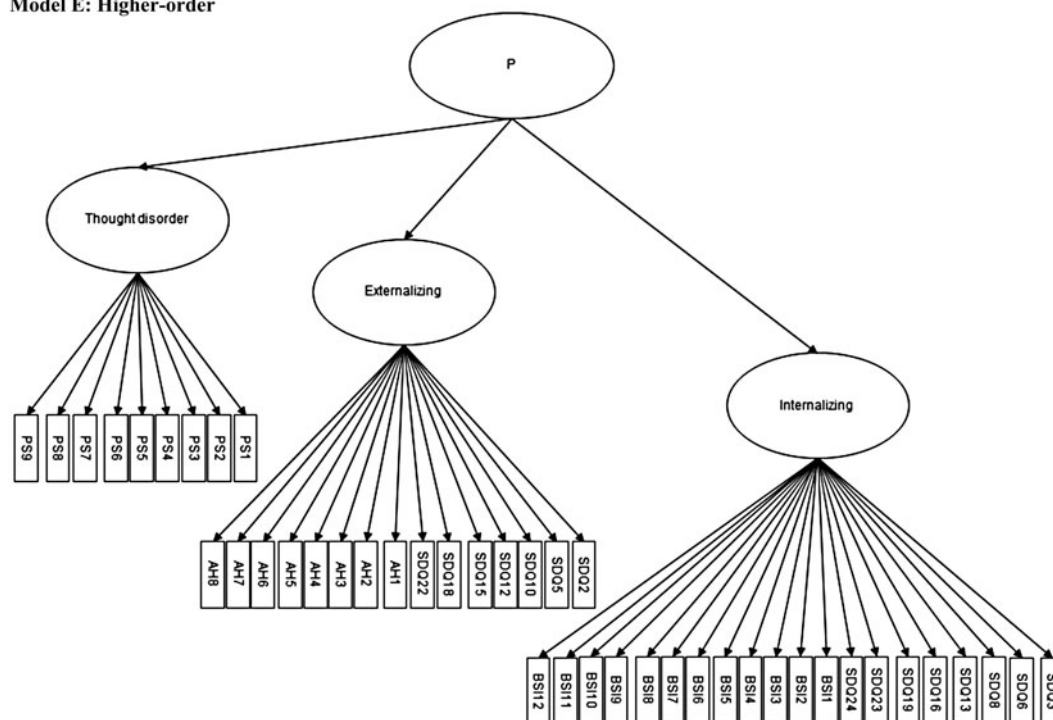


Fig. 1. Model E (higher-order). Alternative models of the structure of adolescent psychopathology. For clarity, dashed lines highlight paths from the general factor to individual symptoms (models C and D). PS, Psychotic symptoms derived from Laurens *et al.* (2012); SDQ, symptom items from the Strengths and Difficulties Questionnaire; AH, alcohol harm items derived from the Rutgers Alcohol Problems Index; BSI, Symptom items from the Brief Symptom Inventory; P, General psychopathology.

and externalizing factors have received robust support (Carragher *et al.* 2015). The addition of a thought disorder dimension reflects a recent observation (Markon, 2010; Kotov *et al.* 2011a,b; Laurens *et al.* 2012; Keyes *et al.* 2013; Wright *et al.* 2013; Fleming *et al.* 2014). *Model C* tests a modified bifactor model (Lahey *et al.* 2012, 2015; Tackett *et al.* 2013; Caspi *et al.* 2014; Noordhof *et al.* 2015; Laccelle *et al.* in press; Patalay *et al.* 2015) with three correlated factors (internalizing, externalizing, thought disorder) and a general psychopathology factor. Extending Caspi *et al.* (2014), *model D* tests a classic bifactor model with three uncorrelated factors (internalizing, externalizing, thought disorder) and a general psychopathology factor. The general factor explains the correlation between all symptoms; the specific factors accounts for residual covariance among a subset of symptoms. The bifactor models differ from a simple structure first-order model with multiple factors [multiple common factors that are (un)correlated] or a second-order factor model (a first-order factor correlated with a higher-order factor). *Model E* estimates a higher-order model in which one overarching general factor explains three primary factors (internalizing, externalizing, thought disorder).

Second, we examined sex and personality risk factors for substance use and psychopathology in differences in psychopathological liabilities in the best-fitting model. Sex differences were evaluated using a multiple-indicators multiple-causes (MIMIC) model. Personality risk profiles were measured by the Substance Use Risk Profile Scale (SURPS; Woicik *et al.* 2009). The SURPS measures personality risk for substance use and other behavioral problems according to four traits: anxiety sensitivity, negative thinking, impulsivity, and sensation seeking. Each profile was assessed using 4–7 items rated on a four-point scale (1 = strongly disagree, 2 = disagree, 3 = agree, 4 = strongly agree). Using CFA, differences in personality were investigated by simultaneously modeling the four SURPS dimensions and corresponding items (see Supplementary Table S2 for a description of the SURPS items and dimensions) with the best-fitting model.

