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DOI:

[10.1002/da.22326](https://doi.org/10.1002/da.22326)

Document Version

Publisher's PDF, also known as Version of record

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Citation for published version (APA):

Waszczuk, M., Zavos, H., Antonova, E., Haworth, C., Plomin, R., & Eley, T. (2015). A multivariate twin study of trait mindfulness, depressive symptoms and anxiety sensitivity. *Depression and Anxiety*, 32(4), 254–261.
<https://doi.org/10.1002/da.22326>

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Research Article

A MULTIVARIATE TWIN STUDY OF TRAIT MINDFULNESS, DEPRESSIVE SYMPTOMS, AND ANXIETY SENSITIVITY

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Background: Mindfulness-based therapies have been shown to be effective in treating depression and reducing cognitive biases. Anxiety sensitivity is one cognitive bias that may play a role in the association between mindfulness and depressive symptoms. It refers to an enhanced sensitivity toward symptoms of anxiety, with a belief that these are harmful. Currently, little is known about the mechanisms underpinning the association between mindfulness, depression, and anxiety sensitivity. The aim of this study was to examine the role of genetic and environmental factors in trait mindfulness, and its genetic and environmental overlap with depressive symptoms and anxiety sensitivity. **Methods:** Over 2,100 16-year-old twins from a population-based study rated their mindfulness, depressive symptoms, and anxiety sensitivity. **Results:** Twin modeling analyses revealed that mindfulness is 32% heritable and 66% due to nonshared environmental factors, with no significant influence of shared environment. Genetic influences explained over half of the moderate phenotypic associations between low mindfulness, depressive symptoms, and anxiety sensitivity. About two-thirds of genetic influences and almost all nonshared environmental influences on mindfulness were independent of depression and anxiety sensitivity. **Conclusions:** This is the first study to show that both genes and environment play an important role in the etiology of mindfulness in adolescence. Future research should identify the specific environmental factors that influence trait mindfulness during development to inform targeted treatment and resilience interventions. Shared genetic liability underpinning the co-occurrence of low mindfulness, depression, and anxiety sensitivity suggests that the biological pathways shared between these traits should also be examined. *Depression and Anxiety* 32:254–261, 2015. ©

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Key words: mindfulness; depression; anxiety sensitivity; twins; genetics; environment; attention

INTRODUCTION

Mindfulness-based therapies have been found to be effective in treating internalizing disorders,^[1–4] reducing both the symptoms of anxiety and depression, and

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Contract grant sponsor: United Kingdom Medical Research Council; Contract grant numbers: G1100559, G0901245, and G0500079; Contract grant sponsor: Leverhulme Foundation, Contract grant number: RPG-210.

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Received for publication 23 May 2014; Revised 27 August 2014; Accepted 29 September 2014

DOI 10.1002/da.22326

Published online 30 January 2015 in Wiley Online Library (wileyonlinelibrary.com).

the cognitive biases that play a central role in the etiology and maintenance of these problems.^[5] Given the marked increase in depression prevalence during adolescence^[6] and the plasticity during this period of brain maturation,^[7,8] there is a growing interest in the application of mindfulness-based approaches in young people.^[9]

Mindfulness refers to a wide range of constructs and can be studied as a psychological trait, with individuals differing in their dispositional level of mindfulness, and as a clinical intervention that aims to increase mindfulness for therapeutic purpose (e.g., mindfulness-based cognitive therapy). At its core trait mindfulness is characterized by nonjudgmental awareness of the present moment experience that is beneficial to psychological well-being.^[10,11] It can therefore be defined both at an attentional level (awareness of the present moment) and the interpretation level (nonjudgmental and with acceptance).^[12,13] Thus, mindfulness may influence cognitive processes at multiple stages, reducing attentional control deficits and negative cognitive styles that are central to mood disorders.^[5,14–17] Measures of trait mindfulness tend to focus on the attentional processes, allowing investigations into whether improved attention control is one of the cognitive mechanisms that might explain this association.

