Stress has vital influences on important life outcomes, such as achievement in work settings, relationship satisfaction, mental health, and physical health (Cohen & Williamson, 1991; Dyson & Renk, 2006; Neff & Karney, 2004; Sauter & Murphy, 1995). The process of stress involves experiencing stimuli, which are commonly referred to as stressors, an appraisal of the stressors, and a response (Cohen, Kessler, & Gordon, 1995). Stressors can be perceived as either challenges or threats. When stressor demands are appraised as uncontrollable and exceeding available coping resources, the stressors will be perceived as threats and the psychological state of stress will be elicited (Lazarus & Folkman, 1984).

Personality traits, consistent, automatic patterns of thoughts, feelings, or behaviour, may lead people toward more or less stress experiences. Personality traits demonstrate significant associations with perceived stress in both cross-sectional and longitudinal studies (Esbstrup, Eplov, Pisinger, & Jørgensen, 2011). However, very little work has explored the developmental processes underlying the relation between personality traits and perceived stress, both of which are subject to genetic influences (Briley & Tucker-Drob, 2014; Federenko et al., 2006). To what extent does personality predict perceived stress due to common genetic effects compared with environmental pathways? Moreover, no research has examined the genetic and environmental pathways underlying the continuity and changes in perceived stress over time in a longitudinal design. Research has also shown that changes in personality are associated with changes in perceived stress (Luo & Roberts, 2015). Whether the dynamic association between personality and perceived stress is attributable to genetic or environmental influences remains unknown. We conducted two studies to address these questions. In study 1, we examined whether the concurrent associations between personality traits and perceived stress were mediated by genetic or environmental factors. In study 2, we examined the contribution of genetic and environmental factors to the continuity and changes in perceived stress in a 10-year longitudinal design, and whether these factors were shared with similar effects on personality traits.

Relations between personality traits and perceived stress

Modern theories of personality articulate systems with multiple levels of functioning. For example, in the sociogenomic personality model of personality traits (Roberts, 2017), personality traits are embedded in a system containing a combination of traits, states, environments, and biological factors. As the sociogenomic model specifies, personality traits are defined as the relatively enduring, automatic patterns of thoughts, feelings, and behaviours that distinguish individuals from each other, whereas states are similarly made up of the thoughts, feelings, and behaviours an individual exhibits in any given moment. Thus,
traits represent aggregations of states that display continuity over time, although states represent thoughts, feelings, and behaviours captured in a specific situation and moment. According to the sociogenomic model, measures of personality traits capture a substantial amount of state variance. Meanwhile, state measures also contain a substantial amount of trait variance as personality trait measures typically demonstrate a correlation about 0.5 with state measures.

Commonly defined as a state, perceived stress may also reflect trait variance. Under the transactional model of stress developed by Lazarus and colleagues (Lazarus, 1966; Lazarus & Folkman, 1984), stress is interpreted as transactional processes that result from the ongoing interplay between the person and the environment. According to the model, two appraisal processes, primary appraisal, and secondary appraisal influence the outcome of the stress processes. In primary appraisal, demands of external stressor and personal stake in the situations are appraised by the person, and personal resources available to cope with the stressors are evaluated in secondary appraisal. Stress results when an imbalance is perceived between the external demands and personal coping resources (Lazarus & Folkman, 1984).

Although the effects of personality traits were not specified in the transactional model, personality traits play crucial roles on the side of the person. Specifically, personality may be related to the likelihood of experiencing stressful events (i.e. selection effects; Kandler, Bleidorn, Riemann, Angleitner, & Spinath, 2012) and to the interpretation of stressful events (i.e. the appraisal processes; Larsson, 1989). The experience of perceived stress is jointly affected by personal dispositions and the environment. As suggested by Vollrath (2001), personality traits may affect both the descriptive situation representations and the evaluative aspects of the perceptions of the situations. For example, neuroticism was found to be related to negative descriptions of the environment, whereas conscientiousness and agreeableness were associated with positive biases. Furthermore, the influences of personality traits on situation evaluation found in previous research are consistent with the reactivity effects of person-environment transactions which state that individuals tend to extract a subjective psychological environment from the objective surroundings based on their personality (Roberts, Wood, & Caspi, 2008). Personality traits may affect cognitive systems that act as a filter for social information, and these personality-based cognitive filters can create an idiosyncratic reality unique to one’s personality.

Previous research has shown consistent associations between personality traits and the subjective perception of stress. For example, extraversion, conscientiousness, and agreeableness were negatively related to perceived stress, but neuroticism was positively related to perceived stress (Ebstrup et al., 2011). Neuroticism has been found to predict higher stressor intensity and lower perceived stressor control, whereas agreeableness predicted lower stressor intensity and conscientiousness predicted higher perceived stressor control (Kaiseler, Polman, & Nicholls, 2012). Similarly, other studies reported positive correlations between neuroticism and perceived stress and negative correlations between conscientiousness, extraversion, agreeableness, openness, and perceived stress (Baldasaro, Shanahan, & Bauer, 2013; Penley & Tomaka, 2002).

Sources of variance in personality and stress

Previous research has provided strong evidence that personality traits are heritable (e.g. Briley & Tucker-Drob, 2014). Numerous twin and adoption studies suggest that about 25% to 50% of variance in personality is attributable to genetic factors (Bouchard & Loehlin, 2001; Bouchard & McGue, 1990; Loehlin, Horn, & Willerman, 1981). In a meta-analytic study, it was found that about 40–55% of variance in adult personality traits is due to genetic influences (Johnson, Vernon, & Feiler, 2008). Moreover, after correction for error variance and nonrandom method specificity (i.e. self-rater-specificity and other-rater-specificity) on the basis of assessments from multiple raters, estimates of heritability were substantially larger than estimates which were based on single self-reports, peer reports (i.e. close friends), or observer ratings (i.e. research assistant behavioural coding) alone (Borkenau, Riemann, Angleitner, & Spinath, 2001; Kandler et al., 2016; Kandler, Riemann, Spinath, & Angleitner, 2010).

Similarly, genetic differences appear to partly explain the individual differences in subjective perception of stress. A study examining the heritability of perceived chronic stress reported heritability estimates that varied between 5% and 45%, depending on the scale used (Federenko et al., 2006). Genetic factors were found to contribute to 44% of the variance in perceived stress in a study examining the genetic etiology of stress and depression (Bogdan & Pizzagalli, 2009). Similar findings have been reported in research on the heritability of neuroticism, depressive symptoms, and perceived psychological stress (the heritability of perceived stress was 52%). This study also reported that the genetic variance in perceived stress was largely shared with that in neuroticism and susceptibility to depressive symptoms (Rietschel et al., 2014).

Therefore, evidence has shown that personality traits and perceived stress are correlated, and personality traits and perceived stress are both partially genetically influenced. However, it remains unclear that whether the relation between personality traits and perceived stress is mediated by genetic or environmental factors. No study has systematically examined the genetic and environmental sources of the association between all of the Big Five personality traits and perceived stress. It may be the case that environmental sources of variance trigger both perceived stress and personality development (e.g. experiencing a stressful life event may make someone perceives more stress and become more neurotic). Alternatively, environmental sources of personality variance may lead some individuals to experience differing amounts of stress or to perceive stressful events differently, more consistent with a selection effect on the basis of personality. On the other hand, there may be genetic variants that independently lead individuals to behave a certain way and perceive a certain level of stress, consistent with a genetic association between personality and stress. This association could manifest in several ways and possibly be mediated by the
environment through gene–environment correlation. We discuss these various interpretive lenses more fully in the discussion. In the present study, we will first examine the extent to which the Big Five and perceived stress share genetic and environmental covariance.