Assessment of model fit

All models were estimated in Mplus v. 7.3, using robust weighted least squares (WLSMV) estimation, which is appropriate for dichotomous data. Analyses were adjusted for school-level clustering. Model fit

Table 1. Fit indices for alternative dimensional models of the structure of psychopathology in Australian adolescents ($n = 2175$)

Model	No. of parameters	χ^2	df	CFI	TLI	RMSEA (90% CI)	AIC	BIC
Model A: one-factor	88	4384.797	902	0.781	0.770	0.042 (0.041–0.043)	71 900.944	72 401.205
Model B: correlated three-factors	91	2268.103	899	0.914	0.909	0.026 (0.025–0.028)	66 776.021	67 293.336
Model C: modified bifactor	135	1245.193	855	0.975	0.973	0.014 (0.013–0.016)	64 741.963	65 509.409
Model D: classic bifactor	132	1344.166	858	0.969	0.966	0.016 (0.014–0.018)	64 796.160	65 546.552
Model E: higher-order	91	2268.103	899	0.914	0.909	0.026 (0.025–0.028)	66 711.672	67 228.988

χ^2 , Chi-square statistic; df, degree of freedom; CFI, comparative fit index; TLI, Tucker–Lewis index; RMSEA, root mean square error of approximation; CI, confidence interval; AIC, Akaike’s Information Criterion; BIC, Bayesian Information Criterion.

Modified bifactor (model C) = specific factors are correlated. Classic bifactor (model D) specific factors are uncorrelated. Weighted least square means and variance adjusted (WLSMV) estimation was used to generate the number of parameters, χ^2 (df), CFI, TLI and RMSEA (90% CI). Robust maximum likelihood estimation (MLR) was used to generate the BIC and AIC. The best-fitting model is in boldface.

was evaluated using the Root Mean Square Error of Approximation (RMSEA), Comparative Fit Index (CFI), and Tucker–Lewis Index (TLI). RMSEA values <0.05 indicate close model fit (Brown, 2006). TLI and CFI values ≥ 0.90 indicate acceptable fit and values ≥ 0.95 imply very good fit (Brown, 2006). Models were re-run with robust maximum likelihood estimation (MLR) to generate the Bayesian Information Criterion (BIC) and Akaike’s Information Criterion (AIC). Lower AIC and BIC values are preferred. A 6- to 10-point BIC difference between models indicates strong support for the model with the lower BIC value and a difference >10 yields very strong support (Raftery, 1995). A single latent factor is supported by salient loadings ≥ 0.40 (Brown, 2006). Multicollinearity – indicating a lack of discriminant validity between two factors – is evidenced by factor intercorrelations ≥ 0.80 (Brown, 2006).

Results

The structure of adolescent psychopathology

Goodness-of-fit indices are presented in Table 1, factor loadings and factor intercorrelations are presented in Table 2. Model A, a one-factor model, did not fit the data well (CFI and TLI <0.90). Model B, a correlated three-factor model, provided a good fit to the data [CFI = 0.914, TLI = 0.909, RMSEA = 0.026 (90% CI 0.025–0.028)]. All factor loadings were positive, salient and significant ($p < 0.001$). The average factor loading for internalizing was 0.723, 0.714 for externalizing, and 0.794 for thought disorder. The factor intercorrelations were all significant ($p < 0.001$) and moderate in size (range 0.449–0.512), ruling out multicollinearity. Model C, a modified bifactor model, provided an

excellent fit to the data [CFI = 0.975, TLI = 0.973, RMSEA = 0.014 (90% CI 0.013–0.016)]. The average factor loading for internalizing was 0.477, 0.473 for externalizing, 0.667 for thought disorder, and 0.480 for general psychopathology. The factor intercorrelations were all significant ($p < 0.05$) and moderate in size (range 0.087–0.287), ruling out multicollinearity. Model D, a classic bifactor model, provided excellent fit to the data, albeit the fit indices deteriorated marginally compared to model C [CFI = 0.969, TLI = 0.966, RMSEA = 0.016 (90% CI 0.014–0.018)]. The average factor loading for internalizing was 0.473, 0.410 for externalizing, 0.573 for thought disorder, and 0.528 for general psychopathology. No factor intercorrelations were specified for this model. Finally, model E, a higher-order model provided good fit to the data [CFI = 0.914, TLI = 0.909, RMSEA = 0.026 (90% CI 0.025–0.028)]. The average factor loading for internalizing was 0.723, 0.714 for externalizing and 0.794 for thought disorder. Factor intercorrelations were all significant ($p < 0.05$) and moderate in size (range 0.644–0.735). The BIC for model C displayed a >10 -point difference with other models, indicating it was the best-fitting model.