Relatively little is known about the genetic and environmental influences on mindfulness. Individual differences in complex traits such as mindfulness are presumed to have arisen through an interaction of inherited predisposition and environmental circumstances, such as explicit training.^[18] However, despite the clinical importance of trait mindfulness, the relative importance of genes, shared environment, and individual-specific experiences is unknown. It also remains to be investigated whether there are any differences in genetic or environmental influences on mindfulness between males and females.

Anxiety sensitivity is one cognitive bias that may play an important role in the association between mindfulness and depression. Anxiety sensitivity refers to an enhanced sensitivity (attentional bias) toward symptoms of anxiety, such as heart palpitations or worry, with a belief that these are harmful (interpretational bias).^[19] Anxiety sensitivity is independently associated with both anxiety and depression across development.^[20–22] Initial evidence suggests that one way trait mindfulness might benefit patients' well-being is by reducing the impact of anxiety sensitivity on their emotional distress.^[23] Reduction of the cognitive biases such as anxiety sensitivity might be one of the cognitive mechanisms that explain the inverse association between mindfulness and depression. Individuals who score high on anxiety sensitivity have been found to exhibit less mindfulness, specifically showing difficulties with limiting attention to the current activity (attentional processing), and with experiencing the present state without evaluating or judging its content (interpretation).^[23–25] This supports the view that mindfulness may be associated with reduction in

cognitive biases such as anxiety sensitivity at both attentional and interpretational processing levels, which in turn might reduce internalizing symptoms.^[26,27]

Examining genetic and environmental influences on the joint associations between trait mindfulness, depression, and anxiety sensitivity can help to clarify some of the mechanisms that underpin these relationships. One possibility may be that mindfulness, depression, and anxiety sensitivity share genetic influences. It is very common for traits that co-vary to have largely similar genetic factors that account for their co-occurrence, while nonshared environmental factors are generally smaller (the “generalist genes hypothesis”).^[28] We have previously shown that depressive symptoms and anxiety sensitivity share high and significant genetic correlations across development.^[20,29] However, mindfulness is associated with a range of other traits, for example, self-esteem, physical well-being, and personality traits such as conscientiousness, agreeableness, and openness to experience.^[10,30] Thus, genetic influences on mindfulness may be largely distinct from the ones influencing internalizing problems. Instead, environmental influences such as parenting or life events, may explain the relationship between mindfulness, depression, and anxiety sensitivity. Investigating the role of genes and environment in the relationship between mindfulness, depression, and anxiety sensitivity will help to understand the relative role of the biological and social mechanisms that link these traits.

The aim of the current study was to investigate the genetic and environmental influences on mindfulness, as well as on its associations with depressive symptoms and anxiety sensitivity. The current study focused specifically on the attentional control aspect of trait mindfulness.^[31] Using a large epidemiological sample of 16-year-old twins, we first investigated the phenotypic correlations between trait mindfulness, depressive symptoms, and anxiety sensitivity. Second, we explored what proportion of variance in mindfulness was accounted for by genetic and environmental influences, and whether any sex differences in these influences were evident. Third, in order to understand the association of low mindfulness with depressive symptoms and anxiety sensitivity, we investigated genetic and environmental correlations shared between these traits. We hypothesized that our results would be in line with the “generalist genes hypothesis,”^[28] resulting in high genetic and moderate environmental correlations. Finally, we were interested in the proportion of genetic and environmental influences on mindfulness not shared with depressive symptoms and anxiety sensitivity.