**Sources of variance in continuity and changes in personality and stress**

Although it is common to test the heritability of phenotypes like personality and stress in cross-sectional studies, it is less common to use longitudinal behaviour genetic methods. Stability and change in personality derive from genetic and environmental factors (McGue, Bacon, & Lykken, 1993; Pedersen & Reynolds, 1998). For example, a study exploring stability and changes in personality from ages 17 to 24 reported comparable genetic and environmental contributions to individual differences in change, whereas genetic effects were more prominent for rank-order stability during the transition into adulthood (Blonigen, Carlson, Hicks, Krueger, & Iacono, 2008). In a longitudinal study that examined the 10-year stability and change of the Big Five and their facets in an adult sample, both genetic and environmental factors were found to contribute to stability; whereas, noticeable differences existed in the magnitude of genetic and environmental contribution to change depending the traits examined (Bleidorn, Kandler, Riemann, Angleitner, & Spinath, 2009). Interestingly, on average, the genetic and environmental variance in change was approximately equal, raising the possibility that fluctuations may simply reflect noise (see Turkheimer, Pettersson, & Horn, 2014, pp. 518–520 for a similar argument focusing on heritability estimates). Finally, in a meta-analysis investigating genetic and environmental continuity in personality development, the contribution of genetic effects to personality stability was found to be moderate and relatively constant with age, but the contribution of environmental effects increased from early childhood to adulthood (Briley & Tucker-Drob, 2014). In adulthood, the relative contribution of genetic and environmental sources of continuity was nearly equivalent.

Therefore, previous research consistently indicates that genetic and environmental factors contribute to personality trait stability as well as change. In the present study, we used longitudinal data over 10 years to examine the relative contribution of genetic and environmental factors to continuity and changes in personality traits.

In terms of the experience of stress over time, very few studies have explored the genetic and environmental contributions to longitudinal patterns of stress. Two studies examined the genetic and environmental influences on stressful life events in longitudinal data. The first longitudinal study (Johnson, Rhee, Whisman, Corley, & Hewitt, 2013) suggested that the influence of genetic factors on exposure to dependent events, which are events due to the behaviour or characteristics of the individual, increased during the transition from childhood to adolescence. In contrast, independent events, which are not related to the individual behaviour or characteristics, were less heritable than dependent events, at least for boys. In a second longitudinal study, both genetic and environmental effects were found to contribute to stability of stressful life events (Kandler et al., 2012). No studies have examined the genetic and environmental influences on continuity and changes in subjective perception of stress over time. The present study investigated the genetic and environmental contributions to continuity and changes in perceived stress over a 10-year period of time.

A final question concerns to what extent the developmental interplay of personality and stress over time is the result of genetic or environmental factors. Like the longitudinal genetic analysis of stress, this interplay has never been tested. Past research has shown that changes in personality are associated with changes in perceived stress over 3 years (Luo & Roberts, 2015). What is not known is the extent to which continuity in the association between personality and stress and the relation between changes in personality and changes in perceived stress are the result of genetic and environmental factors. The longitudinal data used in the current study provided us with the opportunity to explore the questions.

**The current study**

The present study aimed to investigate the genetic and environmental sources underlying the concurrent and longitudinal relation between personality traits and perceived stress. Examining the genetic influences on perceived stress and the link between personality traits and perceived stress would account for the findings that individual differences exist in the perception of stress even for the exposure of the same stressor. Parsing genetic and environmental influences on the continuity in perceived stress and the continuity in the associations between personality traits and perceived stress would explain the mechanisms of the proneness to experience perceived stress over long periods of time, given the well-established relations between psychological reactions to stress and physical and mental health (Dougall & Baum, 2012). Finally, disentangling the changeable portions of personality traits and perceived stress from the consistent patterns and examining the genetic and environmental mechanisms underlying their dynamic associations extend our current understanding of the link between personality traits and perceived stress to a developmental behavioural genetic perspective.

In the current paper, we describe two studies in which we investigated the extent to which (i) the concurrent association between personality and the subjective perception of stress is genetically and environmentally mediated, (ii) the extent to which the continuity and changes in personality and subjective perception of stress are attributable to genetic and environmental influences, (iii) the extent to which the continuity in the associations between personality and subjective perception of stress are driven by genetic and environmental influences, and (iv) the extent to which the longitudinal relations between changes in personality and changes in subjective perception of stress are genetically and environmentally mediated.
Study 1

METHOD

Participants

The data used in study 1 were drawn from the fourth wave of the National Longitudinal Study of Adolescent to Adult Health (Add Health IV). The first wave of the survey data was collected from a nationally representative sample of adolescents in grades 7–12 (typically at age 12–13 to age 17–18 in the United States) in the United States during the 1994–1995 school year. The cohort has been followed up into young adulthood with four waves of data collection. Because of limited availability of personality data, we only made use of wave four. The sample data used in the current study were collected in 2008–2009 from 1104 individuals (51.3% female) including 225 complete MZ, 179 complete same-sex DZ, and 148 complete opposite-sex DZ twin pairs. The mean age of twin sample was 28.34.

Measures

Personality. The Big Five personality traits were measured by the mini-International personality item pool items (Donnellan, Oswald, Baird, & Lucas, 2006). Each of the Big Five personality traits was assessed by four items. Each item was rated on a 5-point Likert scale ranging from 1 (strongly agree) to 5 (strongly disagree). However, one item for openness was dropped because of its low item-total correlation and average inter-item correlation in the current twin sample. The alpha reliabilities for the five personality traits in the current sample were 0.61 for neuroticism, 0.64 for conscientiousness, 0.71 for extraversion, 0.66 for agreeableness, and 0.57 for openness. Although these reliability estimates are relatively modest, they are to be expected given the brief instrument used and this process reinforces conscientiousness, then this produces genetic variance. A portion of heritability likely includes such environmentally mediated processes. Similarly, if genetically influenced levels of neuroticism alter the manner in which individuals respond to stressful life events (i.e. gene × nonshared environmental interaction), then this produces nonshared environmental variance, even though genetic variation impacts the development.

