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Regular Article

Co-development of general psychopathology and high-risk personality traits during adolescence

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Abstract

There is strong evidence for a general psychopathology dimension which captures covariance among all forms of psychopathology, yet its nature and underlying association with personality remain unclear. This study examined the co-development of general psychopathology and four high-risk personality traits: anxiety sensitivity, negative thinking, sensation seeking, and impulsivity. Data from two large Australian school-based randomised controlled trials of substance use prevention programs were analysed (N = 2,083, mean age at baseline = 13.49 years). Adolescents completed self-report measures of psychopathology symptoms and personality at baseline, one-, two-, and three-years post-baseline. Latent curve models with structured residuals, were used to examine the co-development of general psychopathology (extracted from a higher-order model) and personality traits from 13 to 16 years of age, controlling for age, sex, and cohort. Higher than usual levels of anxiety sensitivity and impulsivity were associated with higher than usual levels of general psychopathology at subsequent time points, and higher than usual levels of general psychopathology were associated with higher than usual levels of negative thinking at later time points. Sensation seeking was unrelated to general psychopathology. These findings enhance our understanding of the meaning and validity of general psychopathology, highlighting potential personality-based prevention and intervention targets.

Keywords: adolescence; developmental psychopathology; general psychopathology; personality; within-person

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Introduction

Adolescence is a critical developmental period for psychopathology, with the peak age of onset for all mental disorders being 14.5 years (Solmi et al., 2021). There are close links between psychopathology and childhood temperament and personality (De Fruyt et al., 2017; Tackett & Mullins-Sweatt, 2021), however, the co-development of personality and psychopathology during adolescence - when most psychiatric disorders emerge – is poorly understood (Solmi et al., 2021; Wilson & Olino, 2021). Extant research has focused on broad traits, such as neuroticism (Etkin et al., 2021; Mann et al., 2020), however recent research suggests that narrower aspects of personality have stronger and more diverse patterns of associations with outcomes (Stewart et al., 2022). Further, despite high rates of co-occurrence across symptom domains, most of the existing literature has examined prospective associations with individual disorders or symptom domains, providing only part of the clinical picture (De Bolle et al., 2012, 2016; Hengartner et al., 2016). The co-occurrence among symptoms and disorders can, however, be captured in a general factor of psychopathology, which facilitates the investigation of common or shared processes underlying all mental disorders (Kotov et al., 2021). Whilst the mechanisms of this general factor remains unclear, personality is speculated to play a core role, and studies that allow for causally informed conclusions are lacking (Smith et al., 2020; Southward et al., 2022).

The links between personality and psychopathology are complex with reciprocal and bidirectional effects over time and multiple explanatory models have been proposed (Tackett & Mullins-Sweatt, 2021). Personality can predispose people to experiencing certain mental health problems (vulnerability/risk model); the experience of psychopathology can lead to changes in personality (scar/complication model); or personality can impact the presentation or severity of psychopathology but not necessarily play a causal role (pathoplasty/exacerbation model). It is also possible that personality and psychopathology sit on the same continuum, ranging from general traits and subclinical characteristics through to mental disorders (continuum/spectrum model). These aren't contradictory explanations, but rather highlight the range of underlying associations that have been observed between personality and psychopathology (Tackett & Mullins-Sweatt, 2021; Wilson & Olino, 2021).

Understanding the developmental nature of personality and psychopathology has implications for the targeting of prevention and intervention efforts. For example, personality traits, or states, can be used to identify individuals who may be more vulnerable to certain mental health problems and to tailor interventions to an

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individual's specific traits, thoughts, behaviors, and emotional responses. This in turn, may enhance the effectiveness of interventions by addressing the unique needs, vulnerabilities and challenges associated with an individual's personality profile (i.e., improving the fit between interventions and persons; Edalati & Conrod, 2019; O'Leary-Barrett et al., 2016).

Few studies have examined different etiological models of personality and general psychopathology. Using cross-lagged panel models, Etkin and colleagues (2022) examined associations between the Big 5 personality traits of neuroticism, extraversion, conscientiousness, agreeableness, and openness and a bifactor model of psychopathology in adolescents. Results indicated that greater neuroticism at age 14 was associated with greater general psychopathology at age 15, and vice versa (Etkin et al., 2022). High extroversion and low conscientiousness at age 15 also predicted greater general psychopathology at age 16. These findings are consistent with both vulnerability and scar models, and echo earlier work examining narrower dimensions of psychopathology, such as internalizing and externalizing (De Bolle et al., 2012, 2016). However, further research is needed to better understand the role personality plays in the development of general psychopathology.

The focus on broad personality traits in previous studies may have masked nuanced and dissociable patterns of association between narrower facets of personality and general psychopathology (Brandes et al., 2019). For example, neuroticism has been shown to predict the emergence of multiple forms of psychopathology, and also appears to be influenced by the experience of psychopathology (Brandes & Tackett, 2019; Wilson & Olino, 2021). However, it is not clear how individual facets of neuroticism contribute to the development of general psychopathology. Understanding this could help improve early identification of at-risk adolescents and uncover cognitive or behavioral mechanisms driving the development of general psychopathology. This has significant potential for the identification of more salient intervention targets.

The four-factor model of vulnerability describes four personality traits that increase risk for substance use problems and co-occurring psychopathology (Castellanos-Ryan & Conrod, 2012). These traits are negative thinking, anxiety sensitivity, impulsivity and sensation seeking. Negative thinking, which reflects a tendency to experience hopelessness and low positive affect, and anxiety sensitivity, which refers to a fear of anxiety related physical sensations (arising from beliefs that such sensations could lead to harmful consequences) are both associated with mood and anxiety related problems, as well as substance use problems. Impulsivity, which broadly reflects a failure to inhibit behaviors likely to result in negative consequences, is associated with conduct and substance use problems. In contrast, sensation seeking, which reflects a willingness to take risks for the sake of novel experiences, is associated more specifically with substance use problems. These traits have cross-sectional and prospective associations with general psychopathology (Afzali et al., 2017; Carragher et al., 2016; Castellanos-Ryan et al., 2016; Lynch, Sunderland, et al., 2023; Newton et al., 2016). Notably, negative thinking and impulsivity assessed at age 14 were stronger predictors of general psychopathology at age 16 than neuroticism (Castellanos-Ryan et al., 2016).