In the bifactor models (models C and D), a number of items loaded primarily on the general factor; others loaded primarily on a specific factor, indicating variability not captured by the general factor; and several transdiagnostic items loaded substantially on the general and specific factors. These patterns are consistent with the literature. However, theoretically, it could be argued that to be meaningful all items should be salient (≥ 0.40) on the general factor. To investigate whether alternative models yield a better fit, and extending the work of Caspi *et al.* (2014), we conducted exploratory factor analysis (EFA) models specifying 1–

Table 2. Standardized factor loadings and factor intercorrelations for Models A–E

Symptom	Model A			Model B			Model C			Model D			Model E		
	P	INT	EXT	TD	INT	EXT	TD	P	INT	EXT	TD	P	INT	EXT	TD
Standardized factor loading															
Restless ^a	0.496		0.643			0.020		0.669		−0.069		0.635		0.643	
Somatic symptoms ^a	0.515	0.559			0.178			0.568	0.147			0.558	0.559		
Tempers ^a	0.545		0.700			0.165		0.659		0.094		0.641		0.700	
Solidarity ^a	0.444	0.493			0.043			0.605	0.068			0.549	0.493		
Worries ^a	0.583	0.662			0.420			0.506	0.436			0.494	0.662		
Fidgety ^a	0.520		0.596			0.015		0.725		−0.071		0.685		0.596	
Fights or bullies ^a	0.475		0.631			0.219		0.552		0.156		0.554		0.631	
Unhappy ^a	0.717	0.805			0.379			0.738	0.385			0.709	0.805		
Easily distracted ^a	0.524		0.660			0.083		0.660		0.017		0.627		0.660	
Nervous in new situations ^a	0.490	0.525			0.252			0.482	0.262			0.460	0.525		
Lies or cheats ^a	0.519		0.629			0.247		0.619		0.167		0.620		0.629	
Picked on/bullied ^a	0.514	0.595			−0.048			0.711	−0.027			0.650	0.595		
Steals ^a	0.539		0.626			0.280		0.603		0.203		0.611		0.626	
Better with adults ^a	0.366	0.404			−0.027			0.504	−0.025			0.467	0.404		
Many fears ^a	0.505	0.577			0.231			0.538	0.240			0.511	0.577		
Ending life ^b	0.722	0.786			0.526			0.578	0.470			0.618	0.786		
Lonely ^b	0.772	0.823			0.733			0.430	0.705			0.480	0.823		
Sad ^b	0.769	0.834			0.860			0.319	0.823			0.395	0.834		
No interest ^b	0.684	0.752			0.598			0.473	0.544			0.525	0.752		
Hopeless about the future ^b	0.801	0.861			0.669			0.550	0.628			0.593	0.861		

Worthlessness ^b	0.865	0.926	0.713	0.600	0.667		0.648	0.926	
Feelings easily hurt ^b	0.782	0.807	0.742	0.390	0.719		0.438	0.807	
Feeling tense ^b	0.716	0.761	0.678	0.399	0.648		0.445	0.761	
Scared for no reason ^b	0.734	0.811	0.612	0.548	0.570		0.585	0.811	
Tearful ^b	0.733	0.781	0.733	0.368	0.712		0.416	0.781	
Nervous of shaky ^b	0.751	0.815	0.643	0.515	0.603		0.558	0.815	
Terror or panic ^b	0.801	0.873	0.605	0.628	0.553		0.667	0.873	
Fights ^c	0.683	0.832		0.928	0.110	0.906	0.243		0.832
Shame/embarrassment ^c	0.722	0.854		0.907	0.158	0.880	0.279		0.854
Neglected responsibilities ^c	0.805	0.882		0.918	0.148	0.885	0.287		0.882
Personality change ^c	0.654	0.821		0.918	0.175	0.888	0.298		0.821
Memory loss ^c	0.660	0.754		0.726	0.377	0.640	0.495		0.754
Tolerance ^c	0.528	0.702		0.553	0.451	0.491	0.506		0.702
Attempt to cut down ^c	0.476	0.580		0.505	0.353	0.435	0.423		0.580
Crazy ^c	0.637	0.807		0.616	0.544	0.530	0.612		0.807
Thoughts read ^d	0.477		0.671		0.559	0.373	0.476	0.477	0.671
Special messages ^d	0.542		0.795		0.725	0.398	0.629	0.531	0.795
Spied upon ^d	0.603		0.832		0.566	0.527	0.416	0.629	0.832
Heard voices ^d	0.656		0.881		0.711	0.497	0.601	0.618	0.881
Controlled ^d	0.613		0.869		0.756	0.465	0.670	0.597	0.869
Reads minds ^d	0.473		0.691		0.618	0.349	0.544	0.458	0.691
Body changed ^d	0.603		0.819		0.639	0.477	0.528	0.590	0.819
Special power ^d	0.529		0.718		0.692	0.320	0.656	0.424	0.718
Visual hallucination ^d	0.631		0.874		0.733	0.482	0.638	0.608	0.874
Factor correlations									
Internalizing		0.449	0.512	0.087	0.224	–	–	0.697	
Externalizing			0.474		0.287	–	–		0.644
General psychopathology									0.735

INT, Internalizing; EXT, externalizing; TD, thought disorder; P, general psychopathology.