We first tested for sex limitation for each of the Big Five personality traits and perceived stress (Neale, Roysamb, & Jacobson, 2006). Two forms of sex limitation are generally tested. The first form is qualitative sex limitation, in which different genetic effects operate across gender. Quantitative sex limitation occurs when heritability or environmental differences across gender. We fitted three models using OpenMx (Neale et al., 2016) for each of the five personality traits and perceived stress in Mplus 7.4 (Muthén & Muthén, 1998–2015). Fit indices suggested reasonable to good fit of the models {comparative fit index [CFI] ranged from 0.83 to 1}. All of the factor scores were residualized for age, gender, and age × gender. To investigate the genetic and environmental influences on the association between personality traits and perceived stress, we followed a classical twin model to compare the resemblance of monozygotic (MZ) and dizygotic (DZ) twins. The structural equation model we used assumes that an observed phenotype function is a linear additive combination of three components: additive genetic effects (A), environmental effects shared within twin pairs (C), and environmental effects not shared by twin pairs (E). The variance explained by each of the components is referred to as $a^2$, $c^2$, and $e^2$. The percentage of genes shared between MZ twins is expected to be 100%, and DZ twins share 50% of their segregating genetic material on average. The model is based on the assumption that the trait-relevant environmental influences shared by MZ twins are equal to that shared by DZ twins (Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013). We also assume that there is no assortative mating with respect to the traits of interest and no interaction between genetic and environmental effects (Eaves et al., 1999). Further, the models we use do not explicitly consider nonindependence of genetic and environmental influences, but these developmental processes exert predictable influences on model parameters (see Purcell, 2002). When genes and shared environments are correlated (i.e. passive gene–environment correlation), shared environmental variance is produced; and when genes and nonshared environments are correlated (i.e. active or evocative gene–environment correlation), genetic variance is produced. When genes and shared environments statistically interact, genetic variance is produced; and when genes and nonshared environments interact, nonshared environmental variance is produced. As these developmental processes are likely to occur, we encourage a nuanced interpretation of model parameters that take these issues into consideration (e.g. Briley & Tucker-Drob, 2017). For example, if genetically influenced levels of conscientiousness lead people to experience achievement striving environments (i.e. gene-nonshared environment correlation) and this process reinforces conscientiousness, then this produces genetic variance. A portion of heritability likely includes such environmentally mediated processes. Similarly, if genetically influenced levels of neuroticism alter the manner in which individuals respond to stressful life events (i.e. gene × nonshared environmental interaction), then this produces nonshared environmental variance, even though genetic variation impacts the development.

All of the scripts for the analyses that are described can be found here: https://osf.io/5wj7q/. Factor scores for the Big Five personality traits and perceived stress were used in subsequent analyses. To form factor scores, the items of personality traits and perceived stress were used as manifest indicators to form the latent variables of each personality trait and perceived stress in Mplus 7.4 (Muthén & Muthén, 1998–2015). Fit indices suggested reasonable to good fit of the models {comparative fit index [CFI] ranged from 0.83 to 1}. All of the factor scores were residualized for age, gender, and age × gender. To investigate the genetic and environmental influences on the association between personality traits and perceived stress, we followed a classical twin model to compare the resemblance of monozygotic (MZ) and dizygotic (DZ) twins. The structural equation model we used assumes that an observed phenotype function is a linear additive combination of three components: additive genetic effects (A), environmental effects shared within twin pairs (C), and environmental effects not shared by twin pairs (E). The variance explained by each of the components is referred to as $a^2$, $c^2$, and $e^2$. The percentage of genes shared between MZ twins is expected to be 100%, and DZ twins share 50% of their segregating genetic material on average. The model is based on the assumption that the trait-relevant environmental influences shared by MZ twins are equal to that shared by DZ twins (Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013). We also assume that there is no assortative mating with respect to the traits of interest and no interaction between genetic and environmental effects (Eaves et al., 1999). Further, the models we use do not explicitly consider nonindependence of genetic and environmental influences, but these developmental processes exert predictable influences on model parameters (see Purcell, 2002). When genes and shared environments are correlated (i.e. passive gene–environment correlation), shared environmental variance is produced; and when genes and nonshared environments are correlated (i.e. active or evocative gene–environment correlation), genetic variance is produced. When genes and shared environments statistically interact, genetic variance is produced; and when genes and nonshared environments interact, nonshared environmental variance is produced. As these developmental processes are likely to occur, we encourage a nuanced interpretation of model parameters that take these issues into consideration (e.g. Briley & Tucker-Drob, 2017). For example, if genetically influenced levels of conscientiousness lead people to experience achievement striving environments (i.e. gene-nonshared environment correlation) and this process reinforces conscientiousness, then this produces genetic variance. A portion of heritability likely includes such environmentally mediated processes. Similarly, if genetically influenced levels of neuroticism alter the manner in which individuals respond to stressful life events (i.e. gene × nonshared environmental interaction), then this produces nonshared environmental variance, even though genetic variation impacts the development.

1Assortative mating: studies reported modest assortative mating coefficients of about 0.1 to 0.2 for behavioural attributes including personality traits. Although positive assortative mating can cause genetic effects to be underestimated, the relatively small values would only have limited deflating influences on heritability estimates (Jockin, McGue, & Lykken, 1996).
perceived stress to test the two forms of sex limitation. The first model (ACE model) allowed all the variance components to differ across gender and the additive genetic correlation between opposite sex pairs. The second model (qualitative sex limitation test) fixed the additive genetic correlation within opposite sex DZ twins to be 0.5. In the third model (quantitative sex limitation test), the genetic and environmental variance components were set to be equal across males and females. Model fit indices were used to determine the presence of sex limitation (see Table S1). Based on the test results, no qualitative or quantitative sex limitation was found for the Big Five personality traits or perceived stress. This result supports the inclusion of opposite sex DZ twins in the analysis.

We then fitted univariate structural equation models via maximum-likelihood model fitting analysis to disentangle the additive genetic, shared environmental and nonshared environmental effects (ACE models) on each of the five personality traits and perceived stress, respectively. In order to use the most parsimonious number of parameters, we also fitted AE models (models to disentangle the additive genetic and nonshared environmental effects) for each construct and model fit indices were used to compare fit of the models (ACE versus AE models).

A multivariate Cholesky model was used to explore the additive genetic, shared environmental, and nonshared environmental contributions to the covariance between the personality traits and perceived stress. A simplified version of the basic format of the model is shown in Figure 1. Only factors that demonstrated significant effects in the univariate analyses were estimated in the multivariate model. As Figure 1 illustrates, \( A_p \) and \( A_s \) denote the genetic factors of the personality traits and perceived stress, \( C_p \) and \( C_s \) denote the shared environmental factors of the personality traits and perceived stress, whereas \( E_p \) and \( E_s \) represent the nonshared environmental factors. The path coefficients estimate the contribution of genetic, shared or nonshared environmental influences. For example, the coefficient of the cross path \( a_{12} \) estimates the extent to which the genetic factors of personality explain individual differences stress, and the path coefficient \( a_{22} \) indicates the genetic contribution that is unique to stress after taking the genetic influences of personality into account. The path coefficients denoted by \( c \) and \( e \) explain the effects of shared and nonshared environment in the same way. In the actual model, we included all of the Big Five and stress. The personality variables would come prior to stress. The interpretation is similar to multiple regression except that earlier variables explain variance in later variables.

**RESULTS**

The phenotypic correlations between conscientiousness, neuroticism, extraversion, agreeableness, openness, and perceived stress were \(-0.31 [-0.40, -0.22], 0.64 [0.57, 0.71], -0.22 [-0.30, -0.13], -0.08 [-0.18, 0.01], \) and \(-0.32 [-0.42, -0.23], \) respectively. Table S2 and S3 display the within-pair correlations and results from the univariate model fitting analyses for the Big Five personality traits and perceived stress. The results verified the presence of genetic influences on personality traits and perceived stress as suggested by the within-pair correlations. According to the model fit indices and the chi-square test, the model with only additive genetic factors and nonshared environmental factors (AE model) fit better than the model including shared environmental factors (ACE model).2 We also decomposed the significant bivariate correlations between personality traits and perceived stress into genetic and environmental components. The parameter estimates from these models are reported in Table S4. The genetic cross-path \( (a_{12}) \) was significant for neuroticism, conscientiousness, and openness.