A crucial limitation of previous research is the reliance on methods that conflate between-person and within-person sources of variance, potentially masking important associations or leading to erroneous conclusions. (Curran, Howard, et al., 2014; Hamaker et al., 2015a; Hopwood et al., 2022). New statistical methods have been developed to disaggregate between-person and within-person sources of variance (Bainter & Howard, 2016; Curran & Hancock, 2021). Notably, the latent curve model with structured residuals (LCM-SR) incorporates elements of latent growth curve modeling and cross-lagged panel models to describe time-specific deviations from an individual's growth trajectory, and bidirectional associations of two constructs over time (Bainter & Howard, 2016; Curran, Howard, et al., 2014). In doing so, LCM-SR allows us to examine whether fluctuations in a construct *within* individuals at one time point are associated with fluctuations in another construct at future time point (or at the same time point), after accounting for differences *between* individuals (e.g., differences in direction or rate of change, sex or gender; Curran, Howard, et al., 2014). More specifically, LCM-SR models can test directional vulnerability/risk and scar/complication models and address important questions about the temporal order of within-person associations between personality and general psychopathology, such as "if someone is experiencing higher levels of impulsivity than they usually do, does this predict higher than usual levels of general psychopathology at the next time point? Or do higher than usual levels of general psychopathology at one time predict higher than usual levels of a personality trait at the next time point?". Such questions cannot be examined with other approaches to modeling developmental processes because they do not provide pure within-person, time-specific estimates relative to an individual's growth trajectory (Bainter & Howard, 2016). The theory underlying etiological models of psychopathology and personality posits that effects occur within a given individual, rather than across individuals (Hopwood et al., 2022; Wilson & Olino, 2021). Therefore, methods that allow for direct assessment of within-person association between adjacent time points are essential to advancing our understanding of the temporal order and developmental processes underlying personality and psychopathology.

In light of the need for more longitudinal studies (Wilson & Olino, 2021), this study sought to clarify the within-person and temporal order of associations between general psychopathology and four high-risk personality traits during the transition from early adolescence to mid-adolescence, using LCM-SR to disaggregate between-person and within-person sources of variance.

Methods

Participants and procedure

The sample consisted of 2,083 adolescents (M = 13.49 years, SD = 0.44; 67% female) and most were born in Australia (84%). This sample were drawn from two large cluster randomized controlled trials investigating the effectiveness of eHealth substance use and mental health prevention programs in Australia – the Climate and Preventure (CAP) and Climate Schools Combined (CSC) studies (Newton et al., 2012; Teesson et al., 2014). Given the nature of the interventions tested in the original trials, only students allocated to the control conditions (who received health education as usual) are included in this study.

Students completed surveys in class, through either an online or paper survey. This study examined data collected at baseline, 12-, 24- and either 30- or 36-months post-baseline[1]. The overall retention rate at the final time point (30- or 36-months post-baseline) was 71%. Additional details on participant demographics

[1]The fourth data collection occurred approximately 30-months post-baseline in the CSC cohort, and 36-months post-baseline in the CAP cohort.

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and follow up rates are provided in the supplementary material (Table M1).

Measures

High-risk personality traits

Personality traits were assessed using the Substance Use Personality Risk Profile Scale (SURPS). The SURPS is a 23-item measure of personality risk for substance use problems and co-occurring psychopathology, comprised of four distinct subscales: negative thinking, anxiety sensitivity, sensation seeking and impulsivity (Woicik et al., 2009). The SURPS has demonstrated good validity and reliability as a measure of personality-related risk for substance use and co-occurring psychopathology among young people (Castellanos-Ryan et al., 2013; Newton et al., 2016; Woicik et al., 2009). Factor scores for each subscale were estimated using moderated nonlinear factor (MNLFA; Curran, McGinley, et al., 2014; see below for further details).

General psychopathology

General psychopathology was estimated as a latent factor drawn from a higher-order model of adolescent psychopathology delineated in a separate study (Lynch, Sunderland, et al., 2023). In that study, baseline data from the CAP and CSC studies were used to examine multiple competing structures of psychopathology, including correlated factors, bifactor and higher-order models. Results indicated that a higher-order model comprised of a general psychopathology dimension, and four specific dimensions (fear, distress, alcohol use/harms and conduct/inattention) outperformed alternative structures when compared using contemporary model reliability and replicability indices along with traditional fit indices. Measurement invariance tests also indicated the higher-order structure was invariant across the CAP and CSC cohorts. For the present study, factor scores were extracted for general psychopathology via MNLFA.

Indicators of the lower-order factors included items from the Strengths and Difficulties Questionnaire (Goodman, 2001), the Kessler Psychological Distress Scale (K6; (Kessler et al., 2002), Rutgers Alcohol Problem Index (Neal et al., 2006), and two items about quantity of alcohol consumed in the past 6 months and frequency of drinking at above low risk levels in the past 6 months (McBride et al., 2004). Wording of the specific items and their corresponding factors can be found in the supplementary material (Table M2).

Analysis plan

Analyses were conducted in two broad phases: 1) measurement invariance assessment using moderated nonlinear factor analysis, and 2) assessment of time-specific and developmental trajectories using latent curve models with structured residuals (LCM-SR). Both phases involved sequential, iterative model building processes. A visual summary of the analytic approach (Supplementary Figure S1) and step-by-step details for each phase of analysis are provided in the supplementary material (See Supplementary Tables M3 and M4), and briefly summarized below.

Analyses were conducted in Mplus version 8.4 for Mac (Muthén & Muthén, 2017) using robust maximum likelihood estimation. Missing data were handled via full information maximum likelihood (FIML; see Supplementary Table S9 for missing data patterns). Mplus input files were created with the assistance of R packages aMNLFA (Gottfredson et al., 2019) and Mplus Automation (Hallquist & Wiley, 2018). Analysis code and Mplus output files are available online.

