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Article

Persistence of Anxiety/Depression Symptoms in Early Adolescence: A Prospective Study of Daily Life Stress, Rumination, and Daytime Sleepiness in a Genetically Informative Cohort

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Abstract

In this prospective study of mental health, we examine the influence of three interrelated traits — perceived stress, rumination, and daytime sleepiness — and their association with symptoms of anxiety and depression in early adolescence. Given the known associations between these traits, an important objective is to determine the extent to which they may independently predict anxiety/depression symptoms. Twin pairs from the Queensland Twin Adolescent Brain (QTAB) project were assessed on two occasions ($N = 211$ pairs aged 9–14 years at baseline and 152 pairs aged 10–16 years at follow-up). Linear regression models and quantitative genetic modeling were used to analyze the data. Prospectively, perceived stress, rumination, and daytime sleepiness accounted for 8–11% of the variation in later anxiety/depression; familial influences contributed strongly to these associations. However, only perceived stress significantly predicted change in anxiety/depression, accounting for 3% of variance at follow-up after adjusting for anxiety/depression at baseline, although it did not do so independently of rumination and daytime sleepiness. Bidirectional effects were found between all traits over time. These findings suggest an underlying architecture that is shared, to some degree, by all traits, while the literature points to hypothalamic–pituitary–adrenal (HPA) axis and/or circadian systems as potential sources of overlapping influence and possible avenues for intervention.

Keywords: Adolescence; anxiety; depression; daily stress; rumination; daytime sleepiness

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Adolescent mental health is an issue of global concern, with anxiety and depression symptoms being among the most widely reported complaints to impact wellbeing (Islam et al., 2020; Merikangas et al., 2010; Racine et al., 2021; Tiirikainen et al., 2019). Critically, adolescence is a time of heightened stress and emotional reactivity and vulnerability for the emergence of psychopathology (Ahmed et al., 2015; Fuhrmann et al., 2015; Hauser et al., 2019; Romeo, 2013; Shorey et al., 2021). It is a period of rapid development with extensive neural rewiring and psychosocial change (Andrews et al., 2021; Lichenstein et al., 2016). In addition, normative developmental changes include shifts in adolescent sleep–wake behaviors that contribute to the prevalence of daytime sleepiness, which is a known risk factor for symptoms of anxiety and depression in adolescents (Crowley et al., 2018; Liu et al., 2019; Luo et al., 2018; Meyer et al., 2018).

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Perceived Stress

There is a robust literature supporting links between stressors and child and adolescent psychopathology (Grant et al., 2004; Gunnar, 2021). Early adolescence is marked by significant transitions in stress reactivity, as pubertal development and stress interact to impact one of the body's main stress response systems — the hypothalamic–pituitary–adrenal (HPA) axis (Romeo, 2010). Indeed, studies in rats show that HPA axis function in response to stressors is prolonged in adolescence, compared to adulthood (McCormick & Mathews, 2007). While most studies to date have focused on the effects of adverse life events (Grant et al., 2004; Gunnar, 2021), perceived stress in relation to everyday stressors has also emerged as a marker for risk of developing a mental disorder (Lindholdt et al., 2021). School-related stressors have been identified as a major source of subjective stress in adolescents (Anniko et al., 2019; Kaczmarek & Trambacz-Oleszak, 2021). Perceived stress may offer insights that stress exposure measures do not, as perceived stress reflects an individual's interpretation of environmental stressors and their anticipated ability to successfully manage stressful situations (Hu et al., 2014).

Rumination

Dysfunctional emotion regulation has been implicated as a key transdiagnostic construct in the development and maintenance of psychopathology (Olatunji et al., 2013; Sloan et al., 2017; Watkins & Roberts, 2020). In a metaanalytic review of emotion regulation strategies in adolescents aged 13 to 18 years, rumination was identified as a maladaptive strategy that negatively impacts subclinical adolescent anxiety and depression symptoms (Schafer et al., 2017). Similar results have been reported for 722 younger adolescents, aged 11 to 13 years (Hilt et al., 2010). Depressive rumination is a maladaptive thought process, or cognitive vulnerability, defined as repetitive self-focus on symptoms of depression, including possible causes and consequences, which can influence symptom duration and severity (Nolen-Hoeksema, 1991; Watkins & Roberts, 2020). Rumination has been associated with perceived stress and physiological stress responses (e.g. as evidenced by HPA axis indices) and further may mediate maladaptive HPA axis activation — a pathway through which it may influence mental health (Gianferante et al., 2014; Hilt, Sladek, et al., 2017; Hu et al., 2014). Adolescence is a particularly challenging time for the regulation of emotions as the underlying neural circuitry supporting these skills is still developing, while social demands and challenges are increasing (Ahmed et al., 2015; Andrews et al., 2021; Young et al., 2019).

Daytime Sleepiness

Daytime sleepiness is an additional burden than can exacerbate adolescent mental health problems, and the relationship may be reciprocal (Hestetun et al., 2018; Luo et al., 2018). This may be due in part to influences on perceived stress and level of rumination, with follow-on effects for mental health (e.g. Bian et al., 2020; Lindholdt et al., 2021; Schafer et al., 2017; Thomsen et al., 2003; Ye et al., 2021). For example, excessive daytime sleepiness has been associated with high levels of perceived study stress in high-school students (Luo et al., 2018). Sleepiness may worsen perceived stress levels by reducing an individual's perceived ability to negotiate everyday stressors (Foster, 2020). In addition, poor sleep quality has been associated with a tendency to ruminate in college students (Bian et al., 2020), and similarly, greater sleepiness has been associated with poorer emotional self-regulation in adolescents (Owens et al., 2016). Importantly, daytime sleepiness is a very common adolescent complaint that has multiple etiologies, including pubertal maturation-induced changes in sleep architecture (Agostini & Centofanti, 2021; Hein et al., 2020). As with stress and rumination, daytime sleepiness has been linked to HPA axis activation, and specifically, with blunted cortisol stress responsiveness (van Dalfsen & Markus, 2018). This may reflect the chronic nature of daytime sleepiness, as blunted responsiveness is thought to characterize prolonged, or persistent stress exposure (Fries et al., 2005; Miller et al., 2007).