In the model including neuroticism, there was no residual genetic variance in perceived stress. The nonshared environmental cross-path \( (e_{12}) \) was significant for neuroticism, conscientiousness, and extraversion. Figure 2 displays these results graphically. The x-axis reflects total variance in perceived stress. This variance has been decomposed into four components: genetic variance shared with the personality dimension, environmental variance shared with the personality dimension, genetic variance unique to perceived stress, and environmental variance unique to perceived stress. Most

\[ \text{Figure 1. Basic format of the model used to estimate the additive genetic, shared environmental and nonshared environmental contributions to the covariance between personality traits and perceived stress.} \]

2As some of the within-pair correlations for MZ twins were more than twice as large as that for DZ twins, we also compared ADE models with AE models for univariate analyses. Fit indices (AICs) only indicated better model fit for ADE than AE for extraversion. We decomposed the correlation between extraversion and stress and resulted in that dominance genetic and environmental effects accounted for about 25% and 77% of the association, respectively. It has been suggested that AE models provide a reasonable estimate of genetic influences, broadly defined, and therefore we primarily interpret AE models (Hill, Goddard, & Visscher, 2008).
variance in perceived stress was unique environmental variance. Common variance between personality and stress tended to be genetic for openness (71%), neuroticism (36%), and conscientiousness (60%) but was substantially lower for extraversion (19%).

We then conducted multivariate analysis to examine the contribution of genetic and environmental effects to the covariance between the Big Five personality traits and perceived stress. Figure 3 presents the path coefficients from the multivariate analyses of the associations between the latent scores of personality traits and perceived stress (path coefficients between personality traits are shown in Table S5). The univariate analyses suggested that about 27% of the variance in perceived stress was accounted for by genetic effects. The multivariate analyses indicated that this genetic variation was completely shared with genetic variance for the personality traits \( \left( \frac{-0.252}{0.406} + \frac{-0.012}{0.181} + \frac{-0.091}{0.081} = 0.270 \right) \), the coefficients are for conscientiousness, neuroticism, extraversion, agreeableness, and openness corresponding to shown in Figure 3, but no genetic variance was unique to perceived stress. In terms of the 73% of the variance in perceived stress accounted for by nonshared environmental effects, 61.3% \( (0.783^2) \) was unique to perceived stress, and only the other 11.7% \( \left( [0.104]^2 + 0.296^2 + [0.111]^2 + 0.081^2 + 0.002^2 = 0.117 \right) \), the coefficients are for conscientiousness, neuroticism, extraversion, agreeableness, and openness, respectively of the variation was accounted for by the nonshared environmental variance for the Big Five personality traits. Thus, about 38.7% \( (0.270 + 0.117) \) of the total variance in perceived

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**Figure 2.** Genetic and environmental variance of perceived stress shared with personality traits and genetic and environmental variance unique to perceived stress in add health.

**Figure 3.** Multivariate analyses of the additive genetic and nonshared environmental contributions to the covariance between personality traits and perceived stress in add health twin sample. The paths between personality traits are omitted. Co, conscientiousness; N, neuroticism; Ex, extraversion; Ag, agreeableness; O, openness; S, perceived stress; A, additive genetic factors; E, nonshared environmental factors.
stress was shared with that in personality traits, 70% (0.270/0.387) of which was due to genetic effects.

SUMMARY
In study 1, we examined the influences of genetic factors and environmental factors on the concurrent covariance between the Big Five personality traits and perceived stress. The results suggested the genetic variation in perceived stress was completely shared with personality traits. In total, perceived stress shared about 38.7% of the variance with the Big Five personality traits, and 70% of the variance shared between perceived stress and personality traits was explained by genetic effects. In study 2, we examined the genetic and environmental aetiology of the longitudinal associations of stress and personality traits.

Study 2

METHOD
Participants
The data used in this study were drawn from the Midlife in the United States Survey (MIDUS I and II; Brim, Ryff, & Kessler, 2004). The survey data were collected from a nationally representative sample with the participants completing a 30-min telephone interview and self-administered questionnaires. The sample data used in the current study in the first wave were collected in 1995–1996 from 1590 individuals (56% female) including 317 complete MZ, 279 complete same-sex DZ, and 199 complete opposite sex DZ twin pairs. The mean age of the twin sample was 45.46 (range from 25 to 75, SD = 12.24). The follow-up survey data were collected in 2004–2006. The sample in the second wave was composed of 846 individuals (59% female) including 175 complete MZ, 148 complete same-sex DZ, and 100 complete opposite-sex DZ twin pairs. The mean age of the twin sample in the second wave was 54.5 (range from 34 to 82, SD = 11.45). The sample of individuals who provided data at both waves consisted of 812 individuals, including 170 complete MZ, 142 complete same-sex DZ, and 94 complete opposite-sex DZ twin pairs. We tested whether attrition resulted in an unrepresentative longitudinal sample. The results suggested that there is no significant difference in those who did and did not complete assessment at time 2 in terms of age (t = 0.61, p = 0.54, d = 0.03). There was an effect for gender (χ² = 10.38, p = 0.00, 59.8% female in those completed the assessment at time 2, and 51.8% in those did not) such that males were more likely to drop out from the study than females. Individuals who completed the assessment at time 2 had higher scores on conscientiousness (t = 4.78, p = 0.00, d = 0.24) than those who did not. Those who did and did not complete the assessment at time 2 were not different in terms of neuroticism (t = 1.31, p = 0.19, d = 0.07), extraversion (t = 0.59, p = 0.56, d = 0.03), agreeableness (t = 0.01, p = 0.99, d = 0.00), openness, (t = 0.89, p = 0.38, d = 0.04), and perceived stress (t = 0.58, p = 0.56, d = 0.03).

Measures

Personality. Personality was measured by the Midlife Development Inventory (MIDI; Lachman & Weaver, 1997). The MIDI personality inventory contains 25 adjectives that assess neuroticism, conscientiousness, extraversion, agreeableness, and openness. The items were rated on a 4-point scale from 1 ‘not at all’ to 4 ‘a lot’. Cronbach alphas for each trait at wave 1 and wave 2, respectively, were as follows: 0.76 and 0.74 for neuroticism, 0.55 and 0.51 for conscientiousness, 0.78 and 0.76 for extraversion, 0.80 and 0.81 for agreeableness, and 0.77 and 0.76 for openness. Similar to study 1, the reliability estimates for some dimensions are rather modest due to the short assessment strategy.