Measurement invariance

MNLFA was used to assess the measurement invariance of personality and psychopathology outcomes and generate factor scores adjusted for any measurement bias (Curran, McGinley, et al., 2014). The adjusted factors were then used in subsequent analyses. MNLFA simultaneously assesses differential item functioning (DIF) across multiple grouping variables and ultimately aims to generate factors scores that have been corrected for measurement bias. Drawing on the procedures outlined by Bauer (Bauer, 2017) and Gottfredson and colleagues (Gottfredson et al., 2019), we examined DIF and mean and variance impact effects in an iterative process. As recommended by Curran and colleagues (2014), the MNLFA procedure was applied to a cross-sectional calibration sample (i.e., one measurement occasion per participant randomly drawn from any time point for each participant) to preserve the assumption of independence. Once a final model was reached, the model parameters were then applied to the full longitudinal dataset to generate adjusted factors scores for all available measurements for each participant. The factor scores used in the main analyses were adjusted for potential measurement bias relating to age, sex, and cohort (i.e., CAP or CSC). Age was included as part of the MNLFA procedure to adjust for longitudinal invariance. Further details on this procedure are provided in the supplementary materials.

Latent curve models with structured residuals

Next, we estimated a series of LCM-SR models to examine associations between general psychopathology and the four high-risk personality traits (Curran, Howard, et al., 2014; Wellman et al., 2020). LCM-SR requires an iterative model building procedure. We 1) estimated univariate models to identify the optimal shape of growth for each construct and specified structured residuals for each time point, 2) tested the inclusion of autoregressive paths using the best-fitting model, and 3) combined the best-fitting univariate models into a series of bivariate models. Once an unconditional LCM-SR model was established we then regressed the latent curve and intercept factors onto baseline age, sex, and study cohort to account for any attributable variance. A path diagram of an example model is shown in Figure 1.

Goodness-of-fit for all models was assessed using root mean square error of approximation (RMSEA), comparative fit index (CFI) and Tucker-Lewis index, where RMSEA values<0.6, and CFI and TLI values > .95 indicate close fit (TLI; Brown, 2014). Models were also compared using the information criteria, including the Akaike information criterion, Bayesian information criterion (BIC), and the sample-size adjusted BIC (aBIC), where lower values indicate superior fit (Raftery, 1995). Changes in model fit between nested models were formally evaluated with the likelihood ratio test using a scaled difference chi-square (Curran, Howard, et al., 2014). If there was no statistically significant improvement in model fit, the best-fitting model was determined based on overall fit, parsimony, and theoretical basis for components. For the final models, cross-lagged effects were interpreted using the guidelines proposed by Orth and colleagues (Orth et al., 2022), which suggest that standardized estimates of .03 indicate a small effect, .07 a medium effect, and .12 a large effect.

School-level clustered data

As the data were collected through schools (n = 26, average cluster size 79.923), we assessed the school-level intraclass correlation coefficients (ICCs) for all constructs at each time point (see Supplementary Table S1). The ICC ranged from 0.016 to 0.080,

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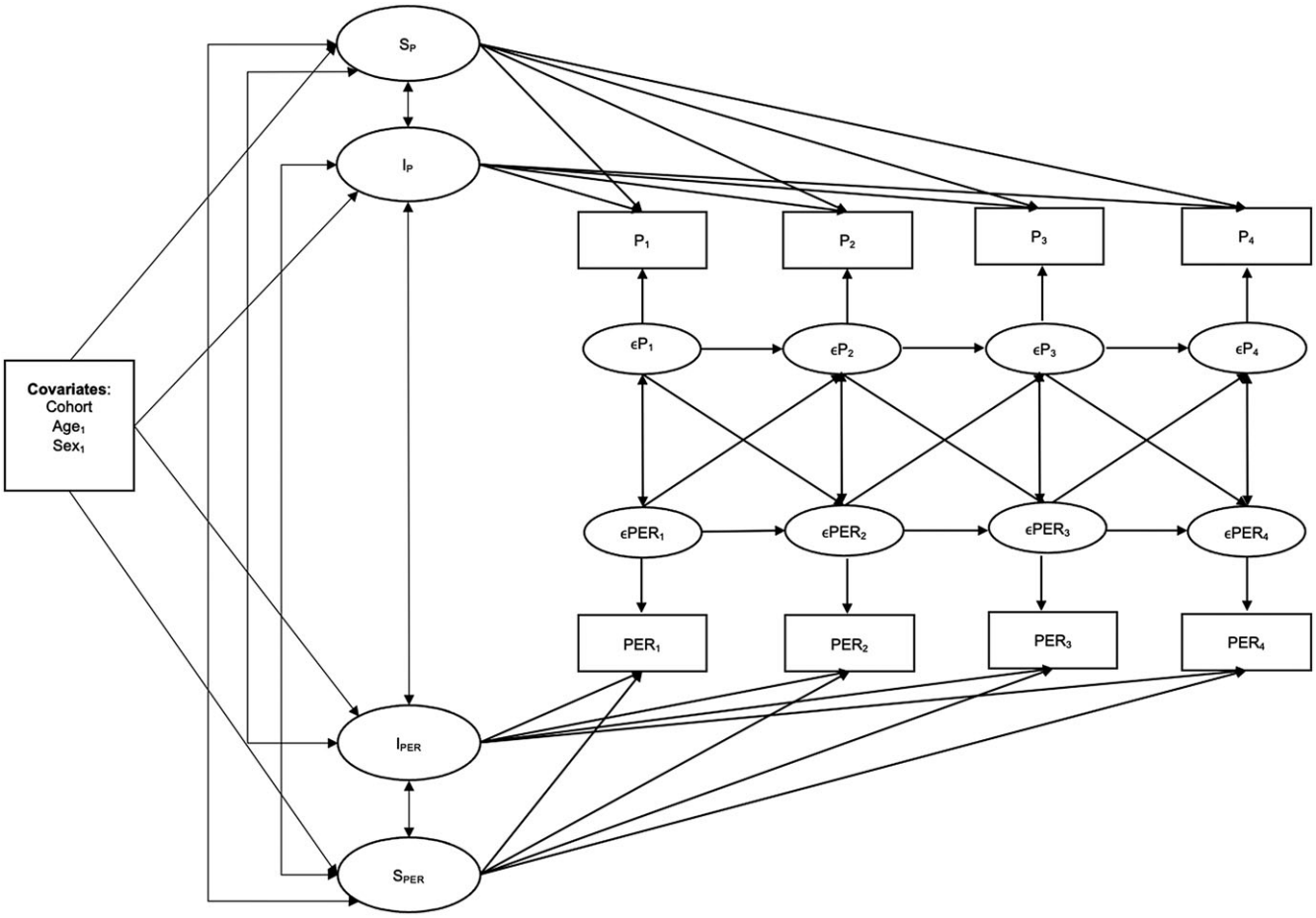