^a Strengths and Difficulties Questionnaire items.

^b Brief Symptom Inventory items.

^c Alcohol harm items derived from the Rutgers Alcohol Problems Index.

^d Psychotic symptoms derived from Laurens *et al.* (2012). Factor loadings and correlations with a p value ≤ 0.05 are displayed in boldface. There are no factor correlations to estimate in the one-factor model (model A) or in the classic bifactor model (model D). Larger positive values reflect a stronger association of a symptom with that dimension; negative values indicate an inverse association with the dimension.

Table 3. Standardized regression coefficients for the effects of four personality risk profiles on internalizing, externalizing, thought disorder and general psychopathology liabilities

SURPS personality profiles	Modified bifactor (model C)			
	Internalizing	Externalizing	Thought disorder	General psychopathology
Negative thinking	0.376	0.001	0.062	0.575
Anxiety sensitivity	0.362	−0.007	0.046	0.155
Impulsivity	−0.022	0.273	0.109	0.598
Sensation seeking	− 0.147	0.303	0.114	0.093

SURPS, Substance Use Risk Profile Scale. Estimates with p value ≤ 0.001 are displayed in boldface.

4 factors. None of these models fit better than the modified bifactor model (BIC range for EFA models = 65638–72539; BIC value of model C = 65509). In summary, after testing a series of EFA and CFA models, a modified bifactor model provided a meaningful alternative to simple factor models, enabling simultaneous estimation of general and specific factors.

Sex differences in psychopathological liabilities

The latent factors in model C were subsequently regressed on sex (i.e. indirect effects only model) using MIMIC modelling. The modification indices did not support the inclusion of direct paths between individual symptoms and sex, providing evidence of gender invariance and permitting direct comparisons of factor means. This model provided an excellent fit to the data [CFI = 0.977, TLI = 0.975, RMSEA = 0.014 (90% CI 0.012–0.015)]. Females reported higher mean levels of latent internalizing than males ($\beta = 0.307$, $p \leq 0.001$), and males reported higher mean levels of externalizing than females ($\beta = -0.139$, $p \leq 0.01$). No significant sex differences were observed in thought disorder or general psychopathology.

Personality risk profile differences in psychopathological liabilities

To examine differences in personality risk profiles for substance misuse and mental health problems, we estimated the latent structure of the SURPS simultaneously with model C using CFA. This model provided excellent fit to the data [CFI = 0.965, TLI = 0.962, RMSEA = 0.011 (90% CI 0.010–0.013)]. As Table 3 indicates, general psychopathology was characterized by high negative thinking, high impulsivity, high anxiety sensitivity and high sensation seeking. Adolescents who scored high on internalizing – over and above general psychopathology – were characterized by hopelessness, a risk factor for the development of depression

(*high negative thinking*); a fear of anxiety-related sensations, driven by beliefs that such sensations have harmful consequences (*high anxiety sensitivity*); and, minimal desire for stimulation and novel experiences (*low sensation seeking*). Adolescents who scored high on externalizing – over and above general psychopathology – were characterized by difficulties in the regulation of behavioral responses (*high impulsivity*) and a desire for stimulation and novel experiences (*high sensation seeking*). Adolescents who scored high on thought disorder – over and above general psychopathology – were characterized, to a lesser extent, by difficulties in the regulation of behavioral responses (*high impulsivity*) and a desire for stimulation and novel experiences (*high sensation seeking*).

Discussion

This study contributes to a nascent literature examining the underlying structure of adolescent psychopathology (Caspi et al. 2014; Patalay et al. 2015; Laceulle et al. in press). This study addresses shortcomings associated with previous adolescent research into covariance structure, by using a wide range of psychotic symptoms, SEM to examine external validity, and symptom-level analysis to facilitate identification of new factors. Our results demonstrated that adolescent psychopathology is well described by an internalizing liability to depression and anxiety; an externalizing liability to alcohol misuse; a thought disorder liability to psychotic symptoms; and, a general psychopathology factor, reflecting an underlying vulnerability to develop all forms of psychopathology.

Our findings align with research from New Zealand (Caspi et al. 2014), the UK (Patalay et al. 2015) and The Netherlands (Laceulle et al. in press), identifying a general psychopathology factor and specific internalizing and externalizing factors. The observation of a general vulnerability to psychopathology factor spanning

childhood (Tackett *et al.* 2013; Lahey *et al.* 2015), adolescence (Tackett *et al.* 2013; Blanco *et al.* 2015; Noordhof *et al.* 2015; Laceulle *et al.* *in press*; Patalay *et al.* 2015), and adulthood (Lahey *et al.* 2012; Caspi *et al.* 2014) lends support to a continuity of symptom presentation throughout the life-span. Consistent with the adult literature, the structure of adolescent psychopathology was gender invariant (Eaton *et al.* 2012). Divergent from previous studies, however, we did not encounter problems estimating a specific thought disorder factor. This may have due to a greater number of thought disorder indicators and symptom-level analysis, which conferred greater capacity to model this dimension.