Stress. A standard measure of perceived stress was not administered to the MIDUS sample; therefore, we developed a measure of stress out of the items in the survey. In developing the perceived stress measure, 19 items rating work, finance, relationship, and life in general were chosen from different sections of the MIDI (Brim & Featherman, 1998). The 19 items were posted together with the PSS (Cohen & Williamson, 1988) on Amazon Mechanical Turk (MTurk) and a sample of 435 participants who rated each item and the PSS (the MTurk data can be found at: https://osf.io/5wj7q/). In the MTurk sample, the item-total correlations of the PSS items ranged from 0.42 to 0.72, the average inter-item correlation was 0.41, and the alpha reliability was 0.88. Therefore, we chose the MIDI items that had correlations higher than 0.41 with the total PSS score as indicators of perceived stress. Seven items met this criterion for inclusion. The seven items are displayed in Table 1. In the current MTurk sample, the 7-item stress scale had item-total correlations ranging from

<table>
<thead>
<tr>
<th>Item</th>
<th>Total</th>
<th>Work</th>
<th>Finance</th>
<th>Relationship</th>
<th>Life in general</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. How would you rate the amount of control you have over your financial situation these days?</td>
<td>0.270</td>
<td>0.269</td>
<td>0.265</td>
<td>0.263</td>
<td>0.261</td>
</tr>
<tr>
<td>2. During the past year, how often have you thought your relationship might be in trouble?</td>
<td>0.270</td>
<td>0.269</td>
<td>0.265</td>
<td>0.263</td>
<td>0.261</td>
</tr>
<tr>
<td>3. How would you rate your life overall these days?</td>
<td>0.270</td>
<td>0.269</td>
<td>0.265</td>
<td>0.263</td>
<td>0.261</td>
</tr>
<tr>
<td>4. How would you rate how much control you have over your life in general?</td>
<td>0.270</td>
<td>0.269</td>
<td>0.265</td>
<td>0.263</td>
<td>0.261</td>
</tr>
</tbody>
</table>

Table 1. Items of the perceived stress measure developed from the survey in Midlife in the United States Survey (MIDUS)
0.30 to 0.74, the average inter-item correlation of 0.37, and alpha reliability of 0.79. The correlation between the total score of the 7-item stress measure and the PSS total score was 0.73 in the MTurk sample. In the MIDUS sample, Cronbach alphas were 0.74 at wave 1 and 0.75 at wave 2.

Statistical analysis
We used a series of latent variable models to test the cross-sectional and longitudinal associations and genetic and environmental etiology of personality traits and stress. Specifically, we first constructed latent variable models of personality and stress at each time point using Mplus 7.4 (Muthén & Muthén, 1998–2015). We used the four neuroticism items, four conscientiousness items, five extraversion items, and five agreeableness items as manifest indicators for the latent trait of neuroticism, conscientiousness, extraversion, and agreeableness. The seven openness items and the seven stress items were grouped into three parcels using the item-to-balance technique recommended by Little, Cunningham, Shahar, and Widaman (2002) to form three manifest indicators for the trait of openness and perceived stress (CFI ranged from 0.96 to 1). Next, we followed a similar procedure to model stability and change across time. The latent mean constructs from both waves were used to form the latent intercept and the latent mean construct from the second wave was used to form the latent slope, which represented changes of the variables over time (McArdle, 2009). The intercept and change parameters of each of the variables were set to correlate with each other. All the item loadings and item residual variance were fixed to be equivalent across the two waves. Each family was viewed as a cluster to account for the dependency within each pair of twins. From these models, we saved factor scores for personality and stress at each time point and for stability and change across time to reduce the number of parameters for the behaviour genetic models. We used these factor scores to investigate genetic and environmental influences on continuity and change of personality traits and stress over time.

Following the assumptions and methods described in study 1, we first constructed multivariate Cholesky structural equation models to examine the contribution of genetic effects, shared environmental and nonshared environmental effects to the cross-sectional relations between personality traits and stress. This first model was conducted in part to determine whether the same approximate pattern of relations replicated given the new measure of stress that was used. Second, we used a series of models to test the genetic and environmental influences on the continuity and change in both personality traits and stress over time. In the second model, we tested the extent to which the rank-order stability of stress was derived from genetic or environmental factors. The basic format of the models used to examine the genetic and environmental influences on the continuity of perceived stress was the same as the one shown in Figure 1 in which the construct assessed at both time points were entered into the model. In the third model, we constructed trivariate models to test the longitudinal relationships among each of the Big Five personality traits and perceived stress at time 2. Specifically, each of the five personality traits assessed at time 1 and time 2 and perceived stress at time 2 were entered into the models in order. In the fourth model, we used latent change modelling to specify changes in both personality traits and stress over time. We then tested the extent to which changes in personality traits and stress were the result of genetic or environmental factors. The fifth model tested the extent to which genetic or environmental factors accounted for the longitudinal stability of the relation between personality traits and stress over time. Finally, the sixth model decomposed the association between changes in personality traits and changes in stress into genetic and environmental components.

We tested sex limitation for the five personality traits and perceived stress at each wave. We found minimal evidence for sex limitation for any variable except perceived stress at the first time point which displayed modest quantitative sex limitation (see Table S6 and Table S7). To ensure that the personality associations reported below were not biased by this effect, we fit models in which the covariance between personality and stress was constrained to be equal across gender. For extraversion, agreeableness, neuroticism, and openness, this constraint did not significantly worsen model fit ($\Delta$CFI < $\Delta$RMSEA < 0.01). For conscientiousness, the constraint did significantly worsen model fit ($\Delta$CFI = 0.03, $\Delta$RMSEA = 0.01). As can be seen in Table S8, the association between conscientiousness and stress was equivalently because of genetic and environmental influences for males, but for females, the association was primarily due to genetic influences. Given this single difference which may reflect a false positive and our relatively low power to model this effect in multivariate models, we proceeded with models that did not differ across gender. Next, we constructed univariate structural equation models (ACE versus AE models) to decompose additive genetic influences, shared environmental and nonshared environmental influences on each of the factor scores specified above. If significant genetic effects were detected in the univariate analyses, the variables were included in the multivariate Cholesky decomposition to investigate the contribution of genetic and environmental influences to the covariance between personality traits and perceived stress at each wave, and the covariance between changes in personality traits and changes in perceived stress.

RESULTS
The phenotypic correlations between conscientiousness, neuroticism, extraversion, agreeableness, and openness and perceived stress were $-0.43$ [−0.50, −0.35], 0.57 [0.52, 0.62], $-0.39$ [−0.45, −0.32], −0.23 [−0.30, −0.17], and −0.27 [−0.34, −0.21] at time 1, and $-0.47$ [−0.58, −0.36], 0.51 [0.41, 0.61], $-0.46$ [−0.55, −0.37], −0.25 [−0.34, −0.15], and −0.31 [−0.40, −0.22] at time 2, respectively. For personality traits and perceived stress at both time 1 and time 2, the within-pair correlations for MZ twins were substantially larger than those for DZ twins, indicating the presence of genetic influences on personality traits and perceived
stress (see Table S9). Following procedures described in study 1, we conducted cross-sectional univariate analyses for each construct and multivariate analyses for the associations between the Big Five and perceived stress for both time 1 and time 2 data. Results are presented in Table S10 and S11 and Figure S1 and S2. Similar to study 1, the variance shared between personality traits and perceived stress was largely accounted for by genetic effects.3

Next we examined the stability of perceived stress in MZ and DZ twins, respectively. The phenotypic cross-twin cross-time correlations of perceived stress were 0.59 [0.43, 0.74] for MZ twins and 0.22 [0.05, 0.39] for DZ twins. The results from bivariate analyses indicated that about 41.0% of the total variance of perceived stress at time 2 was accounted for by genetic effects that contributed to variance of perceived stress at time 1, whereas only 3.6% of the total variance was due to genetic effects that contributed uniquely to perceived stress at time 2 (calculated the same way as shown in study 1). About 7.3% of the total variance of perceived stress at time 2 was explained by nonshared environmental effects that contributed to the variance of perceived stress at time 1, and 49.0% of the total variance was due to nonshared environmental unique to perceived stress at time 2. The genetic correlation between perceived stress at time 1 and at time 2 was 0.96 [0.81, 1], indicating that the same set of genes influenced perceived stress at the two time points. The nonshared environmental correlation was 0.36 [0.24, 0.46].