Figure 1. Example path diagram of final conditional bivariate latent curve model with structured residual models estimating co-development of general psychopathology and a personality trait. *Note.* P = general psychopathology, PER = personality, I = intercept, S = SLOPE, subscript numbers indicate time point.

indicating there was only a small amount of variance at the school level. Attempts to directly account for school-level clustering resulted in model convergence issues in both the LCM-SR and factor score generating MNLFA models, primarily due to the small number of schools. Recent simulation studies have shown that ignoring the clustered data structure has minimal impact, with substantive interpretations consistent across models that do and do not account for school-level clustering (Bailey et al., 2020; Choi, 2022). As such, we proceeded without accounting for school-level clustering. In the MNLFA models, the TYPE = COMPLEX command in Mplus was used to cluster by subject id. This command adjusts the model chi-square and standard error estimates post-hoc using a sandwich estimator, and in the MNLFA models, indirectly accounted for school-level clustering.

Results

Attrition analyses

Participants who were present at baseline only, compared to participants who completed any follow ups, were more likely to have lower negative thinking at baseline (OR 1.37 95% CI 1.01 to 1.85, $p = .044$). No other differences emerged across sex, study cohort or mean baseline scores for general psychopathology, anxiety sensitivity, impulsivity or sensation seeking (see Supplementary Tables S2 to S4).

Measurement invariance

There was some evidence of DIF and mean and variance impact effects across age, sex, and study. Factor loading non-invariance was observed for 5 items in the psychopathology models, and 4 items in the personality models. Latent factor mean differences were observed for two lower-order psychopathology factors (alcohol use/harms conduct/inattention), and negative thinking latent factors. Latent factor variance differences were observed for three lower-order psychopathology factors (fear, alcohol use/harms and conduct/inattention), and all personality factors. However, results indicated that factor scores generated from MNLFA models adjusted for non-invariance were highly correlated with a base model without non-invariance terms ($r = 0.94$ to 1.00, $p < .001$, Supplementary Table S5) suggesting factor scores generated by our base model were robust against non-invariance across sex, age, and cohort. Parameter estimates from the final MNLFA models were used to generate sex, cohort, and age adjusted factor scores for each construct (see supplementary materials for scoring models used to generate factor scores)(Table 1), (Table 2).

Factor loadings for the lower-order factors on general psychopathology indicate that the general factor more strongly reflected fear ($b = 0.998$, se = 0.027,$P < .001$), followed by distress ($b = 0.917$, se = 0.018,$P < .001$), conduct/intention ($b = 0.914$, se = 0.029,$P < .001$) problems and, to a lesser extent, alcohol use/harms ($b = 0.367$, se = 0.038,$P < .001$).

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Table 1. Results of conditional latent curve model with structured residual with general psychopathology and negative thinking

	Unstandardized			Standardized		
Between-person	*b*	SE	*p*	*β*	SE	*p*
Mean						
P Intercept	0.027	0.017	0.12			
P Slope	0.023	0.008	0.005			
NT Intercept	−0.195	0.018	< .001			
NT Slope	0.097	0.008	< .001			
Variance						
P Intercept	0.29	0.083	< .001			
P Slope	0.032	0.015	0.040			
NT Intercept	0.351	0.050	< .001			
NT Slope	0.034	0.011	0.002			
Covariances						
P Intercept with P Slope	0.002	0.029	0.94	0.024	0.320	0.941
P Intercept with NT Intercept	0.205	0.046	< .001	0.658	0.077	< .001
P Intercept with NT Slope	−0.012	0.015	0.425	−0.125	0.135	0.353
NT Intercept with Slope NT	−0.031	0.017	0.071	−0.288	0.102	0.005
NT Intercept with Slope *P*	−0.003	0.016	0.868	−0.025	0.148	0.864
P Slope with NT Slope	0.011	0.008	0.170	0.342	0.177	0.053
Within-person	*b*	SE	*p*	*β*	SE	*p*
Autoregressive coefficients						
P 13 -> *P* 14	0.185	0.098	0.060	0.169	0.100	0.091
P 14 -> *P* 15	0.22	0.055	< .001	0.218	0.052	< .001
P 15 -> *P* 16	0.07	0.121	0.563	0.084	0.139	0.544
NT 13 -> NT 14	0.131	0.069	0.059	0.116	0.064	0.070
NT 14 -> NT 15	0.131	0.069	0.059	0.122	0.065	0.061
NT 15 -> NT 16	0.131	0.069	0.059	0.159	0.078	0.042
Cross-lag coefficients						
NT 13 -> *P* 14	0.097	0.051	0.055	0.077	0.043	0.07
NT 14 -> *P* 15	0.097	0.051	0.055	0.087	0.046	0.059
NT 15 -> *P* 16	0.097	0.051	0.055	0.112	0.059	0.058
P 13 -> NT 14	0.105	0.04	0.009	0.107	0.044	0.015
P 14 -> NT 15	0.105	0.04	0.009	0.109	0.043	0.011
P 15 -> NT 16	0.105	0.04	0.009	0.134	0.054	0.013
Concurrent coefficients						
NT 13 -> *P* 13	0.133	0.046	0.004	0.399	0.089	< .001
NT 14 -> *P* 14	0.128	0.02	< .001	0.324	0.042	< .001
NT 15 -> *P* 15	0.128	0.02	< .001	0.305	0.038	< .001
NT 16 -> *P* 16	0.128	0.02	< .001	0.442	0.056	< .001

Notes. *P* = general psychopathology; NT = negative thinking. Study cohort, sex, and age at baseline included as covariates.

