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Kids becoming less alike: A behavioral genetic analysis of developmental increases in personality variance from childhood to adolescence

René Mõttus[1]
Daniel A. Briley[2]
Anqing Zheng[2]
Frank D. Mann[3]
Laura E. Engelhardt[3]
Jennifer L. Tackett[4]
K. Paige Harden[3]
Elliot M. Tucker-Drob[3]

[1]University of Edinburgh and University of Tartu
[2]University of Illinois at Urbana-Champaign
[3]University of Texas at Austin
[4]Northwestern University

Address Correspondences to:
René Mõttus (rene.mottus@ed.ac.uk) or Elliot M. Tucker-Drob (tuckerdrob@utexas.edu)

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Abstract

Recent work in personality development has indicated that the magnitude of individual differences in personality increases over child development. Do such patterns reflect the differentiation of individuals by genotype, an increasing influence of environmental factors, or some (interactive) combination of the two? Using a population-based sample of over 2,500 twins and multiples from the Texas Twin Project, we estimated age trends in the variances in self- and parent-reported measures of the Big Five personality traits between ages eight and eighteen years. We then estimated age trends in the genetic and environmental components of variance in each measure. Individual differences in personality increased in magnitude from childhood through mid-adolescence. This pattern emerged using both children's self-reports and ratings provided by their parents, and was primarily attributable to increases in the magnitude of genetic influences. Most of the increasing genetic variance appeared non-additive, pointing to the possibility that developmental processes tend to make genetically similar individuals disproportionately more alike in their personality traits over time. These findings could reflect increasing or accumulating effects of trait-by-trait interactions; person-by-environment transactions whereby genetically similar people are disproportionally likely to experience similar environments; the activation of dominant genes across developmental transitions (e.g., puberty); or some combination of these three processes, among other factors. Theories of personality development will need to accommodate these descriptive findings, and longitudinal, genetically informed designs are needed to test some of the specific hypotheses springing from this study.

Keywords: Development; Personality; Variance; Behavioral Genetics; Non-additive

Kids becoming less alike: A behavioral genetic analysis of developmental increases in personality variance from ages 8 to 18 years

A common strategy in science is to describe and then explain (Simon, 1992; Salthouse, 2005). Accordingly, a key goal of research in personality development is to comprehensively document *how* measures of personality change with age in order to formulate, refine, and eventually test hypotheses regarding *why* these changes occur. For example, a substantial body of research has examined developmental trends in mean levels (Roberts, Walton, & Viechtbauer, 2006) and rank-order stability of personality traits over time (Roberts & DelVecchio, 2000). This research has resulted in two highly influential descriptive principles: The maturity principle holds that individuals become increasingly socially mature with age, and the cumulative continuity principle holds that individuals become increasingly consistent in their personality trait levels over time (Caspi, Roberts & Shiner, 2005; Fraley & Roberts, 2005). Until recently, however, little attention has been paid to how *inter-individual variation* in personality traits might change with age. Given that the very concept of personality is based on the observation that people systematically differ from one another in their patterns of thinking, feeling, and behaving, whether the magnitude of such individual differences changes with age would seem a fundamental descriptive question. Along with other descriptive information, such as the shape and direction of mean age trends in different personality traits, empirical results that answer this question can constrain explanatory models of personality development.

Age-differences in variance: existing evidence

In perhaps the first explicit treatment of the question, Mõttus and colleagues (2016) compared the variances of the Five-Factor Model (FFM; McCrae & John, 1992) domain and facet scores across age groups, using self- and informant-report data from Estonia, the Czech Republic, and Russia. No systematic age differences in variance were observed when comparing late adolescents (ages 16 to 20)

to emerging adults (ages 21 to 25) or when comparing young adults (ages 20 to 30) to middle-aged adults (ages 50 to 60). Although these results did not provide evidence that personality trait variance changes from adolescence through middle adulthood, they did not address the possibility of such changes over more circumscribed developmental stages such as childhood.