The substantive interpretation of the bifactor model is that the symptoms are correlated because they share a common general psychopathology trait and one independent source of common variation (e.g. a tendency to endorse internalizing symptoms). From an assessment perspective, the mean score on the p factor represents the general tendency to experience psychopathology and the mean score on, for example, the internalizing specific factor represents the specific internalizing tendency, not captured by the general factor. To corroborate the model's psychometric properties and support theoretical arguments regarding a bifactorial structure of adolescent psychopathology, we examined external validity of the specific factors and general factor in relation to gender and personality traits which are important correlates of a range of mental health and substance use disorders. The p factor demonstrated significant associations with all personality facets and the specific factors evidenced differential associations with various facets (see discussion below). In short, psychometric findings herein corroborate the value and the robustness of the bifactor structure of psychopathology and its substantive interpretation. Research, currently underway, will further contribute to our substantive understanding of the bifactor model by investigating whether it is stable from ages 13–17 years (i.e. longitudinal invariance).

Clinical and classification implications

From a clinical perspective, our findings suggest that separate assessment of general risk for psychopathology, and specific risk for internalizing, externalizing and thought disorder is warranted to provide detailed information about a patient's profile. It follows that treatment and/or prevention should be tailored according to general or specific risk profiles. Theoretically, a patient could have a high p score and not receive a diagnosis if they have low scores on the specific factors. This is possible given that the p factor and the primary factors are uncorrelated.

Relatedly, external validation analyses indicated that adolescent psychopathology is differentially associated with personality traits for substance misuse and mental health problems. Since treatments can have differential effects on substance use and mental health symptoms, our findings underscore the importance of prioritizing treatment targets for those with specific risk profiles. In line with available treatment options and practice, our findings reiterate that: (i) adolescents with internalizing symptoms may benefit most from treatments (either medical or psychotherapeutic) targeting negative thinking and anxiety sensitivity; (ii) adolescents with externalizing symptoms may benefit most from treatments targeting impulsivity and sensation seeking; and (iii) adolescents experiencing psychotic symptoms may benefit from treatments targeting impulsivity and sensation seeking. Given the strong links between internalizing, externalizing and psychotic symptoms through a general psychopathology factor, and affective and psychosis symptom overlap (Birchwood, 2003), these findings suggest that adolescents with psychotic symptoms may also benefit from receiving cognitive therapy. Further validation studies, particularly using prospective research, are needed to confirm these relationships.

The observation of a general factor underlying psychopathology aligns with broader findings documenting a general genetic factor underlying neurodevelopmental symptoms (Pettersson *et al.* 2013). In a large representative sample of twins, Lahey *et al.* (2011) found that a general bifactor model of genetic influences, on which the genetic component of every dimension of psychopathology loaded, explained the majority of genetic influences on youth internalizing and externalizing symptoms. Relatedly, Franić *et al.* (2014) identified a common genetic basis for anxiety, depression, and withdrawal, with a different genetic structure for somatic complaints. Consistent with previous findings (e.g. Kendler *et al.* 1987), the study by Franić *et al.* suggests the existence of differing additive genetic, common environmental and individual-unique environmental structures. Our study did not collect data on genetics or a broad range of environmental variables; thus we were unable to examine the presence of different structures and examine genetic influences on the factors in the bifactor model.

The findings have important implications for research on classification. Dialogue around the organization of mental disorders has often been framed in terms of lumping *v.* splitting. Lumpers emphasize similarities amongst categories and favor fewer diagnostic categories, whereas splitters highlight differences between conditions. Support for a bifactor model could be argued to bring the lumpers and splitters together. That is, we found that one general

dimension is needed to summarize a common liability to experience all types of psychopathology and specific factors, indicating some degree of heterogeneity, are warranted to capture individuals who manifest disorders along that single continuum in a distinct way (e.g. some individuals may be more inclined to developing internalizing). Caspi *et al.* (2014) suggest that the general factor may account for difficulties encountered in identifying unique causes, consequences, biomarkers and treatments for individual disorders. Our replication of a general factor underscores the importance and utility of transdiagnostic treatment and comprehensive prevention approaches which efficiently address multiple problems in a single framework.