METHODS

PARTICIPANTS

The analyses use data from Twins Early Development Study (TEDS), a large epidemiological study of over 10,000 twin pairs born in England and Wales in 1994, 1995, and 1996. Full recruitment details are provided elsewhere.^[32] The current analyses focus on the data

collected when twins were approximately 16 years old (mean age = 16.32, SD = .68 years). Informed consent was obtained from parents of all participating adolescents and the study was approved by the Institute of Psychiatry Ethics Committee. Zygosity was established using parent-report questionnaires of physical similarity, which is estimated to be 95% accurate when compared to DNA testing.^[33] Where zygosity was ambiguous, DNA testing was conducted. The questionnaire booklets were returned by 10,320 individuals (55.51% female; 35.59% monozygotic (MZ), 32.51% same-sex dizygotic (DZ), 31.90% opposite-sex DZ twins). Participants were excluded if they did not provide consent, if they had severe medical disorders, experienced severe perinatal complications, or if their zygosity was unknown ($N = 316$ families). The sample size, internal consistencies, and descriptive statistics of all measures are presented in Table 1.

MEASURES

Mindfulness. Mindfulness was measured using a short version^[31] of the Mindful Attention Awareness Scale (MAAS);^[10] a 5-item self-report questionnaire focusing on statements relating attentional control (e.g., “I find myself doing things without paying attention”). Psychometric studies corroborate the utility of the shortened version of the MAAS scale.^[31,34] Responses were summed to give total trait mindfulness scores; higher total scores reflect lower mindfulness.

Depression. Depressive symptoms were measured using the Short Mood and Feelings Questionnaire;^[35] a 13-item self-report measure assessing how often depressive symptoms occurred in the past 2 weeks. Responses were summed to give total depressive symptoms scores. The measure demonstrates good reliability and validity.^[35]

Anxiety sensitivity. Anxiety sensitivity was assessed using the Children’s Anxiety Sensitivity Index;^[36] an 18-item self-report questionnaire assessing fear of anxiety sensations (e.g., “It scares me when my heart beats fast”). The measure has sound psychometric properties.^[36,37] Responses were summed to give total anxiety sensitivity scores.

ANALYSES

The twin design compares the degree of similarity between MZ (sharing 100% of their genes) and DZ (sharing on average 50% of their segregating genes) twin pairs. These relative differences in within-pair correlations allow estimations of the influences caused by additive genetics (A), shared environment (C), and nonshared environment (E). Where correlations are higher for MZ twins as compared to DZ pairs, genetic influence is assumed to be playing a role. Within-pair similarity that is not due to genetic factors is accounted for by shared environ-

mental influences (C), which contribute to the resemblance between family members. C is evident when DZ correlations are more than half MZ correlations. Nonshared environment (E) accounts for individual-specific factors that create differences among siblings from the same family. These are estimated from within-pair differences between MZ twins. Any measurement error present is included in this term. Quantitative genetic designs and methods are described comprehensively elsewhere.^[38]

All twin analyses were conducted using OpenMx^[39] within R (www.R-project.org),^[40] a structural equation modeling package for the analysis of genetically informative data that controls for nonindependence of family members. As is standard in model fitting analysis, the variables were regressed for age and sex,^[41] and were mapped onto a standard normal distribution using the rank-based van der Waerden’s transformation to correct for skew.

All models were fitted using raw data maximum likelihood. The core fit statistic was minus twice the log likelihood ($-2LL$) of the observations. This is not an overall measure of fit, but provides a relative measure of fit, since differences in $-2LL$ between models are distributed as χ^2 . Therefore, to examine the overall fit of the genetic model we compared the $-2LL$ to that of a saturated model (one which fully describes data using the maximum number of free parameters, estimating variances, covariances, and means for the raw data to get a baseline index of fit). The fit of each submodel was assessed by χ^2 difference tests, Akaike’s and Bayesian’s information criterion ($AIC = \chi^2 - 2df$; $BIC = \chi^2 - k \ln(n)$) with lower χ^2 values, and more negative AIC and BIC values suggesting a better fit. If the difference between the AIC of two models was less than 10, the more parsimonious model was selected.^[42] For all analyses, we also compared models with fewer parameters to the full A , C , and E correlated factors solution.