Latent curve models with structured residuals

Results from the iterative model building procedure are provided in the supplementary material (Supplementary Table S6), including the model fit and nested chi-scare differences likelihood ratio tests from the preliminary unconditional univariate and bivariate latent curve models with structured residuals (correlations between general psychopathology and each personality variable at each time point are available in Supplementary Figure S2). Briefly, models in which the autoregressive parameters were freely estimated for general psychopathology and constrained to equality for each of the personality traits did not degrade model fit, and we therefore retained the more constrained personality

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Table 2. Results of conditional latent curve model with structured residual between general psychopathology and anxiety sensitivity

	Unstandardized			Standardized		
Between-person	*b*	SE	*p*	*β*	SE	*p*
Mean						
P Intercept	0.035	0.017	0.045			
P Slope	0.016	0.008	0.046			
AS Intercept	−0.046	0.011	< .001			
AS Slope	–	–	–			
Variance						
P Intercept	0.286	0.067	< .001			
P Slope	0.047	0.013	< .001			
AS Intercept	0.126	0.011	< .001			
AS Slope	–	–	–			
Covariances						
P Intercept with P Slope	0.002	0.024	0.939	0.017	0.22	0.94
P Intercept with AS Intercept	0.070	0.012	< .001	0.403	0.067	< .001
P Intercept with AS Slope	–	–	–	–	–	–
AS Intercept with Slope AS	–	–	–	–	–	–
AS Intercept with Slope *P*	0.006	0.005	0.258	0.080	0.070	0.256
P Slope with AS Slope	–	–	–	–	–	–
Within-person	*b*	SE	*p*	*β*	SE	*p*
Autoregressive coefficients						
P 13 -> *P* 14	0.231	0.083	0.005	0.203	0.083	0.015
P 14 -> *P* 15	0.204	0.044	< .001	0.228	0.046	< .001
P 15 -> *P* 16	−0.131	0.153	0.392	−0.192	0.257	0.454
AS 13 -> AS 14	0.123	0.046	0.008	0.113	0.041	0.007
AS 14 -> AS 15	0.123	0.046	0.008	0.114	0.044	0.009
AS 15 -> AS 16	0.123	0.046	0.008	0.128	0.048	0.008
Cross-lag coefficients						
AS 13 -> *P* 14	0.080	0.037	0.030	0.055	0.026	0.032
AS 14 -> *P* 15	0.080	0.037	0.030	0.067	0.031	0.031
AS 15 -> *P* 16	0.080	0.037	0.030	0.105	0.054	0.052
P 13 -> AS 14	0.035	0.027	0.187	0.042	0.031	0.180
P 14 -> AS 15	0.035	0.027	0.187	0.044	0.033	0.186
P 15 -> AS 16	0.035	0.027	0.187	0.041	0.031	0.193
Concurrent coefficients						
AS 13 -> *P* 13	0.095	0.013	< .001	0.319	0.040	< .001
AS 14 -> *P* 14	0.078	0.011	< .001	0.219	0.029	< .001
AS 15 -> *P* 15	0.078	0.011	< .001	0.230	0.030	< .001
AS 16 -> *P* 16	0.078	0.011	< .001	0.347	0.090	< .001

Notes. *P* = general psychopathology; AS = anxiety sensitivity. Study cohort, sex, and age at baseline included as covariates.

models(Table 3), (Table 4). Univariate linear and quadratic growth models were estimated for all constructs. Negative residual variances for the quadratic slope were present in all models, and fixing the variance to 0 did not resolve warnings. Inspection of the mean observed scores at each time point (see Supplementary Table S8) also indicated that a linear model may be more suitable for the data, thus linear growth models were retained. All models were centered at the first time point (i.e., 0, 1, 2, 3). Univariate latent growth models indicated that residual variances for all constructs at each time point were statistically significant and ranged from 0.228 to 0.628 (see Supplementary Table S7). Models in which cross-lag effects were held equal did not degrade model fit and were

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thus retained. A correlation>1 between the two slope factors was detected in the full unconditional model with AS and general psychopathology. After adjusting for age and sex the correlation was less than 1 (-0.586, $p = 0.818$), however a correlation of 1.46 ($p = 0.825$) between the AS slope and AS intercept emerged. Therefore, the slope factor for AS was removed from the final model. The final bivariate models were estimated with study cohort, sex, and age at baseline as time-invariant covariates. The standardised and unstandardised model results are provided below in Tables 1-4.

Between-person effects

Mean, variance, and associations between the intercept and slope factors are reported in Tables 1-4. Briefly, there was a significant mean and variance for both the intercept and linear slope for general psychopathology in all models. This indicates that general psychopathology was significantly increasing at a linear rate of change, and that there was significant individual variability around both the initial level and rate of change across adolescence. The same pattern of results was observed for negative thinking. There was a significant mean and variance for the intercept of anxiety sensitivity, indicating individual heterogeneity in the initial level of anxiety sensitivity at age 13. The mean and variance estimates for the intercept of impulsivity were statistically significant, but not for the slope. This suggests that there were between person differences in the initial levels of impulsivity at age 13, but not in the rate of change of impulsivity across adolescence. Significant variability was observed in the intercept and slope of sensation seeking, but mean estimates were not statistically significant. This suggests the initial level and rate of change in sensation seeking did not statistically differ from zero, but that there is individual variability around the mean at age 13 and the rate of change across adolescence

The intercepts for general psychopathology and negative thinking were positively correlated, suggesting that at age 13, individuals with higher levels of general psychopathology tended to also have higher levels of negative thinking. Similar associations were observed between the intercepts for anxiety sensitivity, impulsivity and general psychopathology. The intercept and slope for negative thinking were negatively correlated, as was the intercept and slope for sensation seeking. These associations indicate the adolescents with lower levels of negative thinking and sensation seeking at age 13 tended to report more growth in these personality dimensions across adolescence, respectively. No other between-person covariances were statistically significant

Within-person effects

Negative thinking and general psychopathology

As shown in Table 1, the cross-lagged parameters from negative thinking to general psychopathology were not significant. In contrast, the cross-lagged parameters from general psychopathology to negative thinking were medium to large and significant ($\beta =$ 0.107-0.134 $p < .05$). This indicates that adolescents who experienced higher levels of general psychopathology than they usually do at one time point, tended to also experience higher levels of negative thinking than they were expected to at the next time point. In contrast, the concurrent associations between negative thinking and general psychopathology were stronger than the cross-lagged effects ($\beta = 0.305$-0.442 $p < .001$).