Longitudinal Associations

Longitudinal studies suggest that, in adolescents, mental health is prospectively influenced by both perceived psychological stress (Felton et al., 2017; Lindholdt et al., 2021) and rumination (Orue et al., 2014; Padilla Paredes & Calvete Zumalde, 2015; Wilkinson et al., 2013). Stress has complex and reciprocal associations with anxiety and depression, and these relationships may be influenced by cognitive vulnerabilities, including rumination (Liu & Alloy, 2010; Michl et al., 2013; Morrison & O'Connor, 2005). Studies in adults suggest bidirectional associations between daytime sleepiness and depression (Jaussent et al., 2017; Karunanayake et al., 2019; LaGrotte et al., 2016). Similarly, sleep problems in twins aged 8 years predict depression at age 10, although the converse was not found (Gregory et al., 2009).

Genetic Sources of Influence

Anxiety and depression, perceived stress, rumination, and daytime sleepiness are interrelated traits. All are associated with HPA axis function (Chen, 2022; Gianferante et al., 2014; Romeo, 2010; van Dalfsen & Markus, 2018), which may partly explain their covariation, and twin studies suggest genetic overlap among the traits. In young adults, covariation between perceived psychological stress and depressive symptoms appears to have a strong genetic component (Michalski et al., 2017; Rietschel et al., 2014). Similar associations are reported between rumination and depressed mood in adolescents (Moore et al., 2013), while a modest overlap in genetic factors appears to contribute to covariation between daytime sleepiness and depressive symptoms in elderly men (Lessov-Schlaggar et al., 2008).

The Current Study

This study aims to prospectively investigate the relative importance of perceived stress, rumination, and daytime sleepiness as predictors of adolescent anxiety and depression. Given the known associations between these traits, we set out to determine the extent to which they may independently predict anxiety/depression symptoms. Further, having a twin cohort provides the opportunity to explore the role of genetic and environmental influences on these relationships, providing insight into the nature of factors influencing persistence of anxiety/depression symptoms in early adolescence.

Methods

Participants

The Queensland Twin Adolescent Brain (QTAB) cohort was recruited primarily through the Queensland Twin Registry (QTwin), but with additional twins recruited through Twins Research Australia and through a QTAB study website (Strike et al., 2022). All families, with young adolescent twins (or triplets), resided in south-east Queensland and were able to attend a brain imaging and data collection session at the Centre for Advanced Imaging, University of Queensland (for the 5 triplet sets, only 2 individuals per triplet set were imaged, and they are treated as twin pairs in all analyses). Baseline data were collected from 422 participants (211 families) between June 2017 and October 2019, with twin pairs aged 9.0 to 14.4 years ($M = 11.3 \pm 1.3$ years). Twin pairs comprised 46 monozygotic female (MZF) pairs, 57 monozygotic male (MZM) pairs, 34 dizygotic female (DZF) pairs, 30 dizygotic male (DZM) pairs, and 44 opposite-sex (DZO) pairs. Of these, 152 families (including two families with triplets) participated in a second wave of data collection (November 2019 to January 2021). At follow-up, the twins were aged 10.1 to 16.4 years ($M = 13.0 \pm 1.5$ years), with pairs comprising 36 MZF, 38 MZM, 26 DZF, 19 DZM, and 33 DZO pairs. The interval from baseline to follow-up ranged from 1.1 to 2.5 years ($M = 1.7 \pm .30$ years).

Twin pairs were excluded if either co-twin had serious medical, neurological, or cardiovascular conditions, history of a serious head injury, diagnosis of autism spectrum disorder or psychiatric

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disorder, and any cognitive, physical, or sensory challenges that would limit ability to understand or complete procedures. In addition, MRI contraindication (e.g. metal implants, artifact-inducing orthodontic braces) was cause for exclusion. Written and informed consent was obtained from a parent and from each adolescent. A parent and each adolescent received an honorarium (AUD$50 each) for participating in the study. Ethics approval for the study was obtained from the Children's Health Queensland Human Research Ethics Committee and ratified by The University of Queensland.

Procedure

Participants completed a mental health questionnaire on an iPad during their visit. Twins were assessed in parallel, with one twin being scanned while the other completed iPad and other assessments. Equivalent and additional assessments were completed by a parent. The nonimaging data are stored at The University of Queensland's institutional repository, UQ eSpace (https://doi.org/10.48610/e891597). Access to the dataset can be requested via the *Request access to the dataset link* in the UQ eSpace record.

Measures

Predictor variables: perceived stress, rumination, daytime sleepiness. Perceived stress was obtained from the Daily Life Stressors Scale. This 30-item scale was designed to assess the impact of everyday problems and stressors, at school and at home, for children and adolescents aged 7−17 years, and this age group shows construct and concurrent validity (Kearney et al., 1993). The scale addresses the severity of negative daily life events (e.g. 'My parents yell at me in the morning') and negative affectivity (e.g. 'I am tense or nervous when I have to answer a question in class') within the past week. Items are self-rated on a 5-point Likert-type scale and summed to obtain a total score ranging from 0 to120. Where responses were missing for 1−2 items, we replaced with the participant's mean item score (four participants were missing one item, and one participant was missing two items at baseline). Three participants missing nine or more responses at baseline were not scored. There were no missing data at follow-up.

Rumination was assessed with the Rumination Subscale of the Children's Response Styles Questionnaire. This subscale comprises 13 items describing self-focused responses to depressed mood (Abela et al., 2002) and has good validity and internal consistency in young adolescents (Alloy et al., 2012; Shapero et al., 2017). Items are self-rated on a 4-point Likert scale and summed to obtain a total score ranging from 0 to 39. Baseline data were not available for three participants. There were no missing data at follow-up.

Daytime sleepiness was assessed with the Pediatric Daytime Sleepiness Scale, which has robust psychometric properties in adolescents aged 11 to 15 years (Drake et al., 2003). Multiple studies have used the scale in younger children (see review by Meyer et al., 2017), including validation studies (e.g. Nouri et al., 2021). This self-report 8-item scale is scored on a 5-point Likert-type scale with items summed to obtain a total score ranging from 0 to 32. At baseline, missing data were replaced with the participant's item mean for two participants missing one item. Three participants had no data at baseline for this scale. There were no missing data at follow-up.

Outcome variable: anxiety/depression. A measure of anxiety/depression symptoms was derived from the Somatic and Psychological Health Report (SPHERE). The SPHERE was first developed as a 34-item self-report questionnaire to assess symptoms of mental distress and persistent fatigue (Hickie et al., 2001). Here we use a 14-item anxiety/depression subscale that has been validated in an Australian-based population sample aged 9 to 28 years, where it was associated with later DSM-IV diagnoses of major depressive disorder, social anxiety, and alcohol dependence (OR 1.23–1.47; Couvy-Duchesne et al., 2017). Similarly, it has been associated with concurrent neuroticism in adolescents and young adults ($r = .64$; Hansell et al., 2012). There were no missing data for this scale at baseline or follow-up. Items are self-rated on a 4-point Likert-type scale and summed to obtain a total score ranging from 0 to 42.