Univariate analyses assessing the influences of A , C , and E were conducted on all variables. Sex differences were examined to inform twin modeling. Qualitative sex differences were tested to see whether the same genetic and environmental sources contribute to individual differences in the phenotype for males and females. Second, quantitative sex differences were tested, where the same genetic and environmental sources operate, but they influence the phenotype in males and females to different degree. Third, we tested scalar sex differences, to investigate whether there is a scalar variance difference between males and females.

The Cholesky decomposition, represented as a multivariate correlated factors solution (Fig. 1a), was used to examine the genetic and environmental relationship between mindfulness, depressive symptoms, and anxiety sensitivity. The correlated factors solution assumes that each variable has unique A , C , and E influences, and that these trait-specific influences can be correlated with the A , C , and E influences

TABLE 1. Descriptive statistics, cross twin correlations, and univariate results

	Descriptive statistics				Cross twin correlations		Univariate influences		
	N (individuals)	Mean (SD), range	Skew	α	r_{MZ}	r_{DZ}	A	C	E
Mindfulness	2,118	8.96 (4.36), 0–23	−0.06	.76	.36 (.29–.42)	.14 (.10–.20)	.32 (.14–.41)	.02 (.00–.15)	.66 (.59–.74)
Depression	9,609	3.61 (4.41), 0–26	1.95	.88	.42 (.39–.45)	.21 (.20–.24)	.29 (.19–.38)	.12 (.05–.20)	.59 (.55–.63)
Anxiety sensitivity	9,608	7.95 (5.86), 0–36	1.14	.86	.46 (.43–.49)	.18 (.16–.20)	.36 (.27–.43)	.05 (.00–.12)	.59 (.56–.63)

Note: SD, standard deviation; α , internal consistency; MZ, monozygotic; DZ, dizygotic; A , additive genetic parameters; C , shared environmental parameters; E , nonshared environmental parameters.

Descriptive statistics and cross twin correlations are presented on untransformed and unregressed variables for comparison with other published samples. Univariate analyses are presented on transformed variables. 95% Confidence intervals (CIs) are presented in brackets. CIs not including 0 indicate significant estimates. Nonoverlapping CIs mean significant difference between the values. Some of the DZ correlations are less than half MZ correlations, suggesting that A should be interpreted as both additive and dominant genetic effects. Mindfulness was measured only in a subset of twins (a cohort born between January 1994 and August 1994), while depression and anxiety sensitivity was measured in the whole sample, resulting in larger sample sizes. All twins were approximately 16 years old at the time of data collection.

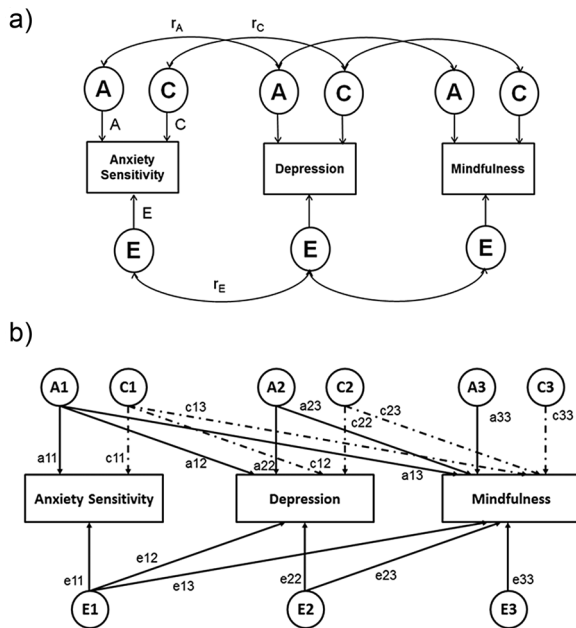


Figure 1. (a) Correlated factors solution; (b) Cholesky decomposition (attached separately).