The autoregressive parameters for the structured residuals for negative thinking were not significant, and for general psychopathology only the parameter from age 14 to 15 years was statistically significant ($\beta = 0.218$, $p < .001$). This indicates that adolescents experiencing higher levels of general psychopathology at 14 years (than expected based on their underlying trajectory), tended to also experience higher levels of general psychopathology at 15 years.

Anxiety sensitivity and general psychopathology

Table 2 shows the final model results for the LCM-SR involving general psychopathology and anxiety sensitivity. There was a small to medium cross-lagged effect from anxiety sensitivity to subsequent general psychopathology ($\beta = 0.055$-0.067 $p < .05$). The cross-lagged parameters from general psychopathology to anxiety sensitivity were not statistically significant. This indicates that adolescents experiencing higher levels of anxiety sensitivity than they usually do at one time point, tended to experience higher levels of general psychopathology than they usually would at the next time point.

The autoregressive parameters for the structured residuals for anxiety sensitivity were large and significant ($\beta = 0.113$-0.128, $p < .01$), indicating that there were enduring adolescent specific deviations, or increases, in anxiety sensitivity over the follow up period. That is, adolescents experiencing higher than their usual level of anxiety sensitivity, consistently tended to experience higher levels of anxiety sensitivity than they usually would at the next time point. For general psychopathology, there were large autoregressive effects from age 13 to 14 years ($\beta = 0.231$, $p < .05$) and 14 to 15 years ($\beta = 0.228$, $p < .001$), indicating that adolescents experiencing higher general psychopathology than they usually do, tended to also experience higher than usual general psychopathology at the next time point, but only up until age 15.

Stronger concurrent associations were observed between anxiety sensitivity and general psychopathology ($\beta = 0.219$-0.347 $p < .001$) than the cross-lagged effects. This indicates that higher than expected levels anxiety sensitivity may be a stronger indicator of current general psychopathology, rather than future levels of general psychopathology or anxiety sensitivity.

Impulsivity and general psychopathology

As shown in Table 3, there was a medium to large cross-lagged effect from impulsivity to general psychopathology ($\beta = 0.071$-0.127, $p < .05$), indicating that adolescents with higher than usual levels of impulsivity tended to also have higher than usual levels of general psychopathology at the next time point. The cross-lagged effect from general psychopathology to impulsivity was non-significant.

The autoregressive parameters for the structured residuals for impulsivity were not statistically significant. For general psychopathology there were medium, significant effects from age 13 to 14 years ($\beta = 0.208$, $p < .05$) and 14 to 15 years ($\beta = 0.228$, $p < .001$). This suggests that adolescents experiencing higher general psychopathology than they usually would, tend to experience higher than usual general psychopathology at the next time point but only up until age 15, whereas any adolescents experiencing deviations in their usual level of impulsivity tended to fall back to their usual level quite quickly.

Concurrent associations between impulsivity and general psychopathology were larger ($\beta = 0.243$-0.413 $p < .001$) than the cross-lagged or autoregressive effects. This indicates that higher than usual levels of impulsivity may be a stronger indicator of current general psychopathology levels, than future general psychopathology or impulsivity.

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Table 3. Results for conditional latent curve model with structured residual between general psychopathology and impulsivity

	Unstandardized			Standardized		
Between-person	*b*	SE	*p*	β	SE	*p*
Mean						
P Intercept	0.028	0.017	0.113			
P Slope	0.021	0.008	0.010			
IMP Intercept	0.270	0.019	< .001			
IMP Slope	−0.013	0.009	0.151			
Variance						
P Intercept	0.270	0.089	0.003			
P Slope	0.045	0.016	0.006			
IMP Intercept	0.317	0.075	< .001			
IMP Slope	0.013	0.017	0.420			
Covariances						
P Intercept with *P* Slope	0.008	0.032	0.804	0.074	0.323	0.818
P Intercept with IMP Intercept	0.124	0.051	0.015	0.445	0.112	< .001
P Intercept with IMP Slope	0.008	0.017	0.614	0.149	0.365	0.683
IMP Intercept with Slope IMP	0.002	0.026	0.954	0.025	0.448	0.956
IMP Intercept with Slope *P*	0.017	0.018	0.335	0.149	0.180	0.408
P Slope with IMP Slope	−0.008	0.008	0.302	−0.361	0.535	0.499
Within-person	*b*	SE	*p*	β	SE	*p*
Autoregressive coefficients						
P 13 -> *P* 14	0.233	0.092	0.012	0.208	0.096	0.031
P 14 -> *P* 15	0.202	0.049	< .001	0.228	0.050	< .001
P 15 -> *P* 16	−0.136	0.169	0.423	−0.200	0.287	0.485
IMP 13 -> IMP 14	0.109	0.074	0.141	0.103	0.073	0.161
IMP 14 -> IMP 15	0.109	0.074	0.141	0.108	0.073	0.143
IMP 15 -> IMP T4	0.109	0.074	0.141	0.113	0.073	0.121
Cross-lag coefficients						
IMP 13 -> *P* 14	0.072	0.033	0.030	0.071	0.035	0.040
IMP 14 -> *P* 15	0.072	0.033	0.030	0.085	0.04	0.032
IMP 15 -> *P* T4	0.072	0.033	0.030	0.127	0.065	0.050
P 13 -> IMP 14	0.094	0.048	0.050	0.081	0.044	0.069
P 14 -> IMP 15	0.094	0.048	0.050	0.089	0.046	0.054
P 15 -> IMP T4	0.094	0.048	0.050	0.083	0.045	0.067
Concurrent coefficients						
IMP 13 -> *P* 13	0.155	0.052	0.003	0.354	0.079	< .001
IMP 14 -> *P* 14	0.121	0.021	< .001	0.243	0.036	< .001
IMP 15 -> *P* 15	0.121	0.021	< .001	0.272	0.038	< .001
IMP T4 -> *P* T4	0.121	0.021	< .001	0.413	0.104	< .001

Notes. *P* = general psychopathology; IMP = impulsivity. Study cohort, sex, and age at baseline included as covariates.