In terms of the stability of personality traits, the phenotypic cross-twin cross-time correlations of conscientiousness, neuroticism, extraversion, agreeableness, and openness were 0.68 [0.47, 0.90], 0.55 [0.41, 0.70], 0.49 [0.33, 0.65], 0.40 [0.23, 0.56], and 0.44 [0.27, 0.61] for MZ twins, and 0.20 [−0.01, 0.40], 0.22 [0.07, 0.37], 0.19 [0.03, 0.34], 0.16 [0.01, 0.31], and 0.25 [0.09, 0.41] for DZ twins. Table 2 displays the path coefficients for the genetic and nonshared environmental influences that are unique to the personality trait at time 1 and time 2 and perceived stress at time 2 (\(a_{12}, a_{22}, a_{23}, e_{12}, e_{22},\) and \(e_{23}\)), the path coefficients for the genetic and nonshared environmental influences shared between personality traits and time 1 and time 2 (\(a_{12} e_{12}\)), the coefficients for genetic and nonshared environmental influences shared between personality traits at time 1 and perceived stress at time 2 (\(a_{13} e_{13}\)), and personality traits at time 2 and nonshared stress at time 2 (\(e_{23}\)) after taking the influences of personality traits at time 1 into account. As shown in Table 2, the results from trivariate models indicated that about 24.0% to 34.8% of the total variance of personality traits at time 2 was explained by genetic effects that contributed to variance of personality traits at time 1, and 1% to 9.6% of the total variance of personality traits was explained by genetic effects unique to personality traits at time 2. About 4.8% to 22.1% of the variance of personality traits at time 2 was due to nonshared environmental effects that contributed to variance of personality traits at time 1, and 41.0% to 62.4% of the total variance was due to nonshared environmental effects unique to personality traits at time 2. The genetic correlations between personality traits at time 1 and at time 2 (\(r_g\) in Table 2) ranged from 0.84 [0.77, 1] to 0.98 [0.86, 1] and the nonshared environmental correlations (\(r_e\) in Table 2) ranged from 0.27 [0.16, 0.39] to 0.59 [0.50, 0.67].

For the longitudinal relations between each of the five personality traits and perceived stress, as shown in Table 2, about 6.3% to 10.9% of the total variance of perceived stress at time 2 was accounted for by genetic effects of personality traits at time 1. When the genetic influences of personality traits at time 1 were controlled, genetic factors of personality traits at time 2 explained only a small proportion (from 0.01% to 4.4%) of the total variance of perceived stress at time 2. About 29.2% to 38.4% of the total variance of perceived stress was due to genetic effects unique to perceived stress at time 2. The total variance of perceived stress explained by nonshared environmental effects was largely unique to that of perceived stress at time 2 (from 49.0% to 54.8%), but nonshared environmental effects of personality traits at time 1 only explained 0.04% to 1.7% of the total variance of perceived stress at time 2 and nonshared environmental factors of personality traits at time 2 explained 0.25% to 4.8% of it.

We used a multivariate Cholesky model to explore the genetic and nonshared environmental influences on the continuity in the relationship between personality traits and perceived stress. Figure 4 presents the path coefficients from the multivariate analyses of the association between personality at time 1 and perceived stress at time 2 (path coefficients between personality traits are shown in Table S5). As shown in the figure, about 21.4% of the total variance in perceived stress at time 2 was accounted for by genetic factors that contributed to the variance in personality at time 1, and 22.6% genetic contribution was unique to perceived stress at time 2. Nonshared environmental factors that influenced the variation in personality at time 1 contributed to only 2.9% of the total variance in perceived stress at time 2, and 53.1% of the total variation was due to nonshared environmental effects unique to perceived stress at time 2. In total, 88% of the variance in the continuity of the relationship between personality traits and perceived stress was attributable to genetic effects.

Finally, we constructed bivariate latent change models to examine the relationship between changes in personality traits and changes in perceived stress. The model fit indices suggested good model fit (CFI ranged from 0.94 to 0.99 and root mean square error of approximation [RMSEA] ranged from 0.03 to 0.06). The correlation between changes in conscientiousness, neuroticism, extraversion, agreeableness, and openness and changes in perceived stress were −0.31 [−0.43, −0.19], 0.24 [0.13, 0.35], −0.32 [−0.42, −0.22], −0.21 [−0.31, −0.10], and −0.24 [−0.34, −0.14], respectively. Table S9 presents the within-pair correlations

3We also compared ADE models with AE models for univariate analyses. Fit indices (AICs) only indicated better model fit for ADE than AE for extraversion in wave 1 and neuroticism in wave 2. We decomposed the correlation between extraversion and stress in wave 1 and results showed that additive genetic, dominance genetic, and environmental effects accounted for about 41%, 24%, and 34% of the association. For neuroticism and stress in wave 2, additive genetic, dominance genetic, and environmental effects accounted for about 36%, 11%, and 53% of the association.
for the factor scores of changes in the Big Five personality traits and perceived stress.

The results from the univariate model fitting analyses for changes in personality traits and perceived stress are presented in Table S12. The variance of changes in conscientiousness and neuroticism were totally due to the influences of environmental factors. Heritability estimate for changes in perceived stress was only 5%. For personality traits, only changes in extraversion, agreeableness, and openness demonstrated small effects of genetic factors, ranging from 6% to 13%.

We also constructed a multivariate model to examine the contribution of genetic and environmental factors to the covariance between personality traits at time 1 and perceived stress at time 2 in MIDUS twin sample. Figure 5 presents the path coefficients from the multivariate analyses of the associations between the factor scores of changes in personality traits and changes in perceived stress. Path coefficients between personality traits are shown in Table S5. According to the univariate analyses, about 5% of the variance in changes in perceived stress was accounted for by genetic effects. The multivariate analyses indicated that the genetic variation in changes in perceived stress was totally shared with genetic variance for changes in personality traits, and no genetic variance was unique to changes in perceived stress. In terms of the variance in changes in perceived stress that was accounted for by nonshared environmental effects, 81.4% of the total variance in changes in perceived stress was unique, and only the other 11.1% of the variation was accounted for by the nonshared environmental variance for changes in the Big Five personality traits. Thus, about 18.6% of the total variance in changes in perceived stress was shared with changes in personality traits, about 40.3% of which was due to genetic effects.

**SUMMARY**

Study 2 examined the contribution of genetic and environmental factors to the continuity and changes in perceived stress and the mediational role of genetic and environmental factors in the cross-sectional and longitudinal relationship between the Big Five personality traits and perceived stress. The cross-sectional association of personality traits and stress is...
largely genetic. Examining the longitudinal patterns, we found that the continuity in perceived stress was primarily accounted for by genetic influences, and nonshared environmental variation in perceived stress at time 2 was largely unique. For the continuity in the association between personality and perceived stress, the association between personality at time 1 and perceived stress at time 2 was mainly due to genetic factors. Nonshared environmental factors made the main contributions to changes in perceived stress and changes in personality traits. Environmental factors made greater contributions than genetic factors to the association between changes in personality and changes in perceived stress.