In a follow-up study based on parental reports of children and adolescents, Mõttus, Soto and Slobodskaya (2017) found that variance in personality increased from early childhood until early adolescence and then plateaued. Specifically, this pattern held for all Little Six personality scales (Soto & John, 2014), except for Extraversion, in a large sample of mostly English-speaking parents. Moreover, the pattern applied to most items of these scales—items were interpreted as being reflective of personality nuances (McCrae, 2015; Mõttus, Kandler, Bleidorn, Riemann, & McCrae, 2017)—as well as to most other personality items that had been administered but were not included in the Little Six. These robust findings did not appear to be an artifact of mean-level changes. On the contrary, the increases in variance were informative for interpreting mean-level trends. For example, an examination of age differences in the distribution of Activity scores revealed that the downward mean-level trend in this trait was partly due to increasing prevalence of lower values. In other words, some children declined in activity while others retained their relative level of the trait with age, shifting the average downward while simultaneously increasing variability across individuals. Furthermore, the tendency for increasing variance until adolescence was largely replicated in an analysis of parent-rated personality in a sample of Russian children and adolescents (Mõttus et al., 2017).

It is worth noting that increases in variance over childhood and adolescence are unlikely to be exclusive to personality characteristics. Possibly among other phenomena, similar tendencies appear for height (Tanner, Whitehouse, & Takaishi, 1966) and scholastic achievement (NWEA, 2015). However, developmental trends in variance remain poorly documented for any stage of life for most

psychological phenomena. This may partly be because of widespread tendencies to transform psychological measurements according to age-specific norms and/or to use standardized effect sizes to represent associations; both of these practices have very good reasons, but they also render potential age differences in variance invisible.

Explaining increasing personality variance in childhood

Several explanations are possible for the pattern of increasing personality variance in childhood (Mõttus et al., 2017). First, it is possible that these changes do not reflect the development of personality *per se*, but changes in how personality becomes observable. For example, children expand their repertoire of cognitive, emotional, and self-regulatory capacities as they develop, which may provide older children with more ways to express their distinctive personality and give observers more cues to detect individual differences among them. Likewise, older children may have a clearer sense of their distinctive identity, which may further promote the expression of individual differences. These increases in the ability to express one's personality, and individual differences in this, may be both genetic and environmental in origin.

Second, the pattern of increasing personality variance is also consistent with the hypothesis of intrinsic, genetically-driven maturation of innate dispositions or basic tendencies, in line with the Five-Factor Theory (FFT; McCrae & Costa, 2008; Mõttus, 2017). It might take time for basic tendencies to achieve their full extents, similarly to how it takes time for individuals to achieve their adult height; notably, variance in height follows a pattern similar to that of personality traits (Tanner, Whitehouse, & Takaishi, 1966). Such intrinsic maturation would not require personality trait development to have any environmental input, apart from a minimum level of "average expectable environment" or "good enough environment" (Scarr, 1992).

Third, external factors, including environmental contexts and social roles, may contribute to the differentiation of personality traits over development. With advancing age, children are less closely supervised by their parents and teachers (Patterson & Stouthamer-Loeber, 1984), such that older children have more freedom to seek out, create or evoke situations that allow them to express their distinctive personalities. Over time, these bespoke experiences may stabilize or reinforce the personality characteristics that led to them in the first place (Caspi & Roberts, 2001; Scarr & McCartney, 1983; Tucker-Drob & Harden, 2012; Roberts & Nickel, 2017). Such person-environment transactions are sometimes referred to as the niche-picking principle, whereby experiences stabilize personality characteristics (Roberts & Nickel, 2017), or the corresponsive principle, whereby experiences reinforce personality characteristics (Caspi & Roberts, 2001; Caspi, Roberts, & Shiner, 2005). In other words, individual differences in personality characteristics may increase over time because children are likely to accumulate opportunities to experience environments that reinforce genetic predispositions. In contrast to the intrinsic maturation hypothesis (i.e., the FFT), the corresponsive principle based (person-environment transactional) hypothesis requires access to an assorted "cafeteria of experience" (Lykken et al., 1993) from which to select and evoke suitable environments. In other words, the corresponsive account allows for environmental experiences to have a systematic role in the development of traits, whereas the FFT strictly postulates no role for them (perhaps barring extreme cases of neglect and abuse). And yet both hypotheses see personality development as a gradual crystallization or amplification of pre-existing characteristics.