A , additive genetic parameters; C , shared environmental parameters; E , non shared environmental parameters; r_A , genetic correlation; r_C , shared environmental correlation; r_E , nonshared environmental correlation; $a11$ – $a33$, genetic influences; $c11$ – $c33$, shared environmental influences; $e11$ – $e33$, nonshared environmental influences.

on other traits (r_A = genetic correlation, r_C = shared environmental correlation, and r_E = nonshared environmental correlation). The proportions of the phenotypic correlations accounted for by A , C , and E influences were also calculated. The data were additionally interpreted as the Cholesky decomposition (Fig. 1b), which assumes three distinct sets of genetic and environmental influences on each variable. $A1$, $C1$, and $E1$ are common factors influences on the first variable via paths $a11$, $c11$, and $e11$ that can also influence the remaining two variables via paths $a12$, $a13$, $c12$, $c13$, $e12$, and $e13$. $A2$, $C2$, and $E2$ influence the second variable via paths $a22$, $c22$, and $e22$ and can also influence the third variable via paths $a23$, $c23$, and $e23$, over and above the influences accounted for by $A1$, $C1$, and $E1$. $A3$, $C3$, and $E3$ are specific influences unique to the third variable only (via paths $a33$, $c33$, and $e33$). Total A , C , and E effects on each individual measure can be obtained by summing all paths to that measure (e.g., total genetic influences on the third variable can be obtained by adding influences from paths $a13$, $a23$, and $a33$). Although any ordering of the variables explains the variance–covariance matrix between variables equally well, mindfulness was placed as the last variable as the aim was to investigate whether there are any specific genetic or environmental influences on mindfulness, over and above those shared with depressive symptoms and anxiety sensitivity. Due to interpretational constraints,^[43] only the specific influences on mindfulness are presented and interpreted (paths $a33$, $c33$, and $e33$).

RESULTS

The descriptive statistics for all measures, such as means, standard deviations, and skew, are presented in Table 1. Females scored significantly higher on all

scales than males (mindfulness: $t(2116) = 3.22$, $P < .05$, $d = .14$; depression: $t(9607) = 19.96$, $P < .05$, $d = .41$; anxiety sensitivity: $t(9606) = 29.06$, $P < .05$, $d = .59$), suggesting that on average females had lower mindfulness and higher depressive symptoms and anxiety sensitivity than males (Supporting Information Table A1). Low mindfulness was moderately correlated with depressive symptoms and anxiety sensitivity ($r_{pb} = .34$ with both, Table 2). Depressive symptoms and anxiety sensitivity were also correlated ($r_{pb} = .48$, Table 2). Of note, some of the DZ correlations were less than half MZ correlations (Tables 1 and 2), suggesting that A should be interpreted as both additive and dominant genetic effects.

Univariate twin modeling results (Table 1) revealed that mindfulness was moderately influenced by genetic ($A = .32$) and nonshared environmental influences ($E = .66$), with no significant influence of shared environment. A similar pattern was found for genetic and environmental influences on depressive symptoms and anxiety sensitivity ($A = .29$ and $.36$, respectively, $E = .59$ for both). At the univariate level, scalar (variance) sex differences were found for both depressive symptoms and anxiety sensitivity. In addition, quantitative sex differences were evident for depression. There were no qualitative sex differences in our sample and these sex differences were not modeled further. At the multivariate level, first a model allowing for quantitative sex differences was fitted, followed by a scalar multivariate model where estimates were equated across sex, with a scalar variable modeled to account for the variance difference in depression and anxiety sensitivity. As estimates were similar for males and females (see Supporting Information Table A2), the scalar model that estimates one set of values for the whole sample is presented here. Model fit comparisons revealed that C could be dropped from the multivariate model without significant deterioration of the fit (Table 3).