Results from study 1 and study 2 are summarized in Figure 6. About 65% to 80% of the associations between personality traits and perceived stress were attributable to genetic influences in the three cross-sectional models. Genetic variance contributed about 90% to the continuity in the relationship between personality traits and perceived stress. For the associations between changes in personality traits and changes in perceived stress, about 40% of the variation was explained by genetic influences. For the sample in study 1, perceived stress shared all the genetic variance with personality traits, but perceived stress only shared about half of its genetic variation with the genetic variation in personality traits in middle-aged adults in study 2. Changes in perceived stress shared all the genetic variance with changes in personality traits.

DISCUSSION

The current research examined the genetic and environmental contributions to the concurrent and longitudinal associations between personality and perceived stress. Across both

Figure 5. Multivariate analyses of the additive genetic and nonshared environmental contributions to the covariance between changes in personality traits and changes in perceived stress in MIDUS twin sample. The paths between personality traits are omitted. Co, conscientiousness; N, neuroticism; Ex, extraversion; Ag, agreeableness; O, openness; S, perceived stress; A, additive genetic factors; E, nonshared environmental factors.

Figure 6. Genetic and environmental shared with personality traits and genetic and environmental variance unique to perceived stress in add health, MIDUS I, and MIDUS II twin samples, the covariance between personality traits at time 1 and perceived stress at time 2 in MIDUS twin sample, and the covariance between changes in personality traits and changes in perceived stress in MIDUS twin sample.
studies, the concurrent relation between personality and perceived stress was largely due to genetic factors. In the longitudinal analyses, genetic factors accounted for the continuity in personality traits and perceived stress. And not surprisingly given, these two findings, the continuity in the association between personality and perceived stress over time, was largely explained by genetic factors. In contrast, environmental factors made larger contributions than genetic factors to the association between changes in personality traits and changes in perceived stress over time.

Given that the substantial genetic overlap between personality traits and perceived stress was found on both the cross-sectional and prospective basis in the present study, it is likely that as suggested by the sociogenomic model of personality traits, the state measure of perceived stress also captured a trait component that shared common genetic variance with the Big Five personality traits. Because of the genetic components contained in the measures of states, it is possible that genetic overlap commonly serve as the mechanism underlying the phenotypical associations between personality traits and states. Genetic effects on personality traits drive genetic effects on environmental experiences (Krueger & Johnson, 2008), thus, genetic factors of personality traits partly shape people’s momentary thoughts, feelings, and behaviours. Similar to our findings, happiness was found to share a general genetic factor with the Big Five personality traits (Weiss, Bates, & Luciano, 2008), and the results were interpreted as indicating that genetic effects of personality traits create an affective reserve that can be called upon in times of stress and recovery.

More generally, the current results are consistent with several plausible developmental mechanisms. Genetic factors may independently influence both personality and perceived stress by exerting pleiotropic effects. Alternative, genetic influences may affect personality which in turn impacts the appraisal or experience of stress. Finally, genetically influenced variance in personality may systematically expose individuals to certain life experiences that raise or lower stress. This pathway is consistent with gene–environment correlation. To the extent that the association between personality and perceived stress was mediated by the nonshared environment (which was a small portion of the association), this common variance may derive from random (i.e. nonsystematic) environmental experiences or possibly due to gene-by-nonshared environmental interactions (i.e. individuals responding to unique life events differentially on the basis of genetically influenced characteristics).

Across both studies, the genetic components in conscientiousness and neuroticism made substantial contributions to the genetic variance in perceived stress. It is likely that both conscientiousness and perceived stress overlap because of their shared control as a core component of their definitions (Roberts, Jackson, Fayard, Edmonds, & Meints, 2009). People high in conscientiousness feel in control, whereas those who are stressed do not. High conscientiousness may also immunize against perceptions of stress through impulsivity control, planning, and goal-directed behaviours which enable individuals to have a sense of control of the situations. Similarly, it is likely that the strong overlap between neuroticism and perceived stress reflects the common emphasis on negative affect underlying both constructs (Clark & Watson, 1999). As neuroticism is viewed as deriving from a highly active behavioural inhibition system that increases sensitivity to signs of threat or punishment (Gray, 1987), individuals with high levels of neuroticism tend to view the world in a negative light, making them more likely to appraise the situations as stressful. Compared with conscientiousness and neuroticism, extraversion and agreeableness displayed smaller genetic contributions to perceived stress. Extraversion and agreeableness may contribute to reduced levels of perceived stress because of the tendency to gain perceived or actual social support as resources to cope with stressful situations (Bowling, Beehr, & Swader, 2005; Swickert, Rosentreter, Hittner, & Mushruf, 2002), as perceived stress results from the perceived imbalance between demands and resources. When the other four personality traits were controlled, openness showed negligible genetic associations with perceived stress. Future research that employs more thorough assessments of both personality traits and stress may profit from exploring the higher-order relations between personality and stress (e.g. DeYoung, 2006). On the other hand, future research may also explore the link between facets of personality traits and stress given that for some personality traits, such as conscientiousness, neuroticism, and agreeableness, evidence was found for specific genetic and environmental influences on facets that were not accounted for by the operation of genetic and environmental influences on higher-order traits (Briley & Tucker-Drob, 2012).

Genetic overlap may imply different underlying ways that two constructs are related to each other. In addition to the shared common genetic basis, the presence of genetic correlations may also suggest that one genetically influenced trait contributes indirectly and phenotypically to the development of another (Johnson, Penke, & Spinath, 2011). It is possible that genetic influence on personality traits contributes to the perception of stress over time, and conversely, in the long run, genetic component of perceived stress is also likely to play a role in the development of personality traits. The current research contributes to prior work by first showing that shared genetic factors are a significant third variable helping to define the cross-sectional link between personality traits and perceived stress. Moreover, we also found that in the sample of young adults (i.e. study 1), almost no genetic variation in perceived stress was unique to stress itself. This result indicates that the experience of stress is not only attributable to personality factors, but that genetic factors underlying both of these constructs are the source of that covariation. However, perceived stress only shared about half of its genetic variation with the genetic variation in personality traits in middle-aged adults (i.e. study 2). Future research should seek to identify potentially developmental pathways that may lead genetic influences on the perception of stress to decouple from personality.

The results examining the continuity of perceived stress suggested that stability in perceived stress was primarily a function of genetic influences. Nonshared environmental factors also contributed, albeit in a small way, to the continuity of perceived stress. The high genetic component of the test–
retest correlation in perceived stress is similar to that found for personality traits. Traditionally, perceived stress is viewed as a state that displays fluctuations from moment to moment so that there is no need to examine its long-term trajectory. However, the results from our study indicates that a large proportion of genetic effects contributed to the continuity of perceived stress over a 10-year period of time, implying the presence of stable trait variance in the state of perceived stress. Thus, the consistent large effect of genetic factors on that portion of personality and stress that is stable over time should not come as a complete surprise. This merely reflects the fact that genes and some part of their effects are invariant over time and that environmental events typical in passive observational studies are not so overpowering as to wipe out the consistent genetic signal over time.