Finally, it is also possible that increasing personality variance results from entirely nongenetic factors. Specifically, the social situations and life paths that are relevant for the magnitude of individual differences in personality may occur at random, especially in earlier stages of life. Childhood and adolescence are periods in which individuals experiment with new behavioral repertoires, roles, identities, and ways of relating to other people (Briley & Tucker-Drob, 2017). Both the impetus for

these experimentations and the behavioral repertoires individuals eventually settle at may result from happenstance, unsystematic experiences such as new friendships or chance encounters. One could think of this aspect of personality development as a "random walk": Individuals try something new and develop a set of relevant characteristics, then try something else and develop (some of the) personality characteristics relevant for these experiences, and so forth. Such a model would imply that personality development is not a consolidation of pre-existing characteristics, but rather a process of environmentally mediated "innovation" (cf. Briley & Tucker-Drob, 2013).

Evidence from animal models, in which genetic and environmental controls can be experimentally implemented, are consistent with the possibility of random walk effects on increasing variance. For example, Freund and colleagues (2013) bred genetically identical mice and reared them in a large, open environment. Despite all individuals possessing identical genetic material and residing in the same environment, individual differences emerged over time, potentially due to the unique social exchanges across chance encounters in the environment as the mice were free to explore. More intriguing still, increasing variance may also result from entirely stochastic processes. Illustrating the importance of randomness, a study by Bierbach, Laskowski, and Wolf (2017) raised genetically identical fish in identical rearing environments for varying amounts of time. Variance increased with development at approximately the same rate regardless of whether the fish were reared entirely in isolation, with other fish for a week, or with other fish for nearly a month. It therefore appears possible that social interaction is not a necessary ingredient of increasing variance, which may rather result from idiosyncratic maturational events (cf. Molenaar, Boomsma, & Dolan, 1993).

In addition to mechanisms that could contribute to increases in personality variance, there may also be mechanisms that countervail them. For example, the typical mean-level changes that occur during adulthood—most people becoming more socially dominant, agreeable, conscientious, and

emotionally stable with age—are often thought to be driven by socialization processes: pressures to behave in socially acceptable and adaptive ways (Caspi et al., 2005). It is possible that these processes tend to make all individuals more alike or more similar to the "average" person.

Regardless of what specifically drives the increases in the magnitude of individual differences in childhood, the *shape* of the trend may also inform research on personality development. Mõttus and colleagues (2017) found that increases in the variance of the majority of personality traits happened before middle adolescence, suggesting that critical processes of personality development may take place earlier in the life course than when they are typically studied. For example, maturation of self-regulatory processes has often been studied in adolescence (Denissen, Wood, Penke & van Aken, 2013), and person-environment transactions have often been studied in late adolescence and early adulthood (Bleidorn, 2015). But the curvilinear pattern of variance increases points to the possibility that some developmental processes may already have become less relevant by middle adolescence. Alternatively, the countervailing forces described above may have started to offset the expansion of personality variance.

Behavioral genetic decomposition of variance in personality traits

With the observable (phenotypic) increases in personality variance over childhood and adolescence described in one previous study, the current study also sought to estimate the extents to which genetic and environmental factors contribute to this pattern. We employed behavioral genetic models, which capitalize on the relationship between genetic relatedness [e.g., identical or monozygotic (MZ) twins *vs.* fraternal or dizygotic (DZ) twins] and similarity on a trait (phenotype) in order to make inferences about the extent to which the trait is influenced by genetic and environmental factors. For example, the extent to which individuals who share all of their genetic variants (i.e., MZ twins) are more similar in their extraversion than are individuals who share, on average, about half of their

segregating genetic variants (e.g., DZ twins) is an index of the degree to which extraversion is influenced by genetic factors. When MZ twins are more than twice as similar on a trait relative to DZ twins, the trait could be influenced by genetic factors in a non-additive manner, with alleles interacting within and/or across genetic loci (behavioral genetic models typically address only within-genetic loci interactions, although we also considered across-loci interactions). There is substantial evidence of non-additive genetic variance in personality traits (Boomsma et al., 2017; Keller et al., 2005; Saudino, 1997; Tellegen et al., 1988; Vukasovic & Bratko, 2015). We note, however, that behavioral genetic models may also give an impression of non-additive genetic variance for reasons other than interactions at the level of genes themselves. For example, genetically very close individuals may be disproportionately similar in personality traits when genetically separable components of these traits interact with one another over development (Mõttus & Allerhand, 2017).