Covariates. Symptoms of anxiety and/or depression are higher in females than males (Altemus et al., 2014; Salk et al., 2017) and increase with: age across adolescence (Salk et al., 2017), advancing pubertal development (Altemus et al., 2014; Stumper & Alloy, 2021), and socioeconomic disadvantage (Peverill et al., 2020). A potential confounder for prediction analyses is the varying interval between baseline and follow-up assessment. In addition, follow-up assessment was interrupted by a COVID-19-related lockdown of approximately 3 months, during which schools were closed. At follow-up, 31% of the sample was assessed prior to the lockdown, with the remainder assessed postlockdown.

Pubertal development was assessed using self-report and parental-report versions of the Pubertal Development Scale (Carskadon & Acebo, 1993; Petersen et al., 1988). This scale assesses the development of secondary sexual characteristics, with questions about growth spurts, body hair growth, and skin changes (e.g. pimples), as well as breast development and menarche in girls, and voice changes and facial hair growth in boys. Except for menarche, which is scored yes/no, items are scored on a 4-point Likert-type scale with options ranging from 'has not begun' to 'already complete'. The average scale score (ranging from 1 to 4) was determined. At baseline, a pubertal scale score was available for all but one participant (supplemented with data from parent-report for 179 participants and scored on four rather than five items for one participant). At follow-up, a pubertal scale score was available for all but three participants (supplemented with data from parent-report for 83 participants and scored on four rather than five items for eight participants).

Neighborhood socioeconomic status (SES) was computed using the Australian Bureau of Statistics (ABS) 2016 Census-based Socioeconomic Index for Areas, which ranks suburbs in Australia according to relative socioeconomic advantage and disadvantage (ABS, 2016). Higher scores indicate greater advantage in general, including more high-income households and more people in skilled occupations. There were no missing data.

Analyses

Anxiety/depression, perceived stress, and rumination had minor positive skew and were square root-transformed then standardized ($M = 0$, $SD = 1$) and minor outliers (all <4**SD**) winsorized to ±3.3 *SD*. Analyses were conducted using IBM SPSS Statistics Version 27 and R version 4.1.2 (R Core Team, 2021). Unless stated otherwise, all analyses including baseline data use the full sample.

Phenotypic analyses. A series of linear regression models were used to explore cross-sectional and longitudinal associations.

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Predicting anxiety/depression. Cross-sectional analyses examined, first, the extent to which perceived stress, rumination, and daytime sleepiness were associated with concurrent anxiety/depression. Second, they explored the independence of such associations with concurrent anxiety/depression.

Longitudinal analyses first determined that the proportion of variance in anxiety/depression at follow-up was predicted individually by baseline measures of daytime sleepiness, perceived stress, and rumination. The second series of longitudinal analyses included anxiety/depression at baseline as a predictor, to test whether perceived stress, rumination, or daytime sleepiness predicted change in anxiety/depression (i.e. residual variance in anxiety/depression at follow-up after accounting for baseline anxiety/depression). Third, relative to each other, the extent to which daytime sleepiness, perceived stress, and rumination predicted independent change in anxiety/depression was assessed.

Bidirectionality of influence across time. Using both baseline and follow-up measures, these analyses explored the extent of bidirectionality between anxiety/depression, perceived stress, rumination, and daytime sleepiness.

Mixed-effects linear regressions were conducted using the R package lme4 (Bates et al., 2015). Covariates having at least a nominally significant association with the outcome variable were included as fixed effects. To account for the nonindependence of twins, family, and zygosity (identical vs. nonidentical) were included in the model as random effects (Visscher et al., 2004). An experiment-wide significance threshold of $p < .0085$, based on the identification of six effective independent predictor/outcome variables (equation 5 of Li & Ji, 2005), was adopted to keep the Type I error rate at 5%.

Genetic analyses. The genetic and environmental etiology of associations between baseline predictors (anxiety/depression, daytime sleepiness, perceived stress, and rumination) and follow-up anxiety/depression was examined using a twin study approach. In the classic twin design, structural equation modeling exploits twin relationships to determine quantitative parameter estimates of genetic and environmental contributions to trait variability (Rijsdijk & Sham, 2002). The model assumes that monozygotic (MZ) twins share 100% of their genetic material, as they result from a single egg fertilized by a single sperm, while dizygotic (DZ) twins result from two eggs fertilized by two sperm, and share, on average, 50% of their genetic material. When twin pairs are raised together, it is possible to determine the impact of environmental influences that are shared by the twins (i.e. same family, school, neighborhood) as well as those that are unique to each twin, such that both MZ and DZ pairs share 100% of shared, or common, environmental influences, while unique environmental influences (including measurement error) are uncorrelated.

A series of bivariate Cholesky decompositions (Neale & Maes, 1998) were used to decompose variance and covariance and, in particular, longitudinal associations between baseline predictors and follow-up anxiety/depression symptoms. Cholesky decomposition enabled the proportion of phenotypic association due to additive genetic (A), common/shared environmental (C), and unique or nonshared environmental (E) influences to be computed (Supplementary Figure S1). Prior to analysis, each variable was regressed on nominally significant covariates (i.e. $p < .05$), as identified in Supplementary Table S1 for the full baseline sample (i.e. age and neighborhood SES for anxiety/depression and perceived stress; sex and age for rumination) and Supplementary Table S2 for the sample at follow-up. Heritability for each variable at baseline and follow-up was derived from an 8-variable Cholesky decomposition. Analyses were conducted in R, with genetic analyses using OpenMx (Boker et al., 2011).

Results

Sample descriptive statistics are shown in Table 1. Note that while group means are similar, the highest scorers for anxiety/depression, perceived stress, and daytime sleepiness did not participate in the study at Time 2 (compare maximum scores for the full baseline sample and Time 1 longitudinal sample). Covariates are assessed in Supplementary Table S1 for variables Time 1 and Supplementary Table S2 for variables at Time 2. For the outcome variable (i.e. anxiety/depression at Time 2), only puberty and sex were nominally significant (Supplementary Table S2). Puberty accounted for approximately 5% of the variance in anxiety/depression at Time 2 (more advanced pubertal status = higher symptom level) and sex for approximately 2% of variance (higher symptom level in girls). Anxiety/depression symptoms were moderately stable from baseline to follow-up ($r = 0.42$, see Supplementary Table S3 for all correlations).