Low mindfulness had significant moderate genetic correlations and small nonshared environmental correlations with depressive symptoms and anxiety sensitivity ($r_A = .52$ and $.53$, respectively, $r_E = .22$ for both, Table 2). Depressive symptoms and anxiety sensitivity showed moderate genetic and nonshared environmental correlations ($r_A = .67$ and $r_E = .34$). Genetic influences accounted for over half of the phenotypic correlations between the variables (Table 2).

The Cholesky decomposition revealed that about two-thirds of genetic influences and almost all nonshared environmental influences on mindfulness are independent of the influences on depressive symptoms and anxiety sensitivity (specific influences on mindfulness: $a33 = \sqrt{.23}$, CI: $.16$ – $.30$; and $e33 = \sqrt{.61}$, CI: $.54$ – $.68$).

DISCUSSION

This is the first study to investigate the genetic and environmental underpinnings of trait mindfulness and

TABLE 2. Multivariate results—phenotypic, genetic, and nonshared environmental correlations, and proportion of phenotypic correlation explained by *A* and *E*

	Cross twin cross trait correlations		Phenotypic, genetic and environmental correlations			Proportion of the phenotypic correlation explained by <i>A</i> and <i>E</i>	
	r_{MZ}	r_{DZ}	r_{pb}	r_A	r_E	<i>A</i>	<i>E</i>
Mindfulness–depression	.20 (.15–.28)	.06 (.01–.11)	.34 (.30–.37)	.52 (.40–.64)	.22 (.15–.29)	.60 (.47–.72)	.40 (.28–.53)
Mindfulness–anxiety sensitivity	.19 (.12–.26)	.10 (.05–.15)	.34 (.30–.38)	.53 (.39–.66)	.22 (.15–.30)	.59 (.44–.73)	.41 (.27–.56)
Depression–anxiety sensitivity	.31 (.28–.34)	.15 (.13–.17)	.48 (.47–.50)	.67 (.63–.72)	.34 (.31–.37)	.60 (.55–.64)	.40 (.36–.45)

Note: MZ, monozygotic, DZ, dizygotic, r_{pb} , phenotypic correlation; r_A , genetic correlation; r_E , nonshared environmental correlation; *A*, additive genetic parameters; *E*, nonshared environmental parameters.

95% Confidence intervals (CIs) are presented in brackets. CIs not including 0 indicate significant estimates. Nonoverlapping CIs mean significant difference between the values. Some of the DZ correlations are less than half MZ correlations, suggesting that *A* should be interpreted as both additive and dominant genetic effects. Partial correlations revealed that mindfulness was independently associated with depression ($r = .22$ (95% CIs: .18–.26)) and anxiety sensitivity ($r = .17$ (95% CIs: .13–.21)). Furthermore, controlling for mindfulness significantly reduced the correlation between depression and anxiety sensitivity ($r = .43$ (95% CIs: .41–.45)), suggesting that mindfulness might play a role in the relationship between the anxiety sensitivity and depression. AE models are presented, as *C* influences were small and not significant (except depression), and were dropped from the model without a significant deterioration of the fit (Table 3). The results of the full ACE model are presented in the appendix (Supporting Information Table A3).

its relationship with depression and anxiety sensitivity. Quantitative genetic analysis revealed that mindfulness is influenced by both genetic and nonshared environmental factors, with no influence of shared environmental factors. Common genetic influences were found to explain most of the moderate association between low mindfulness, depressive symptoms, and anxiety sensitivity. Despite the significant genetic and environmental associations with depressive symptoms and anxiety sensitivity, mindfulness was also characterized by unique genetic and environmental influences.