Behavioral genetic predictions for increasing trait variance

To the extent that the corresponsive principle holds for personality development, we would expect that genetically influenced personality characteristics become amplified over time as individuals seek out and evoke experiences that reinforce their genetic differences, a phenomenon also known as "genetic amplification" or the "genetic multiplier effect" in the context of cognitive abilities (Briley & Tucker-Drob, 2013; Tucker-Drob, Briley, & Harden, 2013; Dickens & Flynn, 2001). If so, the genetic variance components of personality traits should increase in magnitude with age (Purcell, 2002; Kandler & Zapko-Willmes, 2017), although it is important to stress that this would reflect the accumulation of genetically influenced environmental experiences, rather than direct genetic influences becoming stronger over time. According to this model, there is no *a priori* reason to expect concomitant decreases (or increases) in environmental variance.

With respect to the intrinsic maturation hypothesis (i.e., the FFT), there is no reason to hypothesize that observable increases in the magnitude of individual differences should result from any form of environmental influences. Instead, increasing observable variance would reflect personality becoming increasingly governed by genetically programmed processes, which would be represented by increases in genetic variance and concomitant decreases in environmental variance (the latter being essentially developmental noise and measurement error). In short, the "nature" should increasingly shine through any other influences.

In contrast, to the extent that the random walk-like processes apply to personality development, these are not systematically linked to genetically influenced dispositions and therefore genetically influenced characteristics would not necessarily develop in their pre-existing directions. Instead, genetic influences would serve as a stabilizing backdrop for the accumulation of environmental variance (i.e., environmental mold; Cattell, 1946) that would drive increases in the magnitude of observable individual differences (Mõttus et al., 2017). This possibility is in line with the relatively modest phenotypic stability of personality in childhood and adolescence (Roberts & DelVecchio, 2000). Under such a model, socialization processes may also override pre-existing genetic influences, thereby possibly even reducing the magnitude of genetic variation in personality with age.

As a final alternative, any mechanisms contributing to the increases of personality variance may flow through observable traits only, rather than emerging distinctly from genetic or environmental factors. Whether someone has a genetic or environmental predilection for high or low trait scores may not impact the processes that matter to their development. Turkheimer, Petterson, and Horn (2014) labeled this perspective as the "phenotypic null hypothesis" for personality. If this holds, both genetically influenced and environmentally acquired aspects of personality are only tools that help organisms respond to whatever demands they encounter. Put differently, social pressures on trait

development (i.e., obtaining and maintaining a job or earning good grades) may not care whether someone has high conscientiousness for genetic or environmental reasons, and similarly, evolutionary pressures cannot act directly on genotype. Organisms respond to such pressures on the basis of their phenotypic traits. Bosses and teachers care about the level of observable conscientiousness, not genetic conscientiousness. This entails a testable hypothesis: In the present context, it would imply that any age trends in variance components should be proportional across genetic and environmental sources of variance because age trends in total variance occur through the observable traits alone.

Existing evidence for changes in genetic and environmental variance

Previous behavioral genetic work on developmental trends has predominately focused on the relative *proportions* of genetic and environmental variance, and have not paid much attention to changes in raw variance. In principle, such findings could provide hints about what patterns might be expected from age trends in raw variance contributions. For example, if heritability of traits decreases with age, it seems less plausible that increases in oberved variance are driven by the amplification of genetic variance. However, the existent results are inconsistent: although the relative contribution of genetic factors appears to robustly increase over childhood and adolescence for some psychological phenomena such as intelligence (Tucker-Drob, Briley, & Harden, 2013), meta-analytic findings indicate contrasting patterns for personality characteristics. Specifically, one meta-analysis (Briley & Tucker-Drob, 2014) found that heritability of personality traits generally decreases from early childhood to adolescence, whereas another meta-analysis reported increasing heritability estimates for this period of life (except for agreeableness; Kandler & Papendick, 2017). The former pattern would be more consistent with the random walk hypothesis and the latter with the intrinsic maturation or corresponsive principle based hypotheses.