Limitations and future directions

The results should be considered in light of some limitations. First, whilst our study included a range of mental health symptoms, our assessment of psychopathology was not exhaustive. Notably, youth-onset disorders, such as obsessive-compulsive disorder and autism, were not assessed. Researchers with more comprehensive psychiatric data may provide more detailed insight into the structure of adolescent psychopathology. Due to our restricted range of psychopathology we could not empirically test whether the internalizing spectrum bifurcates into distinct fear and distress sub-dimensions, as observed in some adolescent and adult studies (Kendler *et al.* 2003; Krueger, 1999; Slade & Watson, 2006; Blanco *et al.* 2015). Similarly, we were unable to empirically evaluate whether the externalizing factor bifurcates into specific conduct disorder symptoms and substance misuse. Relatedly, our analysis of the externalizing spectrum focused on alcohol misuse symptoms due to low endorsement of drug misuse items. However, the subsequent waves of this study, which assess students up to age 16/17 years, offer an opportunity to examine substance misuse more broadly. Indeed, given that epidemiological studies indicate that the median age of onset for substance use disorder is 15 years, a longitudinal examination of the structure of psychopathology over time may reveal nuances in factor structure.

Second, this study is not based on a representative sample, therefore caution should be used when extrapolating findings beyond the study population. Third, our community-based sample displayed a limited range of severity; a more clinically severe sample may demonstrate a different factor structure. Fourth, similar to the majority of studies in this area, this study relies on self-report data, therefore it is important to investigate whether method effects impact structure. That is, do studies integrating multi-method and multiple informant (caregivers, teachers) data identify nuanced

developmental differences in psychopathology structure? Limitations notwithstanding, this study has several important strengths, including a large sample size and comparison of a range of dimensional models. It provides the first investigation of the underlying structure of psychopathology in Australian adolescents and adds to a nascent literature examining the existence of novel thought disorder and general psychopathology dimensions.

Conclusions

In closing, the structure of psychopathology is much more than a semantic issue. How symptoms and disorders are classified has implications for assessment, prevention and treatment (Carragher *et al.* 2015). The meta-structure provides a succinct means of organizing disorders according to shared commonalities and supports the development of transdiagnostic treatment approaches, which offer an efficient means of addressing multiple problems in a single framework. Longitudinal invariance of adolescent psychopathology structure represents the next step in this burgeoning field.

Supplementary material

For supplementary material accompanying this paper visit <http://dx.doi.org/10.1017/S0033291715002470>.

Acknowledgements

This research was supported by grant funding (1004744) from the National Health and Medical Research Council, Australia. The authors wish to thank the team of interviewers, data management staff, and study participants.

Declaration of Interest

None.

Notes

- ¹ Initially, 27 schools agreed to participate. Due to time constraints, one school withdrew after randomization but prior to completing baseline questionnaires.
- ² Psychotic symptoms and SDQ items were recoded into 'untrue *v.* true (somewhat true and certainly true)'. BSI items were recoded into 'not at all *v.* all others (a little bit, moderately, quite a bit or often)'. RAPI items were recoded into 'never *v.* all others (1–2 times, 2–3 times, 5–6 times, >6 times)'.