To What Extent Are Perceived Stress, Rumination, and Daytime Sleepiness Associated with Concurrent Measures of Anxiety/Depression? and Do They Have Any Independent Influence?

Perceived stress, rumination, and daytime sleepiness were moderately associated with concurrent symptoms of anxiety/depression at both baseline (Time 1) and follow-up (Time 2), where they accounted for 24% to 33% of variance (Supplementary Tables S4 and S5, Models 1–3), with 3% to 8% of this variance trait-specific.

Specifically, at Time 1, perceived stress is associated with 32% of the variance in anxiety/depression, of which 6% (comparing R^2 for Models 6 and 7, Supplementary Table S4) is independent of rumination and daytime sleepiness (33% and 3% respectively at Time 2, Supplementary Table S5). Rumination at Time 1 is associated with 24% of variance in anxiety/depression, of which 5% (comparing Models 5 and 7, Supplementary Table S4) is independent of perceived stress and daytime sleepiness (28% and 4% respectively at Time 2, Supplementary Table S5). Similarly, at Time 1, daytime sleepiness is associated with 24% of variance in anxiety/depression, of which 6% (comparing Models 4 and 7, Supplementary Table S4) is independent of perceived stress and rumination (33% and 8% respectively at Time 2, Supplementary Table S5).

Do Perceived Stress, Rumination, and Daytime Sleepiness Prospectively Predict Variance in Anxiety/Depression?

These results are shown in Tables 2–4 (Model 1). When assessed as the only predictor, perceived stress predicts approximately 11% of the variance in later anxiety/depression (i.e. $R^2 = .106$), while rumination and daytime sleepiness each predict approximately 8%.

Do Perceived Stress, Rumination, and Daytime Sleepiness Prospectively Predict Change in Anxiety/Depression?

Only perceived stress passes the significance threshold (i.e. $p < .0085$) when concurrent anxiety/depression is included in the model (Tables 2–4, Model 3). Approximately 3% of the variance in anxiety/depression at Time 2 is change predicted by

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Table 1. Raw data descriptive statistics

	Full baseline sample (51.7% boys)			Longitudinal sample (48.4% boys)					
	(*N* ranges 418–422)			Time 1 (*N* ranges 303–304)			Time 2 (*N* ranges 301–304)		
	Mean (*SD*)	Min	Max	***Mean (SD)***	Min	Max	***Mean (SD)***	Min	Max
Anxiety/Depression	9.3 (5.9)	0	33	8.8 (5.8)	0	31	9.7 (6.0)	0	36
Perceived stress	22.5 (12.3)	0	90	21.4 (11.4)	0	65	24.5 (13.1)	0	88
Rumination	9.5 (7.3)	0	39	9.3 (6.9)	0	39	11.9 (8.3)	0	39
Daytime sleepiness	12.0 (5.9)	0	30	11.9 (5.8)	0	26	12.7 (6.1)	0	29
Age in years	11.3 (1.4)	9.0	14.4	11.4 (1.3)	9.0	14.4	13.0 (1.5)	10.1	16.4
Puberty	1.9 (0.6)	1.0	3.8	1.9 (0.6)	1.0	3.8	2.4 (0.7)	1.0	3.8
Neighborhood SES (Decile)	7.1 (2.3)	1	10	7.2 (2.2)	1	10	7.1 (2.2)	1	10

Note: All analyses of baseline (Time 1) data use the full sample unless stated otherwise.

Table 2. Results of linear regression models predicting anxiety/depression symptoms at Time 2 (T2) from perceived stress at Time 1 (T1)

	Output variable: Anxiety/Depression at Time 2					
	Model 1		Model 2		Model 3	
Fixed effects	β (*SE*)	*p* value	β (*SE*)	*p* value	β (*SE*)	*p* value
Perceived stress T1	.323 (.053)	3.79e-09*			.192 (.060)	.0015*
Anxiety/Depression T1			.364 (.052)	1.58e-11*	.269 (.059)	8.37e-06*
	R^2	AIC	R^2	AIC	R^2	AIC
	.106	799.7	.138	792.5	.169	782.2
	Model 4		Model 5			
	β (*SE*)	*p* value	β (*SE*)	*p* value		
Perceived stress T1	.194 (.058)	.00095*	**.194 (.058)**	**.00087***		
Anxiety/Depression T1	.278 (.058)	2.57e-06*	**.287 (.058)**	**1.04e-06***		
Puberty T2	.349 (.078)	1.02e-05*	**.294 (.081)**	**.0003***		
Sex			**-.268 (.111)**	**.016**		
	R^2	AIC	R^2	AIC		
	.226	757.1	**.239**	**753.6**		

Note: Family and zygosity are included as random effects. Model 5 shown in bold type is the best-fitting model. The inclusion of puberty T2 (Model 4) and sex (Model 5) do not result in beta weight drops for perceived stress T1 (i.e. compared to Model 3).
*p values less than the experiment-wide significance threshold ($p < .0085$).

perceived stress that is independent of anxiety/depression at Time 1 (comparing R^2 for Models 2 and 3, Table 2).

While the inclusion of puberty and sex accounts for additional variance and improves model fit (Models 4 and 5), their inclusion does not cause a drop in beta weight for perceived stress as identified in Model 3 (Table 2).

Do Perceived Stress, Rumination, and Daytime Sleepiness Independently Predict Later Anxiety/Depression?

Perceived stress, rumination, and daytime sleepiness together predict approximately 14% of variance in later anxiety/depression symptoms, with both perceived stress and daytime sleepiness accounting for significant independent variance (Table 5, Model 4). Perceived stress independently accounts for 2.6% of variance in later anxiety/depression (R^2 difference between Models 3 and 4, Table 5), while daytime sleepiness independently accounts for 2.1% (comparing Models 1 and 4). However, when concurrent anxiety/depression symptoms were added as a predictor (Table 5, Model 5), neither perceived stress nor daytime sleepiness remained significant. Therefore, while perceived stress as a single trait predicts change in later anxiety/depression symptoms (Table 2, Model 5), it does not do so independently of either daytime sleepiness or rumination at the experiment-wide significance threshold of $p < .0085$. The inclusion of puberty at Time 2 and sex (Table 5, Model 9) improves model fit and takes overall variance accounted for in anxiety/depression at Time 2 to approximately 24%.