However, these two meta-analyses had some important limitations. First, by virtue of compiling data across a range of studies, different measures were used in different age groups that could have been differentially sensitive to genetic and environmental influences. For example, decreases in heritability observed by Briley and Tucker-Drob (2014) may not have stemmed from developmental shifts in genetic and environmental contributions to personality variance *per se*, but from differences between personality measures that were socially contextualized in middle childhood and beyond, but more context-free and temperament-focused in younger samples. The trends may have also resulted from shifting from the parent-report method in early childhood samples to the self-report method in middle childhood samples and beyond. Indeed, Briley and Tucker-Drob (2014) found that when effect sizes derived from parent-reported measures were excluded from their meta-analysis, age trends in heritability flattened, although the increases in environmental contributions to rank-order stability largely remained. Notably, the meta-analysis by Kandler and Papendick (2017) relied mostly on self-reported personality as of age nine and found evidence for generally increasing personality trait heritability in children and adolescence. Second, pooling of effect sizes across studies required scaling results to a standardized metric, such that age-related differences in raw variance could not be examined. Individual studies that implement consistent personality measurements across a wider age range, such as the current study, are necessary to more conclusively test for genetic and environmental contributions to age trends in personality variation.

The present study

In the present study, we used data from a large sample of child and adolescent twins who provided self-ratings of their Big Five personality traits and who were also rated by a parent; we considering the Big Five traits as paralleling those of the FFM. We decomposed the variance in personality traits into genetic and environmental components and estimated developmental trajectories

in the magnitudes of these components. As the functional form of age trends in variance has been found to be nonlinear with a plateau in late adolescence (Mõttus et al., 2017), we supplemented more parsimonious and powerful parametric techniques with a non-parametric approach capable of identifying nonlinearities.

We expected increases in observed variance, but we did not endorse any *a priori* hypotheses concerning whether this increase was driven by increases in genetic variance, environmental variance, or a relatively equal combination of these components. As discussed above, opposing explanations for personality development entail different hypotheses, so we expected the results to be informative as to the *relative* plausibility of these explanations. We had no reason to expect variance patterns to differ across personality traits. Therefore, we would interpret the robustness of the overall conclusions in the context of consistency across traits.

Methods

Participants

The data for the current study were collected as part of the Texas Twin Project (TTP; Harden, Tucker-Drob, & Tackett, 2013), an ethnically and socioeconomically diverse sample of school-aged (primarily ages 8 to 18 years) twins or other types of multiples and their parents. Families were recruited using public school directory information. The TTP includes several subprojects that have collected both child- and parent-reports of children's Big Five personality traits, among other measurements. The TTP subprojects were approved by the Institutional Review Board of the University of Texas at Austin as projects 2009-12-0040 ("A Sibling and Twin Study of Healthy Development in Children and Adolescents"), 2011-11-0066 ("A Twin Study of Healthy Development in Infants and Young Children"), 2011-11-0067 ("Genetic Influences on Adolescent Decision-Making and Alcohol Use"), 2013-02-0011 ("The Genes and Development Study"), 2014-11-0021 ("Cortisol,

Socioeconomic Status, and Genetic Influence on Cognitive Development"), and 2016-01-0004 ("Genetic & Hormonal Influences on Adolescent Decision- Making"); see Online Supplement for additional details of each subproject. In total, we obtained observations for 2,913 children, including 2,640 child-reports and 2,456 parent-reports; data from both parent and child was available for 2,183 individuals. Missing data occurred for several reasons: non-response on the part of the parent (N = 457); time constraints for in-lab child reports (N = 13); for one subproject, self-reported personality was not obtained for children in 5th grade or below (roughly age 10 years; N = 235); and non-response due to other reasons on the part of the child (N = 25). The sample included 358 child participants that provided longitudinal data, typically resulting from children reaching an age that made them eligible to participate in a new subproject. Due to the relatively small number of repeated measurements, we did not fit models of change over time. We treated all observations as cross-sectional and used an estimation method that corrected standard errors for the non-independence due to repeated measurements of the same individuals over time (further description below).

The participants came from 1,225 unique families. The dataset included 38 families that only contributed singleton observations (due to non-participation from siblings), 34 families with triplets, four families with multiple twin pairs, two families with quadruplets, with the remaining families comprised of twin pairs. A total of 962 families contributed two observations each (a single time-point for each twin pair), 31 families contributed three observations each (triplets, as well as twin families that participated incompletely at multiple time points), 159 families contributed four observations (twin pairs that contributed two waves of data, as well as quadruplets), two families contributed five observations each (one family contained two twin pairs with an incomplete longitudinal assessment and the other family contained one twin pair with two complete waves and one incomplete), 27 families contributed six observations each (twins that contributed three waves or triplets that contributed two waves of data), five families contributed eight observations each (all twins that contributed four waves

of data), and finally, one family contributed ten observations (triplets that incompletely participated at multiple waves). As behavior genetic models treat sibling pairs rather than single individuals as the units of analysis, models included all possible within-time point combinations of sibling pairs, which included triplets and higher-order multiples. For example, triplets could contribute three pairs to the analysis (Sibling 1 with Sibling 2; Sibling 1 with Sibling 3; Sibling 2 with Sibling 3). We corrected for the non-independence of observations due to constructing all possible sibling pairs (described below). For brevity, we henceforth refer to all multiples as twins.