References

- Achenbach TM, Edelbrock CS** (1984). Psychopathology of childhood. *Annual Review of Psychology* **35**, 227–256.
- Beesdo-Baum K, Knappe S, Pine DS** (2009). Anxiety and anxiety disorders in children and adolescents: developmental issues and implications for DSM-V. *Psychiatric Clinics of North America* **32**, 483–524.
- Birchwood M** (2003). Pathways to emotional dysfunction in first-episode psychosis. *British Journal of Psychiatry* **182**, 373–375.
- Blanco C, Wall MW, He JP, Krueger RF, Olfson M, Jin CJ, Burstein M, Merikangas KR** (2015). The space of common psychiatric disorders in adolescents: comorbidity structure and individual latent liabilities. *Journal of the American Academy of Child and Adolescent Psychiatry* **54**, 45–52.
- Brown TA** (2006). *Confirmatory Factor Analysis for Applied Research*. Guilford Press: London.
- Carragher N, Krueger RF, Eaton NR, 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.
- Caspi A, Houts RM, Belsky DW, Goldman-Mellor SJ, Harrington H, Israel S, Meier MH, Ramrakha S, Shalev I, Poulton R, Moffitt TE** (2014). The p factor: one general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science* **2**, 119–137.
- Conrod PJ, Castellanos N, Mackie C** (2008). Personality-targeted interventions delay the growth of adolescent drinking and binge drinking. *Journal of Child Psychology and Psychiatry* **49**, 181–190.
- Costello A, Edelbrock C, Kalas R, Kessler M, Klaric S** (1982). *Diagnostic Interview Schedule for Children: Child Version*. National Institute of Mental Health: Rockville, MD.
- Del Giudice M** (in press). The life history model of psychopathology explains the structure of psychiatric disorders and the emergence of the p factor: a simulation study. *Clinical Psychological Science*.
- Derogatis LR** (1993). *BSI: Administration, Scoring and Procedures Manual for the Brief Symptom Inventory*. National Computer Systems: Minneapolis.
- Derogatis LR, Melisaratos N** (1983). The Brief Symptom Inventory: an introductory report. *Psychological Medicine* **13**, 595–605.
- Eaton NR, Keyes KM, Krueger RF, Balsis S, Skodol AE, Markon KE, Grant BF, Hasin DS** (2012). An invariant dimensional liability model of gender differences in mental disorder prevalence: evidence from a national sample. *Journal of Abnormal Psychology* **121**, 282–288.
- Eaton NR, Keyes KM, Krueger RF, Noordhof A, Skodol AE, Markon KE, Grant BF, Hasin DS** (2013). Ethnicity and psychiatric comorbidity in a national sample: evidence for latent comorbidity factor invariance and connections with disorder prevalence. *Social Psychiatry and Psychiatric Epidemiology* **48**, 701–710.
- Eaton NR, Krueger RF, Oltmanns TF** (2011). Aging and the structure and long-term stability of the internalizing spectrum of personality and psychopathology. *Psychology and Aging* **26**, 987–993.
- Fleming S, Shevlin M, Murphy J, Joseph S** (2014). Psychosis within dimensional and categorical models of mental illness. *Psychosis* **6**, 4–15.
- Franić S, Dolan CV, Borsboom D, van Beijsterveldt CE, Boomsma DI** (2014). Three-and-a-half-factor model? The genetic and environmental structure of the CBCL/6–18 internalizing grouping. *Behavior Genetics* **44**, 254–268.
- Giannakopoulos G, Dimitrakaki C, Papadopoulou K, Tzavara C, Kolaitis G, Ravens-Sieberer U, Tountas Y** (2013). Reliability and validity of the Strengths and Difficulties Questionnaire in Greek adolescents and their parents. *Health* **5**, 1774–1783.
- Goodman A, Lamping DL, Ploubidis GB** (2010). When to use broader internalising and externalising subscales instead of the hypothesised five subscales on the Strengths and Difficulties Questionnaire (SDQ): data from British parents, teachers and children. *Journal of Abnormal Child Psychology* **38**, 1179–1191.
- Goodman R** (2001). Psychometric properties of the strengths and difficulties questionnaire. *Journal of the American Academy of Child and Adolescent Psychiatry* **40**, 1337–1345.
- Kendler KS, Heath AC, Martin NG, Eaves LJ** (1987). Symptoms of anxiety and symptoms of depression. Same genes, different environments? *Archives of General Psychiatry* **44**, 451–457.
- Kendler KS, Prescott CA, Myers J, Neale MC** (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.
- Kessler RC, Chiu WT, Demler O, Merikangas KR, Walters EE** (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry* **62**, 617–627.
- Keyes KM, Eaton NR, Krueger RF, Skodol AE, Wall MW, Grant BF, Siever LJ, Hasin DS** (2013). Thought disorder in the meta-structure of psychopathology. *Psychological Medicine* **43**, 1673–1683.
- Kotov R, Chang SW, Fochtmann LJ, Mojtabai R, Carlson GA, Sedler MJ, Bromet EJ** (2011b). Schizophrenia in the internalizing-externalizing framework: a third dimension? *Schizophrenia Bulletin* **37**, 1168–1178.
- Kotov R, Ruggero CJ, Krueger RF, Watson D, Yuan Q, Zimmerman M** (2011a). New dimensions in the quantitative classification of mental illness. *Archives of General Psychiatry* **68**, 1003–1011.
- Krank M, Stewart SH, O'Connor R, Woicik PB, Wall AM, Conrod PJ** (2011). Structural, concurrent, and predictive validity of the Substance Use Risk Profile Scale in early adolescence. *Addictive Behaviors* **36**, 37–46.
- Krueger RF, Caspi A, Moffitt TE, Silva PA** (1998). The structure and stability of common mental disorders (DSM-III-R): a longitudinal-epidemiological study. *Journal of Abnormal Psychology* **107**, 216–227.
- Krueger RF, Chentsova-Dutton YE, Markon KE, 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.
- Krueger RF** (1999). The structure of common mental disorders. *Archives of General Psychiatry* **56**, 921–926.