Sensation seeking and general psychopathology

The cross-lagged parameters from sensation seeking to general psychopathology, and general psychopathology to sensation seeking, were not statistically significant (see Table 4). The autoregressive parameters for the structured residuals for sensation seeking were also not statistically significant. For general psychopathology, only the autoregressive effect from 14 to 15 years was statistically significant. Altogether, the findings from this model suggest that fluctuations in sensation seeking and general psychopathology were not related.

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Table 4. Results of conditional latent curve model with structured residual between general psychopathology and sensation seeking

	Unstandardized			Standardized		
Between-person	*b*	SE	*p*	*β*	SE	*p*
Mean						
P Intercept	0.028	0.017	0.110			
P Slope	0.021	0.008	0.008			
SS Intercept	−0.007	0.014	0.853			
SS Slope	0.003	0.007	0.655			
Variance						
P Intercept	0.337	0.089	< .001			
P Slope	0.067	0.017	< .001			
SS Intercept	0.245	0.022	< .001			
SS Slope	0.032	0.007	< .001			
Covariances						
P Intercept with *P* Slope	−0.018	0.033	0.582	−0.123	0.196	0.531
P Intercept with SS Intercept	−0.003	0.021	0.870	−0.012	0.074	0.869
P Intercept with SS Slope	0.002	0.009	0.851	0.016	0.086	0.850
SS Intercept with Slope SS	−0.023	0.009	0.011	−0.264	0.071	< .001
SS Intercept with Slope *P*	0.000	0.009	0.977	−0.002	0.069	0.977
P Slope with SS Slope	−0.007	0.004	0.097	−0.158	0.093	0.089
Within-person	*b*	SE	*p*	β	SE	*p*
Autoregressive coefficients						
P 13 -> *P* 14	0.200	0.127	0.114	0.166	0.12	0.168
P 14 -> *P* 15	0.183	0.039	< .001	0.209	0.041	< .001
P 15 -> *P* T4	−0.306	0.231	0.186	−0.800	1.315	0.543
SS 13 -> SS 14	0.020	0.072	0.775	0.015	0.052	0.777
SS 14 -> SS 15	0.020	0.072	0.775	0.02	0.069	0.775
SS 15 -> SS T4	0.020	0.072	0.775	0.025	0.086	0.772
Cross-lag coefficients						
SS 13 -> *P* 14	−0.013	0.043	0.764	−0.007	0.023	0.765
SS 14 -> *P* 15	−0.013	0.043	0.764	−0.011	0.037	0.763
SS 15 -> *P* T4	−0.013	0.043	0.764	−0.030	0.096	0.750
P 13 -> SS 14	−0.028	0.031	0.367	−0.031	0.034	0.367
P 14 -> SS 15	−0.028	0.031	0.367	−0.035	0.039	0.366
P 15 -> SS T4	−0.028	0.031	0.367	−0.038	0.042	0.368
Concurrent coefficients						
SS 13 -> *P* 13	−0.005	0.020	0.815	−0.023	0.096	0.813
SS 14 -> *P* 14	0.003	0.010	0.760	0.009	0.029	0.759
SS 15 -> *P* 15	0.003	0.010	0.760	0.010	0.032	0.760
SS T4 -> P T4	0.003	0.010	0.760	0.051	0.280	0.855

Notes. *P* = general psychopathology; SS = sensation seeking. Study cohort, sex, and age at baseline included as covariates.

Discussion

This study examined the co-development of a higher-order general psychopathology factor and four high-risk personality traits from early to mid-adolescence. The use of a novel statistical approach allowed us to clarify the temporal ordering and gain a better understanding of the bidirectional associations between fluctuations in personality and general psychopathology from early- to mid-adolescence. We found that certain aspects of personality were associated with the development of general psychopathology while others were instead associated with prior fluctuations in general psychopathology.

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Specifically, within-person deviations in anxiety sensitivity and impulsivity predicted subsequent deviations in general psychopathology, and deviations in general psychopathology predicted subsequent deviations in negative thinking over adolescence. In other words, if a person scores higher (or lower) than their expected level of anxiety sensitivity or impulsivity, they are likely to score higher (or lower) than their expected level of general psychopathology. Conversely, if a person exhibits higher (or lower) than their expected level of general psychopathology, they are likely to score higher (or lower) than their expected level of negative thinking the following year. Thus, there was evidence for both vulnerability and pathoplasty models, depending on the aspect of personality. This supports previous research that has indicated bidirectional and reciprocal associations between general psychopathology and neuroticism (Brandes et al., 2019; Etkin et al., 2021; Khan et al., 2005; Mann et al., 2020; Williams et al., 2021). However, the present study adds nuance to these findings by highlighting the different roles played by different aspects of neuroticism in the development of psychopathology. This suggests that focusing on broader personality traits may mask underlying and differing patterns of association with general psychopathology. Additionally, the findings suggest that monitoring for fluctuations in personality traits may help identify young people at risk of future increases in general psychopathology.

The large concurrent associations between general psychopathology and negative thinking, anxiety sensitivity and impulsivity indicate that temporal deviations in these traits coincide with deviations in general psychopathology and reinforce the potential utility of personality as a monitoring and screening tool for emerging adolescent psychopathology. This finding is consistent with previous research and provides further support for the continuum/spectrum model (Afzali et al., 2017; Carragher et al., 2016; Castellanos-Ryan et al., 2016; Etkin et al., 2021). Furthermore, the concurrent and prospective associations between negative thinking, anxiety sensitivity and impulsivity lend support to the notion that dispositional negative affect and impulsivity are core features of general psychopathology (Smith et al., 2020; Southward et al., 2022).

Deviations from a person's usual trajectory of negative thinking, impulsivity and sensation seeking at one time point tended not to carry over to the next time point. In contrast, deviations in anxiety sensitivity on one occasion were associated with future deviations in anxiety sensitivity. This suggests that individuals experiencing higher than usual levels of anxiety sensitivity in early adolescence tend to experience persistent higher than usual levels of anxiety sensitivity through to mid-adolescence. In contrast, fluctuations in negative thinking, impulsivity and sensation seeking tend to be temporary. Interestingly, deviations in general psychopathology tended to carry-over between the ages of 14 and 15. This aligns with evidence for the peak age of onset for any mental disorder being 14.5 years and reinforces the importance of continuous, reliable measures of psychopathology and of delivering prevention and early intervention during early to mid-adolescence (Fusar-Poli et al., 2021; Solmi et al., 2021).