The sample ranged in age from 3.74 to 21.29 years (M = 13.19 years, SD = 3.21). Over 90% of participants were between 8 and 18 years old. Figure 1 presents a histogram of the age distribution. We included all available data in our analyses but primarily interpret our results in reference to the 8 to 18 years age range of high data density. The full sample was 50.08% female and was composed of 660 Hispanic, 1974 White, 351 Black, 19 Native American, 181 Asian, and 38 some other race/ethnicity participants. Note these values sum to more than the total sample size because participants were instructed to select all races/ethnicities that applied, with 367 participants selecting more than one race/ethnicity.

The mothers of the focal children had obtained varying levels of education: less than high school (2.29%), a high school degree (6.28%), some college (21.95%), a college degree (37.23%), some graduate training (4.59%), a master's degree (19.16%), or a doctorate/professional degree (8.50%). Similarly, fathers of the focal children had obtained varying levels of education: less than high school (4.32%), a high school degree (10.48%), some college (18.94%), a college degree (35.87%), some graduate training (2.74%), a master's degree (17.21%), or a doctorate/professional degree (10.44%). According to census estimates (U.S. Census Bureau, 2012-2016), Texas residents older than 25 have obtained: less than high school (17.23% for males and 17.11% for females), a high school degree

(25.54% for males and 24.66% for females), some college (21.75% for males and 22.96% for females), a college degree (24.46% for males and 26.06% for females), or a graduate or professional degree (10.03% for males and 9.22% for females). Of course, the census estimates do not exactly match the target population (i.e., those with twins or multiples of school age). For instance, the census estimates include older (grandparent-aged) adults for which rates of education are generally lower. With this caveat in mind, the current sample matches the general Texan population fairly well, with somewhat higher levels of college and professional degrees. As another indicator of socioeconomic diversity, parents were asked if they had used a form of need-based public assistance, and 34.53% of families reported using these services at some point in the twins' life, compared to census estimates of 13.56%. As family size factors into some forms of public assistance, it was expected that rates of public assistance would be higher in this sample compared to non-multiple families. Overall, this substantial level of usage points to considerable socioeconomic diversity in the current sample.

Big Five personality data from the TTP subprojects have been used in three previous publications (Mann, Briley, Tucker-Drob, & Harden, 2015; Mann et al., 2017, Tucker-Drob, Briley, Engelhardt, Mann, & Harden, 2016), but never in ways similar to how they were used in this study and never from all subprojects at the same time.

Zygosity

In order to determine zygosity, we used physical similarity ratings provided by parents, research assistants, and the twins themselves. Not all pairs had information from each of these sources (e.g., participants in a home-based survey subproject were not rated by research assistants or themselves, and only high school aged twins rated their physical similarity to their co-twin), but parents rated the physical similarity of all twins. We used all available information to conduct a latent class analysis to

classify each same-sex twin pair as MZ or DZ. This approach has been found to be more than 99% accurate when compared with genotyping (Heath et al., 2003). Opposite-sex twin pairs were classified as DZ. The sample included 614 MZ pairs, 592 same-sex DZ pairs, and 560 opposite-sex DZ pairs.

Measures

The primary measures were the adult (for parent-reported personality) and child (for self-reported personality) versions of the Big Five Inventory (BFI; John et al., 2008). All items were ipsatized for acquiescence (i.e., the tendency to yea- or nay-say) and extreme responding (i.e., the tendency to use extreme *vs.* central response options of Likert-type scales), respectively based on person-specific means and standard deviations of responses to pairs of items with opposite implications for personality (Soto, John, Gosling, and Potter, 2008): specifically, acquiescence was subtracted from each item response, which was then divided by extreme responding. Soto and colleagues (2008) demonstrated that these corrections of BFI items can be relevant in the current age range. In particular, they found that child self-reports showed substantially more variation in acquiescence at younger ages compared to older ages, and that correcting item responses for both acquiescence and extreme responding removed what was interpreted as an artefactual factor, allowing the expected Big Five factor structure to emerge. When left uncorrected for, response biases such as acquiescent and extreme responding could have influenced the variance of observed scale scores and thereby confounded our results. We found an age trend of decreasing acquiescence in self-reports [r = .27; 95% confidence intervals (*CI*) = .23, .30], whereas extreme responding did not correlate with age in self-reports (r = .02; 95% *CI* = -.02, .06; not reported by Soto et al., 2008); the correlations of children's age with acquiescence and extreme responding in parental reports were near-zero. Most importantly and replicating Soto and colleagues (2008), we found that the variance of acquiescence in self-reports decreased with age. Soto and colleagues (2008, Figure 1) found that the variance of acquiescence was twice as large at age 10 as it