- Krueger RF, Markon KE** (2011). A dimensional-spectrum model of psychopathology: progress and opportunities. *Archives of General Psychiatry* **68**, 10–11.
- Laceulle OM, Vollebergh WAM, Ormel J** (in press). The structure of psychopathology in adolescence: replication of a general psychopathology factor in the TRAILS Study. *Clinical Psychological Science*.
- Lahey BB, Applegate B, Hakes JK, Zald DH, Hariri AR, Rathouz PJ** (2012). Is there a general factor of prevalent psychopathology during adulthood? *Journal of Abnormal Psychology* **121**, 971–977.
- Lahey BB, Rathouz PJ, Keenan K, Stepp SD, Loeber R, Hipwell AE** (2015). Criterion validity of the general factor of psychopathology in a prospective study of girls. *Journal of Child Psychology and Psychiatry* **56**, 415–422.
- Lahey BB, Van Hulle CA, Singh AL, Waldman ID, Rathouz PJ** (2011). Higher-order genetic and environmental structure of prevalent forms of child and adolescent psychopathology. *Archives of General Psychiatry* **68**, 181–189.
- Laurens KR, Hobbs MJ, Sunderland M, Green MJ, Mould GL** (2012). Psychotic-like experiences in a community sample of 8000 children aged 9 to 11 years: an item response theory analysis. *Psychological Medicine* **42**, 1495–1506.
- Links PS, Eynan R** (2013). The relationship between personality disorders and Axis I psychopathology: deconstructing comorbidity. *Annual Review of Clinical Psychology* **9**, 529–554.
- Markon KE** (2010). Modeling psychopathology structure: a symptom-level analysis of Axis I and II disorders. *Psychological Medicine* **40**, 273–288.
- Measelle JR, Stice E, Hogansen JM** (2006). Developmental trajectories of co-occurring depressive, eating, antisocial, and substance abuse problems in female adolescents. *Journal of Abnormal Psychology* **115**, 524–538.
- Merikangas KR, He JP, Burstein M, Swanson SA, Avenevoli S, Cui L, Benjet C, Georgiades K, Swendsen J** (2010). Lifetime prevalence of mental disorders in U.S. adolescents: results from the National Comorbidity Survey Replication-Adolescent Supplement (NCS-A). *Journal of the American Academy of Child and Adolescent Psychiatry* **49**, 980–989.
- Muthén BO** (2007). Factor scores in SPSS vs. Mplus (<http://www.statmodel.com/discussion/messages/9/2528.html?1412177501>). Accessed 28 April 2015.
- Neal DJ, Corbin WR, Fromme K** (2006). Measurement of alcohol-related consequences among high school and college students: application of item response models to the Rutgers Alcohol Problem Index. *Psychological Assessment* **18**, 402–414.
- Newton NC, Teesson M, Barrett EL, Slade T, Conrod PJ** (2012). The CAP study, evaluation of integrated universal and selective prevention strategies for youth alcohol misuse: study protocol of a cluster randomized controlled trial. *BMC Psychiatry* **12**, 118.
- Noordhof A, Krueger RF, Ormel J, Oldehinkel AJ, Hartman CA** (2015). Integrating autism-related symptoms into the dimensional internalizing and externalizing model of psychopathology. The TRAILS Study. *Journal of Abnormal Child Psychology* **43**, 577–587.
- Patalay P, Fonagy P, Deighton J, Belsky J, Vostanis P, Wolpert M** (2015). A general psychopathology factor in early adolescence. *British Journal of Psychiatry* **207**, 15–22.
- Pettersson E, Anckarsater H, Gillberg C, Lichtenstein P** (2013). Different neurodevelopmental symptoms have a common genetic etiology. *Journal of Child Psychology and Psychiatry* **54**, 1356–1365.
- Raftery AE** (1995). Bayesian model selection in social research. *Sociological Methodology* **25**, 11–163.
- Røysamb E, Kendler KS, Tambs K, Orstavik RE, Neale MC, Aggen SH, Torgersen S, Reichborn-Kjennerud T** (2011). The joint structure of DSM-IV Axis I and Axis II disorders. *Journal of Abnormal Psychology* **120**, 198–209.
- 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.
- Tackett JL, Lahey BB, van Hulle C, Waldman I, Krueger RF, Rathouz PJ** (2013). Common genetic influences on negative emotionality and a general psychopathology factor in childhood and adolescence. *Journal of Abnormal Psychology* **122**, 1142–1153.
- van de Looij-Jansen PM, Goedhart AW, de Wilde EJ, Treffers PD** (2011). Confirmatory factor analysis and factorial invariance analysis of the adolescent self-report Strengths and Difficulties Questionnaire: how important are method effects and minor factors? *British Journal of Clinical Psychology* **50**, 127–144.
- Vollebergh WA, Iedema J, Bijl RV, 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.
- White HR, Labouvie EW** (1989). Towards the assessment of adolescent problem drinking. *Journal of Studies on Alcohol* **50**, 30–37.
- Wittchen HU, Beesdo-Baum K, Gloster AT, Höfler M, Klotsche J, Lieb R, Beauducel A, Böhner M, Kessler RC** (2009). The structure of mental disorders re-examined: is it developmentally stable and robust against additions? *International Journal of Methods in Psychiatric Research* **18**, 189–203.
- Woicik PA, Stewart SH, Pihl RO, Conrod PJ** (2009). The Substance Use Risk Profile Scale: a scale measuring traits linked to reinforcement-specific substance use profiles. *Addictive Behaviors* **34**, 1042–1055.
- Wright AG, Krueger RF, Hobbs MJ, Markon KE, Eaton NR, Slade T** (2013). The structure of psychopathology: toward an expanded quantitative empirical model. *Journal of Abnormal Psychology* **122**, 281–294.