Sensation seeking was unrelated to general psychopathology at the within-person level. This is likely due to sensation seeking being more directly related to substance sue problems (Castellanos-Ryan & Conrod, 2011; O'Connor et al., 2021), and therefore less likely to be associated with general psychopathology across adolescence.

Strengths and limitations

This study had several strengths, including a large adolescent sample, multi-wave design over a critical developmental period and statistical approach that allowed for the examination of temporal order of individual level fluctuations in personality and psychopathology. However, there are some limitations of the study that should also be considered. First, although the general psychopathology factor in this study was comprised of a broad range of mental health symptoms, some common symptom domains were not assessed, including psychosis and eating pathology. Second, 67% of the sample were female. To account for this, we used factor scores adjusted for sex-related measurement bias in the primary analyses and included sex as a covariate in final models. Third, as self-report measures of psychopathology and personality were used it is possible that the observed association may be a product of shared method variance. Future research using a multi-informant approach would be beneficial. Fourth, although modern measurement invariance procedures were applied to ensure scores were adjusted for bias relating to sex, cohort and age, the specific model constraints applied during this procedure prevented the calculation of reliability coefficients for the latent factors. However, previous analyses have demonstrated the latent general psychopathology factor to have good reliability in this sample at age 13 (Lynch, Sunderland, et al., 2023). Fifth, the two RCT cohorts had slightly different intervals between the 2 year and 3 year follow up (6 months in CSC vs. 12 months in CAP), which may have limited our ability to detect autoregressive effects between ages 15 and 16. We attempted to use phantom variables (i.e., variables with missing data for all participants at 6- and 18-months; and 30- and 36-month variables with data missing for all CAP and CSC participants, respectively) to accommodate the unequal assessment intervals, however these models did not converge, potentially due to the number of phantom variables needed (Hounkpatin et al., 2018). Unfortunately, low covariance coverage within the CAP sample meant we were unable to conduct sensitivity analyses to examine potential influences of the uneven assessment periods. Thus, time-specific effects between ages 15 and 16 should be interpreted with some caution. In addition, there was substantial missing data due to attrition. Although attrition analyses indicated there were some differences in baseline levels of negative thinking, there were no differences in other personality or general psychopathology variables. Finally, the personality variables examined in this study are moderately correlated, and it would be informative to examine the associations between general psychopathology and these personality facets in a multivariate framework. Attempts to model all variables simultaneously were found to be unfeasible due to the large number of parameters. A larger sample size might be needed to estimate the shared and distinct reciprocal influences of different personality facets in the same model.

Implications and future directions

This study sheds light on the association between personality and psychopathology during adolescence and has implications for prevention timing and targeting as well as substantive interpretations of general psychopathology. Results support the idea that general psychopathology likely reflects interactions between dispositional negative emotionality, impulsive emotional responsiveness, and nonspecific impairment (Carver et al., 2017; Smith et al., 2020; Southward et al., 2022). The different pattern of effects for negative thinking compared to anxiety sensitivity and impulsivity suggests that a core functional mechanism of general psychopathology may be a sensitivity or responsivity to aversive and rewarding stimuli, which results in negative emotionality (or

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potentially manifests as other maladaptive traits or symptoms). Understanding the substantive meaning of general psychopathology is crucial to understanding its causes and advancing intervention development.

Although the observational nature of the present limits causal interpretations of the results (due to the possibility of unmeasured confounding variables), the pure within-person cross-lagged and autoregressive effects satisfy the Granger criteria for causality and at the very least reveal temporal precedence that can be actioned upon (Granger, 1969; Hamaker et al., 2015b). As such, findings from the present study may have implications for the timing and targeting of prevention and early intervention strategies. For example, the present study corroborates existing evidence for early adolescence as an ideal time for preventive interventions. In this study, deviations in general psychopathology at age 15 were consistently predicted by deviations in general psychopathology at age 14. This suggests that early adolescence may be an optimal time to implement programs to prevent or disrupt development of general psychopathology. Given the likely onset of new symptom domains during this period, it seems plausible that a booster, or early intervention, at age 15 could also reduce growth in general psychopathology beyond mid-adolescence, warranting further empirical investigation. Research spanning a longer developmental period could shed further light on the optimal time to deliver interventions, and whether timing varies among different personality risk profiles.

Results also support using personality to identify adolescents at risk of developing multiple forms of psychopathology (as represented by general psychopathology) and highlight the potential for tailoring interventions to different personality risk profiles. Notably, the medium-large effect size from impulsivity to general psychopathology suggests this may be a particularly important target for prevention programs, compared to the small-medium effect observed from anxiety sensitivity to general psychopathology. Sensation seeking may be useful for early detection and prevention of substance use specific symptom domains, whereas other traits, such as impulsivity, negative thinking, or anxiety sensitivity, may be able to identify those at risk of psychopathology more broadly, and suggest potential mechanisms of change. Moreover, the sustained increases in general psychopathology point to the potential utility of interventions targeting multiple symptom domains, such as anxiety, depression, and substance use, across adolescence (Lynch, Chapman, et al., 2023; Teesson et al., 2020).

This three-year study examined longitudinal associations between a higher-order general psychopathology factor and four high-risk personality traits among adolescents. Spectrum, scar/complication, vulnerability/risk and pathoplasty models were supported. Fluctuations in impulsivity and anxiety sensitivity preceded fluctuations in general psychopathology, and fluctuations in general psychopathology were associated with subsequent fluctuations in negative thinking. Deviations in general psychopathology persisted from age 14 to 15, suggesting this may be a critical developmental window for the progression of general psychopathology. The modifiable processes underlying personality-general psychopathology associations, and the functional utility of general psychopathology, need further research. Such evidence could help develop interventions that disrupt the mechanisms linking personality and psychopathology, and ultimately prevent the onset of multiple forms of psychopathology simultaneously. This study advances understanding of the role of certain aspects of personality in the development of general psychopathology and highlights the potential of general psychopathology preventative interventions that are tailored to different personality traits and delivered during early adolescence to effectively and efficiently target those at greatest risk.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579424001871.

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