Bidirectionality of Influences Across Time

As expected, anxiety/depression, daytime sleepiness, perceived stress, and rumination have significant bidirectional influences with each other (Figure 1, Supplementary Tables S6a–f). Perceived stress has a stronger influence on later anxiety/

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Table 3. Results of linear regression models predicting anxiety/depression symptoms at Time 2 (T2) from rumination at Time 1 (T1)

	Output variable: Anxiety/Depression at Time 2					
	Model 1		Model 2		Model 3	
Fixed effects	β (*SE*)	*p* value	β (*SE*)	*p* value	β (*SE*)	*p* value
Rumination T1	.271 (.053)	6.31e-07*			.126 (.059)	.034
Anxiety/Depression T1			.364 (.052)	1.58e-11*	.304 (.059)	6.19e-07*
	R^2	AIC	R^2	AIC	R^2	AIC
	.076	810.5	.138	792.5	.154	787.9
	Model 4		Model 5			
	β (*SE*)	*p* value	β (*SE*)	*p* value		
Rumination T1	.118 (.057)	.040	**.091 (.058)**	**.114**		
Anxiety/Depression T1	.319 (.058)	8.85e-08*	**.339 (.059)**	**1.8e-08***		
Puberty T2	.347 (.078)	1.41e-05*	**.302 (.082)**	**.00026***		
Sex			**-.227 (.114)**	**.048**		
	R^2	AIC	R^2	AIC		
	.209	763.9	**.218**	**762.3**		

Note: Family and zygosity are included as random effects. Model 5 shown in bold type is the best-fitting model. The inclusion of puberty T2 (Model 4) and sex (Model 5) results in beta weight drops for rumination T1, thus reflecting overlapping variance.
**p* values less than the experiment-wide significance threshold ($p < .0085$).

Table 4. Results of linear regression models predicting anxiety/depression symptoms at Time 2 (T2) from daytime sleepiness at Time 1 (T1)

	Output variable: Anxiety/Depression at Time 2					
	Model 1		Model 2		Model 3	
Fixed effects	β (SE)	*p* value	β (*SE*)	*p* value	β (SE)	*p* value
Daytime sleepiness T1	.271 (.051)	2.15e-07*			.143 (.069)	.011
Anxiety/Depression T1			.364 (.052)	1.58e-11*	.287 (.058)	1.08e-06*
	R^2	AIC	R^2	AIC	R^2	AIC
	.078	796.8	.138	792.5	.155	775.6
	Model 4		Model 5			
	β (SE)	*p* value	β (SE)	*p* value		
Daytime sleepiness T1	.122 (.055)	.027	**.121 (.055)**	**.028**		
Anxiety/Depression T1	.309 (.057)	1.05e-07*	**.318 (.057)**	**4.08e-08***		
Puberty T2	.321 (.077)	4.40e-05*	**.273 (.080)**	**.00076***		
Sex			**-.229 (.110)**	**.038**		
	R^2	AIC	R^2	AIC		
	.205	753.7	**.217**	**749.4**		

Note: Family and zygosity are included as random effects. Model 5 shown in bold type is the best-fitting model. The inclusion of puberty T2 (Model 4) results in a beta weight drop for daytime sleepiness T1, thus reflecting overlapping variance.
**p* values less than the experiment-wide significance threshold ($p < .0085$).

depression than vice versa (i.e. perceived stress at Time 1 accounted for 11% of variance in anxiety/depression at Time 2, while anxiety/depression at Time 1 accounted for 5% of variance in perceived stress at Time 2, Supplementary Table S6a, Models 1 and 4) and similarly for daytime sleepiness (8% vs. 3%, Supplementary Table S6c, Models 1 and 4). In contrast, levels of association between anxiety/depression and rumination over time were similar in both directions (8% vs. 7%, Supplementary Table S6b, Models 1 and 4). Bidirectional associations over time between perceived stress and rumination (7% vs. 12%, Supplementary Table S6d, Models 1 and 4), perceived stress and daytime sleepiness (9% vs. 6% Supplementary Table S6e, Models 1 and 4), and rumination and daytime sleepiness (7% vs. 7%, Supplementary Table S6f, Models 1 and 4) were of similar magnitude.

Genetic and Environmental Influence on Total Variation

While sample size is a limiting factor for our genetic analyses, we can nonetheless make some observations. In our adolescent sample, common environment and/or additive genetic sources account

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Table 5. Results of linear regression models predicting anxiety/depression symptoms at Time 2 (T2) from perceived stress, rumination, and daytime sleepiness at Time 1 (T1)

	Output Variable: Anxiety/Depression at Time 2					
	Model 1		Model 2		Model 3	
Fixed effects	β (*SE*)	*p* value	β (*SE*)	*p* value	β (*SE*)	*p* value
Perceived stress T1	.255 (.066)	.00013*	.242 (.057)	2.81e-05*		
Rumination T1	.132 (.067)	.050			.200 (.057)	.00060*
Daytime sleepiness T1			.184 (.056)	.0011*	.215 (.055)	.00013*
	R^2	AIC	R^2	AIC	R^2	AIC
	.122	797.9	.134	779.8	.117	785.8
	Model 4		Model 5		Model 6	
	β (*SE*)	*p* value	β (*SE*)	*p* value	β (*SE*)	*p* value
Perceived stress T1	.191 (.066)	.0039*	.138 (.067)	.039	.171 (.067)	.012
Rumination T1	.100 (.066)	.128	.047 (.067)	.485	.055 (.069)	.427
Daytime sleepiness T1	.172 (.056)	.0026*	.011 (.059)	.066		
Anxiety/Depression T1			.215 (.064)	.00092*	.256 (.062)	4.78e-05*
	R^2	AIC	R^2	AIC	R^2	AIC
	.143	779.5	.177	770.7	.173	783.5
	Model 7		Model 8		Model 9	
	β (*SE*)	*p* value	β (*SE*)	*p* value	β (SE)	*p* value
Perceived stress T1	.158 (.060)	.0096	.150 (.065)	.022	**.165 (.065)**	**.011**
Rumination T1			.040 (.065)	.537	**.009 (.066)**	**.896**
Daytime sleepiness T1	.111 (.059)	.060	.089 (.058)	.125	**.087 (.057)**	**.130**
Anxiety/Depression T1	.2226 (.062)	.00033*	.233 (.063)	.00025*	**.250 (.063)**	**9.25e-05***
Puberty T2			.320 (.077)	3.96e-05*	**.271 (.080)**	**.00080***
Sex					**-.240 (.112)**	**.032**
	R^2	AIC	R^2	AIC	R^2	AIC
	.175	769.1	.228	747.9	**.237**	**743.5**