was at age 20, whereas the variance of acquiescence was 1.87 times as large at age 8 as at age 18 in our data. However, we found somewhat lower overall variance in acquiescence (.09 at age 10, compared to .14 as reported by Soto et al., 2008). Variance of extreme responding differed less across the age range, increasing by 1.15 times from age 8 to 18.

Following the ipsatization and reverse scoring where appropriate, child- and parent-reported items were summed into scores for children's extraversion (child α = .80; parent α = .87), agreeableness (child α = .75; parent α = .83), conscientiousness (child α = .78; parent α = .87), neuroticism (child α = .72; parent α = .81), and openness to experience (child α = .70; parent α = .79). Prior to analysis, we also standardized all variables in reference to the full sample (M = 0, SD = 1). Therefore, estimates of variance less than 1 indicate lower than average variance, and estimates greater than 1 indicate higher than average variance.

Analyses and Results

Trait and twin correlations are reported in Table S1 (Online Supplement). The correlations between child- and parent-reported personality traits were moderate *(*r = .30 to .44), which is lower than is generally observed for adults but is typical for children (e.g., Laidra, Allik, Harro, Merenäkk, Harro, 2006). Correlations among the Big Five traits varied from 0 to .48, which is also a typical range (van der Linden, te Nijenhuis, & Bakker, 2010). The MZ twin correlations for the Big Five (r = .27 to . 51) were all more than twice the DZ twin correlations (r = -.15 to .12), indicating non-additive genetic variance. We fit biometric structural equation models in M*plus* 8 (Muthén &Muthén, 1998-2017) using a Huber-White sandwich estimator (Huber, 1967; White, 1980; Muthen & Satorra, 1995) to correct standard errors for clustering associated with multiple twin pairs from the same family and repeated measurements of some participants; this was implemented using the TYPE=COMPLEX and CLUSTER commands. Due to higher order multiples (i.e., triplets and quadruplets), some individuals

appeared more than once in the dataset. As a correction, we assigned them a weight equal to 1 divided by the number of times an individual observation appeared in the dataset. For example, triplet sets contributed three unique pairs to the dataset with each observation appearing in two pairs and these pairs were assigned a weight of .5. Twin pairs, because each twin appears in only one pair, were assigned a weight of 1. All models controlled for age, age^2, age^3, sex, and age × sex.

Main Effects Biometric Models

We began by comparing three alternative biometric models of personality: the *ACE*, *ADE*, and *AE* models. *A* refers to additive genetic effects[1] on the trait that serve to make individuals who are more genetically related (MZ twins) linearly more similar on the trait than those who are less genetically related (DZ twins). *C* refers to common, or shared, environmental effects that serve to make individuals raised together more similar regardless of genetic relatedness. *E* refers to nonshared environmental effects that are uncorrelated across members of the same twin pair, and also encompasses measurement error. *D* refers to dominant genetic effects that serve to make individuals who are more genetically related (MZ twins) disproportionately more similar on the trait than those who are less genetically related (DZ twins). While *D* is mathematically operationalized as dominance effects, it is statistically very difficult to distinguish from other sorts of non-additive effects (Neale and Maes, 2004), such as epistatic effects; *D* is therefore best conceptualized as a general non-additive genetic factor.