Phenotypic analyses confirmed previous findings that low mindfulness is associated with both depressive symptoms^[44] and anxiety sensitivity.^[23–25] The mindfulness measure used in the current study focuses specifi-

cally on the attentional aspect of this trait. The shared genetic and environmental risk for depression, anxiety sensitivity, and our attentional measure of low mindfulness is in line with the evidence that cognitive impairment, including attentional control deficits, is an important aspect of depression^[16,17] and of anxiety sensitivity. The mental concerns dimension of anxiety sensitivity that measures worry regarding cognitive control was found to be particularly strongly associated with depression,^[20] suggesting that concerns about own attentional performance may be an important content of the cognitive biases in depressed individuals. Furthermore, a recent study showed that improvements in attentional control following mindfulness training were associated with reductions in depressive symptoms.^[26]

TABLE 3. Multivariate model fit statistics

	–2LL	<i>df</i>	χ^2	Δdf	<i>P</i>	AIC	Size-adjusted BIC
(a) Comparison to saturated model							
Saturated model	54727.96	21200				12327.96	55451.66
Correlated factors solution (ACE)	55298.39	21312	570.43	112	<.05	12674.39	55421.69
(b) Comparison to correlated factors solution (ACE)							
Correlated factors solution (AE)	55309.93	21318	11.54	6	0.07	12673.93	55401.06
Correlated factors solution (CE)	55386.44	21318	88.05	6	<.05	12750.44	55477.57
Correlated factors solution (E)	56213.32	21324	914.93	12	<.05	13565.32	56272.29

Note: –2LL, minus twice the log likelihood; *df*, degrees of freedom; *P*, probability; AIC, Akaike's information criterion; BIC, Bayesian's information criterion.

The correlated factors solution did not fit as well as the saturated model. This occurs frequently in studies with very large sample sizes because minimal variance differences between groups can be highly statistically significant. The best fitting model (correlated factors solution, AE) was selected based on the principle of parsimony and lowest AIC and BIC value. A difference in AIC between two models of 2 or less, provides equivalent support for both models (in which case the most parsimonious model should be chosen), a difference of 3 indicates that the lower AIC model has considerably more support, and a difference of more than 10, indicates that the lower AIC model is a substantially better fit compared to the higher AIC model.^[42] For completeness, the results of the full ACE model are presented in the appendix (Supporting Information Table A3).

Given that adolescence is a period of heightened brain plasticity,^[7] mindfulness might be especially useful in depression prevention and treatment in young people by means of improving attentional control.^[9] However, due to the cross-sectional nature of the data, the direction of the associations and the possible causal links between mindfulness, depression, and anxiety sensitivity need to be explored in future longitudinal studies. Furthermore, genetically sensitive interventions are needed to investigate how the role of genes and environment in mindfulness and its etiological relationship with anxiety sensitivity and depression might change due to an intervention.^[45]

The univariate twin modeling results highlight the role of both genetic and individual-specific environmental influences in adolescent mindfulness. Environmental factors might include parenting, life events, cultural exposure, and meditation-related training. Future research focused on identifying these environmental factors may inform targeted clinical and resilience interventions in adolescence. We did not find evidence for a role of shared environmental influences on trait mindfulness, suggesting that environmental factors are individual specific. This might be because shared environmental influences are thought to play a diminishing role in adolescence and adulthood.^[38] It would be interesting to investigate the genetic and environmental influences on mindfulness in younger age groups, when trait mindfulness emerges and when the earliest mindfulness-based interventions may be implemented.^[9, 46, 47] We found that on average females were less mindful than males; however, we did not find evidence for sex differences in the etiology of mindfulness. Sex differences are rarely examined in mindfulness, so these results warrant further investigation.