Note: Family and zygosity are included as random effects in Models 1–8. Model 9 was overfitted if both family and zygosity were included and so was run with only family included as a random effect. Model 9 shown in bold type is the best-fitting model.
*p values less than the experiment-wide significance threshold ($p < .0085$).

for approximately a third to half of the total variance in all traits (Figure 2, Supplementary Table S7). Remaining variance was influenced by unique environmental sources, which may include measurement error. Genetic sources of influence were significant for baseline anxiety/depression and perceived stress, rumination at follow-up, and for daytime sleepiness at both time points. Common environment had a significant influence on perceived stress and rumination at both time points, as well as anxiety/depression symptoms at follow-up. However, confidence intervals were broad for all significant additive genetic and common environmental influences, with the lower bound close to zero for many (Supplementary Table S7). Unique environmental influences were greater at baseline than follow-up for all traits, although confidence intervals overlap.

To What Extent Do Genetic and Environmental Factors Account for Associations Between Time 1 Predictors (Anxiety/Depression, Perceived Stress, Rumination, Daytime Sleepiness) and Symptoms of Anxiety/Depression at Time 2?

Phenotypic correlations between the Time 1 predictor variables and anxiety/depression at Time 2, as derived from bivariate

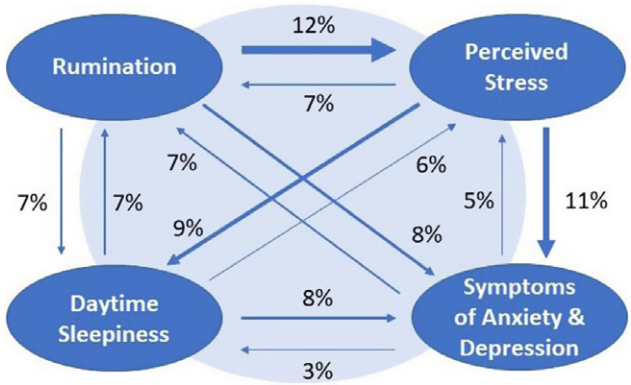

Fig. 1. Bidirectional influences over time, showing percentage of variance accounted for in the outcome variable at Time 2. For example, rumination at Time 1 accounts for 12% of variance in perceived stress at Time 2 (while perceived stress at Time 1 accounts for 7% of variance in rumination at Time 2). Each percentage represents the R^2 identified in a series of linear regressions (Supplementary Tables S6a–f, Models 1 and 4).

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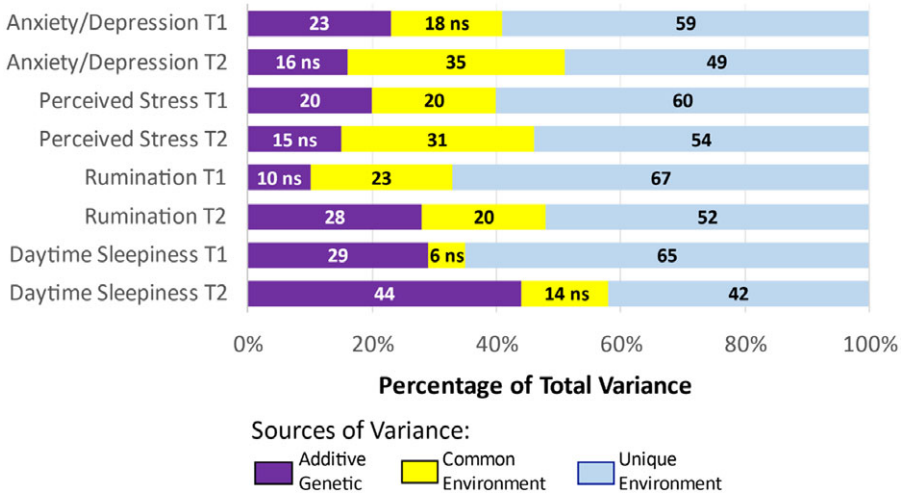

Fig. 2. Additive genetic (A), common environmental (C), and unique environmental influences on traits at Time 1 (T1) and Time 2 (T2) are shown as a percentage of total variance. Estimates are derived from multivariate analyses including all eight variables and using the full sample at Time 1. For results in the same sample at both timepoints, see Supplementary Figure S2 — this allows for better comparison, but using the larger Time 1 sample provides more accurate results overall. Nonsignificant estimates are notated 'ns'. 95% confidence intervals are shown in Supplementary Table S7.

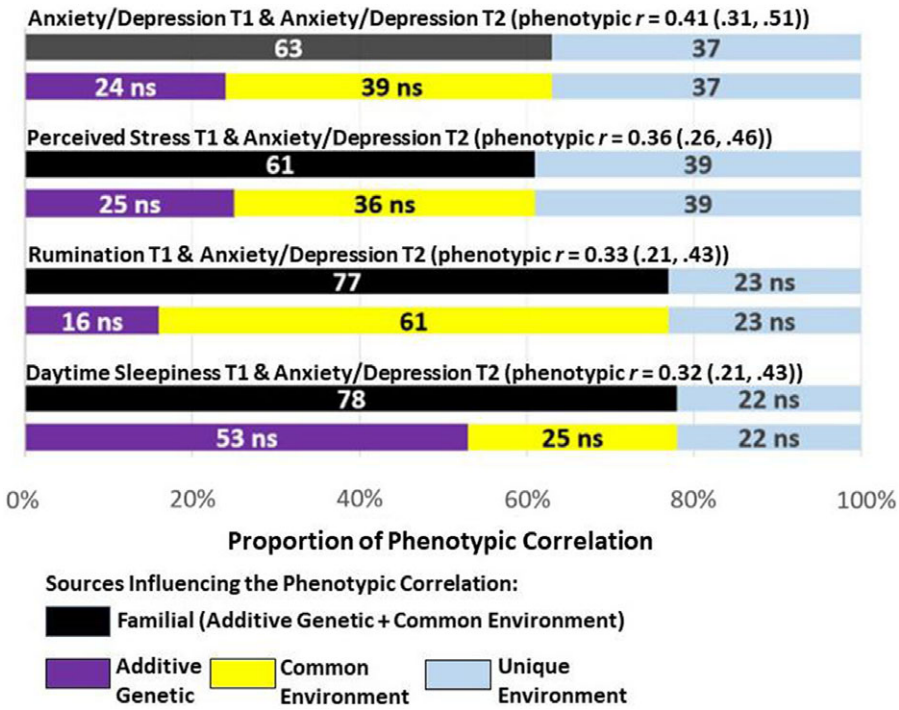

Fig. 3. Additive genetic (A), common environmental (C), unique environmental, and familial (A + C) contributions to phenotypic associations between Time 1 (T1) predictors and anxiety/depression symptoms at Time 2 (T2). Nonsignificant estimates are notated 'ns'. 95% confidence intervals are shown in Supplementary Table S8.