It is not uncommon for genetically informed research to only examine the cross-sectional genetic overlap and at most the genetic contribution to stability over time (Krueger, Johnson, & Kling, 2006). Stopping at these two components of the association between personality traits and stress would leave the incorrect impression that the overlap is largely dictated by genes. However, decomposing the patterns of both continuity and change over time reveals a more complex picture. First, there were relatively large nonshared environmental factors unique to perceived stress at each specific time of assessment. Thus, environmental factors, such as stressors encountered at a specific time point, had substantial influences on individuals’ subjective experiences of stress but the effects were not likely to be long-lasting. This would indicate that the experience of stress, while overlapping with a genetic factor common to personality traits, was still widely influenced by environmental circumstances.

Moreover, when we examined changes in perceived stress, the majority of the variance was attributable to environmental factors. Thus, nonshared environmental factors were a primary source of variance in changes in perceived stress over the 10-year period in the current sample. This reflects the influence of the dynamics of the environment that were not shared among twins and that created differences between them. One possible nonshared environmental component may be the experience of different major stressors. As suggested, such stressors may include physiological stressors, such as a disease diagnosis, the associated demands related to disease progression and treatment, and role changes. Moreover, in the dynamic process, one stressor may lead to others, and the loss of resources associated with the stressor is likely to cause further loss (Segerstrom & O’Connor, 2012). The differences in life experience over years for this middle aged sample might lead them to develop different appraisals and perceptions of situations.

In terms of the associations between changes in personality and changes in perceived stress, like previous research, we found substantial links between changes in specific personality traits, such as neuroticism, and changes in perceived stress (Luo & Roberts, 2015). Akin to the cross-sectional analyses, we first tested the extent to which the longitudinal relation between personality traits and stress was genetic or environmental in origin. Environmental factors made larger contributions than genetic factors in mediating such associations. This would appear to indicate that both genetically and environmentally driven changes over time create a stronger developmental covariance between personality traits and stress, with environmental factors playing a major role. Just as future research would attempt to identify common genetic factors, the current research indicates that common environmental factors contributing to changes in both personality traits and stress should also be investigated.

There are several limitations to the current research. First, across both studies, we found little evidence that the patterns of genetic and environmental influence differed across gender. In MIDUS at one time point, but not the other, we found evidence of sex-limitation for perceived stress. More research is needed to identify whether this was a false positive, as we did not find evidence of sex-limitation for the two other measures of perceived stress, or possibly a result of some substantive difference, such as the MIDUS sample being 10 years younger at the initial time point. Sample attrition may also play a role. Similarly, we found that the genetic and environmental sources of the association between conscientiousness and stress differed by gender at this time point. Again, more research is needed to confirm this finding.

We used twin models that assume the effects of genes and environments are independent. This assumption can be violated as people do select their own environmental circumstances or are selected into certain situations because of their own characteristics (Roberts et al., 2008). However, the absence of enough information about the pertinent environments makes modelling this process challenging. Also, power is likely too low to model gene–environment interactions given the sample sizes in the current study. For example, Tucker-Drob and Bates (2016) estimated that roughly 3000 pairs would be necessary to achieve 80% for one of the larger examples of gene–environment interaction found in the literature. Larger sample sizes would allow modelling gene–environment interplay to reveal a more comprehensive picture of the relationship between personality and perceived stress (Purcell, 2002). Incorporating life events into behaviour genetic models linking personality and stress represents a promising area for future research. For example, it may be the case that the link between personality and stress emerges through selection into certain environments, a form of gene–environment correlation. By measuring life events along with personality and stress in a longitudinal, genetically informative sample, it would be possible to assess the extent to which the association drives perceived stress development compared with personality development, and the extent to which these pathways are mediated through the environment. Additionally, life events may also moderate the genetic and environmental influences on personality and stress, as well as the association between personality and stress. For example, genetic factors may be especially important during stressful life events as such genetically influenced characteristics could alter how individuals respond to the event.
Despite the strengths of employing a longitudinal design to examine the dynamic relation between personality and perceived stress, the data available were only based on assessment at two time points. It is possible that changes estimated based on two time points reflected regression to the mean, and it is difficult to predict the effect that such regression to the mean would play for associations across stress and personality. Future research should include multiple waves to detect the influence of personality on perceived stress over the life span in a more reliable way. The personality inventories used in the current study were relatively short and displayed relatively low levels of reliability. Future work using more extensive surveys would be beneficial to enhance reliability and investigate facet-level associations. We also constructed our PSS in MIDUS based on available items, rather than a psychometrically validated scale. We conducted validity tests using an MTurk sample, which tend to sample a wide range of adult participants (e.g., Buhrmester, Kwang, & Gosling, 2011). However, we were not able to perfectly match the demographics of the MTurk sample with the MIDUS sample. Additionally, part of the association between personality and perceived stress may result from somewhat similar assessment indicators, such as specific items of neuroticism. More extensive scales would allow for testing whether facets without such content are similarly associated, or allow for more complex modelling options to assess construct validity. Finally, the use of self-report may cause the nonshared environmental effects to be confounded with measurement error.

The present research adopted a behaviour genetic design to improve our understanding of the underlying the relation between personality traits and perceived stress. The longitudinal design also contributed to the current literature by revealing the mechanisms underlying changes in perceived and the mechanisms underlying the dynamic association between personality and perceived stress over time. There is a need for future research to provide more in-depth understandings of how individual differences in the experience of stress are influenced by genetic and environmental pathways of development.

SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

Table S1. Model fit and model comparison indices of sex limitation tests for the Big Five and perceived stress using Add Health twin sample.
Table S2. Within-pair correlations for the Big Five personality traits and perceived stress in Add Health twin Sample.
Table S3. Parameter estimates (95% confidence intervals) for the Big Five and perceived stress for the univariate ACE and AE models using Add Health twin sample.
Table S4. Parameter estimates for the genetic and environmental etiology of the associations between personality traits and perceived stress using Add Health twin sample.

Table S5. Path coefficients between personality traits in the multivariate analyses displayed in Figures 3, 4, and 5.
Table S6. Model fit and model comparison indices of sex limitation tests for the Big Five and perceived stress using MIDUS twin sample at Time 1.
Table S7. Model fit and model comparison indices of sex limitation tests for the Big Five and perceived stress using MIDUS twin sample at Time 2.
Table S8.
Table S9. Within-pair correlations for the Big Five personality traits and perceived stress in MIDUS twin sample.
Table S10. Parameter estimates (95% confidence intervals) of the Big Five and perceived stress for the univariate ACE and AE models using MIDUS twin sample at Time 1.
Table S11. Parameter estimates (95% confidence intervals) of the Big Five and perceived stress for the univariate ACE and AE models using MIDUS twin sample at Time 2.
Table S12. Parameter estimates (95% confidence intervals) for changes of the Big Five and perceived stress for the univariate ACE and AE models using MIDUS twin sample.

Figure S1. Multivariate analyses of the additive genetic and nonshared environmental contributions to the covariance between personality traits and perceived stress at Time 1 in MIDUS twin sample. The paths between personality traits are omitted. Co = conscientiousness; N = neuroticism; Ex = extraversion; Ag = agreeableness; O = openness; S = perceived stress; A = additive genetic factors; C = shared environmental factors; E = nonshared environmental factors.

Figure S2. Multivariate analyses of the additive genetic and nonshared environmental contributions to the covariance between personality traits and perceived stress at Time 2 in MIDUS twin sample. The paths between personality traits are omitted. Co = conscientiousness; N = neuroticism; Ex = extraversion; Ag = agreeableness; O = openness; S = perceived stress; A = additive genetic factors; C = shared environmental factors; E = nonshared environmental factors.

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