Multivariate twin modeling analyses revealed that mindfulness, depressive symptoms, and anxiety sensitivity share moderate genetic and small nonshared environmental correlations. Furthermore, we found that genetic influences account for over half of the moderate phenotypic association between mindfulness, depressive symptoms, and anxiety sensitivity. Thus, as expected, the association between mindfulness and these internalizing problems is explained largely by underlying genetic liability, in line with the “generalist genes hypothesis.”^[28] The results are suggestive of a biological pathway linking mindfulness, depression, and anxiety sensitivity. Recent studies point to epigenetic regulation of inflammatory pathways as one of the biological mechanisms underpinning the mindfulness-based interventions.^[48] The biological pathways associated with mindfulness that may benefit mental health could include positive regulation of brain, endocrine, and immune function.^[49]

Despite its etiological links with depressive symptoms and anxiety sensitivity, mindfulness is characterized by significant unique influences, with about two-thirds of genetic factors and almost all nonshared environmental factors being independent of depressive symptoms and anxiety sensitivity. It is in line with a growing body of research suggesting that mindfulness is associated with a

range of other constructs over and above its link with depressive symptoms and anxiety sensitivity.^[10, 30] Overall, the current study adds to the evidence that above its role in mood problems, mindfulness might also be characterized by unique developmental patterns and protective factors worth investigating.

LIMITATIONS

The large, genetically-informative sample is the strength of the study. However, a number of limitations are worth noting. First, it remains debated whether mindfulness can be accurately assessed using self-report questionnaires, and it is suggested that it may be better captured by measures such as interviews.^[50] Although there are no objective markers of mindfulness that the questionnaires could be validated against, self-report mindfulness is negatively associated with behavioral measures of related constructs, such as mind wandering^[51] and attention lapses.^[52] An additional limitation of self-report data is that it could have inflated nonshared environmental correlations due to shared measurement error. Second, the current study used a relatively narrow definition of mindfulness in terms of attentional processing, but it did not measure other facets of the trait, such as the nonjudgmental and accepting attitude.^[50] This somewhat limits the interpretability of the current results and mindfulness as a broader and multifactorial concept remains to be investigated in future twin studies. However, the focus on attentional control allowed more precise investigations of one specific cognitive mechanism central to mindfulness and its association with depression and anxiety sensitivity. Similarly, the relatively narrow age-range of the current sample limits the generalizability of the results to other ages, although the etiology of depression and anxiety sensitivity is not expected to change markedly across adolescence.^[53, 54] Furthermore, the precise age-range allows closer understanding of a specific developmental stage. Future research should elucidate etiology of mindfulness across development to further inform this debate. Finally, there are a number of limitations inherent to the twin design, comprehensively discussed elsewhere.^[38] These limitations have minimal and contrasting effects but suggest that parameter estimates should be taken as indicative rather than absolute values.

CONCLUSIONS

The present study is the first to show that both nature and nurture play an important role in adolescent mindfulness. Future research should focus on elucidating the specific environmental factors that promote trait mindfulness across development. Furthermore, the current study revealed shared genetic influences underpinning the associations between mindfulness, depressive symptoms, and anxiety sensitivity, and suggests that attention control may be a key cognitive mechanism that explains this association. Future research should focus on examining the specific biological pathways underlying this

association. Finally, the evidence of a significant proportion of unique genetic and environmental influences on mindfulness suggests largely independent influences on this trait.

Acknowledgments. The authors have declared no competing or potential conflicts of interest. TEDS was supported by the United Kingdom Medical Research Council [G1100559 to Angelica Ronald; G0901245, and previously G0500079, to Robert Plomin]. Monika A. Waszczuk was supported by a Ph.D. studentship funded by the Alexander von Humboldt Foundation. Claire M. A. Haworth was supported by a research fellowship from the British Academy. Robert Plomin was supported by a Medical Research Council Research Professorship award [G19/2] and also a European Research Council Advanced Investigator award [295366]. This work is also supported by the Leverhulme Foundation [RPG-210 to Thalia Eley]. This study presents independent research part-funded by the National Institute for Health Research (NIHR) Biomedical Research Centre at South London and Maudsley NHS Foundation Trust and King's College London. The views expressed are those of the author(s) and not necessarily those of the NHS, the NIHR, or the Department of Health. We thank the families for their participation as well as numerous staff and students from the MRC SGDP Centre, Institute of Psychiatry, Psychology & Neuroscience, London.

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