Cholesky decomposition, range from 0.32 to 0.41 (Figure 3, Supplementary Table S8). These associations are largely influenced by familial sources (i.e. genetic predisposition and environments that are shared by the twins, such as their home and school environment), which in total account for 61% to 78% of the associations. Analyses are not sufficiently sensitive to significantly distinguish between additive genetic and common environmental influences, excepting the association between Time 1 rumination and anxiety/depression at Time 2, where common environment accounted for 61% of the phenotypic correlation.

Discussion

Anxiety and depression are complex traits, with adolescence being a core risk period for the emergence of symptoms. Underlying mechanisms may also differ from those in adults (Beesdo et al., 2009; Hazell, 2021; Hunter & McEwen, 2013; Schmaal, Hibar et al., 2017; Schmaal, Yucel et al., 2017). Notable risk factors for adolescent anxiety/depression include current life stressors, negative thought processes such as rumination, and sleep-related problems (Blake et al., 2018; Palmer et al., 2018). Here we show that prospectively, perceived stress to everyday events, rumination, and daytime sleepiness account for variation in later anxiety/depression symptoms in young adolescents. However, only perceived stress predicted change in anxiety/depression (i.e. after accounting for anxiety/depression at baseline) although it did not do so independently of rumination and sleepiness, reflecting the considerable overlap among these traits. Familial influences (i.e. additive genetic and/or shared environment) accounted for approximately two-thirds or more of the association between baseline predictor and follow-up anxiety/depression, with shared environment being a prominent component of the association between baseline rumination and follow-up anxiety/depression.

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Bidirectional influences were found between all the traits of interest, with the strongest associations over time being for baseline rumination on follow-up perceived stress, and baseline perceived stress on follow-up self-reported symptoms of anxiety/depression. Bidirectional influences involving daytime sleepiness were strongest in the direction of baseline perceived stress on follow-up sleepiness, with baseline sleepiness accounting for a similar amount of variation in follow-up anxiety/depression.

Our results are consistent with studies suggesting that rumination may mediate associations between stressful life events and symptoms of anxiety and depression in adolescents (Hamilton et al., 2015; Hosseinichimeh et al., 2018; Michl et al., 2013; Skitch & Abela, 2008). Hosseinichimeh et al. (2018), using a system dynamics simulation model approach in 661 young adolescents, determined that rumination contributes to depression by keeping past stressful experiences 'alive', which in turn feeds back to stimulate more rumination.

The current study suggests that rumination may also exacerbate responses to everyday stressors (as opposed to keeping stressful *life events* 'alive') — thereby contributing to the maintenance of symptoms of anxiety and depression in adolescents. The daily life stressors scale used in this study reflects, in part, an individual's perceived control over their environment (Kearney et al., 1993). Rumination may play a role in enhancing perceptions that environmental pressures are beyond the individual's capacity to cope, thereby increasing the levels of perceived stress and risk for anxiety and depression.

Daytime sleepiness adds another level of complexity to relationships between perceived stress, rumination, and anxiety/depression. Daytime sleepiness is a common problem among adolescents and is considered to be a clinical marker of adolescent sleep problems (Gradisar et al., 2011). Importantly, adolescence is a vulnerable time for sleep-related problems. Sleep regulatory and other brain systems (e.g. the 'social' brain) are still maturing, while psychosocial and societal pressures are increasing (Andrews et al., 2021; Crowley et al., 2018; Kuula et al., 2018). Indeed, these factors are proposed to culminate in a 'perfect storm' that may heighten adolescent risk for developing mental health problems (Carskadon, 2011; Crowley et al., 2018).

Studies specific to daytime sleepiness, especially longitudinal studies in adolescents, are few compared to those exploring other sleep parameters. Daytime sleepiness is an important sleep measure that may provide novel insights in addition to those obtained from standard behavioral sleep parameters (Hong et al., 2021), although it has been associated with other measures of sleep perception such as sleep quality and insomnia (O'Callaghan et al., 2021). Previous longitudinal studies in high-school students have reported reciprocal relationships between daytime sleepiness and both anxiety and depression, as well as perceived study stress predicting later sleepiness ($N = 2787$, Luo et al., 2018), and reciprocal associations between sleep disturbances, including daytime sleepiness, and both negative mood and rumination ($N = 350$, Yip et al., 2022). In addition, relatively large cross-sectional studies ($N > 1000$) have reported associations between daytime sleepiness and perceived psychological stress in high-school students (Chung & Cheung, 2008; Merdad et al., 2014).

Focusing on anxiety/depression at follow-up as an outcome variable, our analyses indicate that familial influences (i.e. additive genetic and common, or shared, environmental influences) account for most of the phenotypic associations with baseline measures of anxiety/depression, perceived stress, rumination, and daytime sleepiness. But we lack power to disentangle additive genetic and common environmental influences. However, common environment accounts for most of the association between baseline rumination and follow-up anxiety/depression. Common environmental factors are those shared by cotwins, and those associated with rumination may include aspects of family functioning and shared peer group influences and lifestyle factors such as sports participation. For example, parenting styles characterized by high control and protectiveness have been identified as risk factors for the development of ruminative brooding (Manfredi et al., 2011), while higher levels of brooding are found in adolescents who report having more friends who use alcohol (Hilt, Armstrong et al., 2017). Further, a study among young adults has posited that exercise may be a buffer against difficulties with emotion regulation, with a less ruminative response style found for those who exercise regularly (Bernstein & McNally, 2018).

To estimate genetic and environmental influences for each trait, multivariate Cholesky decomposition of all eight variables (four traits by two timepoints) was conducted. Multivariate analysis has the benefit of increased statistical power to detect effects that are correlated across measures (Schmitz et al., 1998). Even so, power was hampered by sample size and confidence intervals were broad, particularly for additive genetic and common environmental estimates. Overall, daytime sleepiness was the most genetic of the traits, with genes accounting for 44% of variance at follow-up. In contrast, we found relatively strong common environmental influences for anxiety/depression at follow-up (accounting for approximately a third of variance) and at both time points for perceived stress and rumination. This likely reflects the correlated nature of common environmental factors on these traits, which would enhance the power of the multivariate analysis to detect them (Schmitz et al., 1998).

While our results are broadly consistent with prior work examining anxiety/depression (Nivard et al., 2015; Zheng et al., 2016) and rumination (Chen & Li, 2013; Moore et al., 2013), previous adolescent twin studies of anxiety and depression symptoms have reported some inconsistencies, particularly in relation to common environmental sources of influence. For example, in a study of symptoms of anxiety and depression, Nivard et al. (2015) reported common environmental influences accounting for 11% of variance (37% for additive genetic influences) in Dutch adolescents aged 12 years ($N > 1000$), but dropping to 0% at age 14 (51% for additive genetic influences), while considerably higher common environmental influences (ranging 20–60%) have been reported for 712 Chinese adolescents aged 10 to 12 years (Zheng et al., 2016). It is plausible that common environmental influences on anxiety and depression symptoms may be differentially influenced by cultural practices (e.g. Pinquart & Kauser, 2018).

To our knowledge, no twin studies examining the heritability of perceived stress have been specific to adolescents. However, studies in adolescents and/or young adults report higher heritability than found in the current study and no indication of common environmental influence (Michalski et al., 2017; Rietschel et al., 2017). This is to be expected, as common environmental factors necessarily become less influential as individuals become more independent as they transition to adulthood. Similarly, no independent twin studies of daytime sleepiness specific to adolescents older than 8 years of age were found. Breitenstein et al. (2021) examined daytime sleepiness in children aged 8 and found low heritability (accounting for 27% of variance) and strong common environmental influences (accounting for 66% of variance). In the current study, with older adolescents, common environmental influences were considerably smaller and nonsignificant. Our results are consistent with those found in large adult twin studies, where little or

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no common environmental influence is found (Desai et al., 2004; Watson et al., 2006).

One possible pathway for genetic and environmental influences on the traits of interest may be through their effects on HPA axis regulation. The HPA axis is a major modulator of responses to external and internal stimuli, including psychological stressors, and alterations in this system have been robustly linked to psychiatric illness, including anxiety and depression (Juruena et al., 2020; Murphy et al., 2022). Indeed, most depression-related genes identified in replicable gene × environment interactions have been connected to the HPA axis and stress regulation (Gonda et al., 2019; Starr & Huang, 2019). In addition, daytime sleepiness has been associated with a blunting of the cortisol response, which is a marker of HPA axis activity (see review: van Dalfsen & Markus, 2018). However, associations between rumination, or brooding, and cortisol response have been inconsistent (Hilt, Sladek, et al., 2017; Katz et al., 2019; Rnic et al., 2022; van Santen et al., 2011).

Another mechanism affecting the predictor traits and impacting adolescent mental health may be maturational changes in the circadian timing system during adolescence. Adolescents experience a delayed shift in their sleep onset and offset times that may clash with societal expectations and pressures, leading to daytime sleepiness and reduced capacity to cope with stressors, increased propensity to ruminate, and, ultimately, greater risk of experiencing symptoms of anxiety and depression (Carpenter et al., 2021; Carskadon, 2011; Crowley et al., 2018; Owens et al., 2016). Consistent with this, circadian clock genes have been posited as a potential nexus for sleep and mood regulation in adolescents (Blake et al., 2018; Dueck et al., 2015). Further, the HPA axis and mammalian clock gene systems interact, such that stress may regulate clock gene levels (Bolsius et al., 2021; Razzoli et al., 2014) and several clock genes are expressed in brain regions implicated in emotion regulation (Kim et al., 2017; Mendoza & Vanotti, 2019; Patton & Hastings, 2018).

The current study has some limitations. Sample size limits our ability to disentangle genetic and environmental influences, such that additive genetic and common environmental estimates have broad confidence intervals, with lower bounds generally close to zero. Our measures of wellbeing were obtained from self-report questionnaires, and trajectories of change in adolescents may differ for self-report compared to diagnostic interview (e.g. Long et al., 2020). In addition, families with higher socioeconomic status are somewhat over-represented in the QTAB cohort (Strike et al., 2022) and results may not best represent individuals in a low socioeconomic environment. Further, this work identifies a relatively small (though important) proportion of factors influencing individual variation in adolescent anxiety/depression symptoms. The problem is a complex one, and diverse approaches are needed to extend our understanding of the underlying issues and to address these problems.

In conclusion, we speculate that HPA axis-related dysfunction and circadian rhythm disturbances (as evidenced by perceived stress, rumination, and daytime sleepiness) may be key drivers in the persistence of anxiety/depression symptoms in young adolescents. However, co-opting these systems in the treatment of psychiatric conditions remains a challenge. Nonetheless, HPA axis genes found to regulate stress responses have been identified as possible drug targets for depression and stress disorders, although many uncertainties remain (Dunlop & Wong, 2019; Gonda et al., 2019) and researchers are exploring the potential of plants and phytonutrients to moderate HPA axis activity (Lopresti et al., 2021). In addition, personalized circadian-targeted therapies for adolescents and young adults with depression and circadian dysregulation have recently been proposed as an enhanced model of care (Crouse et al., 2021). Perceived stress, rumination, and daytime sleepiness are easily assessed measures that may hint at underlying HPA axis and circadian rhythm dysregulation and may be valuable markers, in addition to anxiety and depression, for assessing the success of HPA axis and circadian-related interventions. Alternatively, directly addressing perceived stress, rumination, and daytime sleepiness (e.g. Murray et al., 2022; Pincus & Friedman, 2004; Puolakanaho et al., 2019; Wassenaar et al., 2019; Watkins & Roberts, 2020) may help to regulate HPA axis and circadian system function and ultimately reduce symptoms of anxiety and depression.

Supplementary Material. To view supplementary material for this article, please visit https://doi.org/10.1017/thg.2022.26.

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Conflict of Interest. None.

Ethical Standards. Children's Health Queensland HREC Reference Number, HREC/16/QRCH/270.

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