These respective models can be written as

$$Y_{t,p} = b_0 + \sum b_k \times x_k + a \times A_{t,p} + c \times C_p + e \times E_{t,p} \quad ,$$

$$Y_{t,p} = b_0 + \sum b_k \times x_k + a \times A_{t,p} + d \times D_{t,p} + e \times E_{t,p} \quad ,$$

[1] We use the word "effect" to refer to behavioral genetic variance components in order to be consistent with literature, but we do acknowledge that the conveys strong causal assertions that may not be tenable. These are variance components and not causes of observable variance in any direct sense.

and

$$Y_{t,p} = b_0 + \sum b_k \times x_k + a \times A_{t,p} + e \times E_{t,p} \quad ,$$

where *A, C, D*, and *E* are latent factors scaled to have unit variances. The subscript *t,* refers to terms that vary across twins within a pair (arbitrarily twin 1 or twin 2), and the subscript *p* refers to terms that vary across twin pairs.

In these models $r(A_{1,p},A_{2,p}) = 1.0$ and $r(D_{1,p},D_{2,p}) = 1.0$ for MZ twins; and $r(A_{1,p},A_{2,p}) = .50$ and $r(D_{1,p},D_{2,p}) = .25$ for DZ twins. C_p is a twin pair-level factor. The term $\sum b_k \times x_k$ represents the sum of the regression effects of the covariates x_1 through x_k, such that

$$\sum b_k \times x_k = b_1 \times age_{t,p} + b_2 \times age_{t,p}^2 + b_3 \times age_{t,p}^3 + b_4 \times sex_{t,p} + b_5 \times age_{t,p} \times sex_{t,p} \, .$$

As is common in behavioral genetic variance decomposition, such models rely on certain assumptions that are often violated in practice. The most important of those is that genetic and environmental processes are independent of each other, whereas one of our hypotheses rests on the idea that they are not. However, a systematic violation of this assumption also allows us to test this hypothesis, because (active and positive) gene-environment correlations (Plomin et al., 1977), or person-environment transactions, are expected to inflate the genetic variance estimates over time (Kandler & Zapko-Willmes, 2017; Purcell, 2002). Other ways in which this assumption may be violated is correlations between genetic and parental influences (e.g., passive and positive gene-environment correlations, which could lead to inflated estimates of shared environmental influences; Plomin et al., 1977) or gene-environment interactions, which could lead to either inflation of deflation of genetic influence estimates, depending on whether they occur with shared or non-shared environment, respectively. We cannot directly test any of these violations. Additionally, such models assume no assortative mating and that MZ twins are not treated systematically more similar than DZ

twins simply due to their zygosity status, an assumption that largely holds (Conley, Rauscher, Dawes, Magnusson, & Siegal, 2013).

Model Fit Comparisons and Parameter Estimates for Main Effects Biometric Models

Parameter estimates for the effects of the covariates on self- and parent-reported personality traits are reported in Table 1. There were no associations that were consistent across self- and parent-reports. For example, openness and extroversion significantly linearly increased with age according to self-reports but not parent-reports. Parameter estimates for the biometric portions of main effects *ADE*, *ACE*, and *AE* models are reported in Table 2, whereas fit statistics for these models are reported in Table 3. For model comparisons, we used the Akaike Information Criterion (AIC; lower values indicate comparatively better fit) and Bayesian Information Criterion (BIC; lower values indicate comparatively better fit), with the latter more strongly favoring parsimonious models. AIC comparisons favored the *ADE* model in all cases except parent-reported agreeableness, in which case the more parsimonious *AE* model was favored. BICs favoured *ADE* models in 60% of comparisons and simpler *AE* models in the remaining cases. Parameter estimates for *C* were, in all cases, estimated at 0, indicating no role for the shared environment on either self-reports or parent-reports of any of the BFI scales. In the *ADE* models, the *D* effects were always appreciable in magnitude, and, with the exception of parent-reported agreeableness and self-reported openness, *A* effects were estimated at 0. For parent-reported agreeableness, the *A* effect was nearly identical in magnitude to the *D* effect, but for self-reported openness, the *D* effect was much larger than the *A* estimate. In aggregate, there was consistent evidence from both model fit comparisons and parameter estimates that shared environmental effects were entirely lacking and that non-additive genetic effects were appreciable. The *ADE* model was therefore carried forward for the moderation analyses. Note, in the classical twin design assortative mating is

detected as shared environmental variance, which was estimated at zero across all personality traits for both self- and parent-reports, suggesting little role for assortative mating.

Parametric Age Moderation Models

A straightforward approach to modeling age-related trends in the variance of personality traits is to model the effect of the covariate-independent residual (U) as a linear function of age:

$$Y_{t,p} = b_0 + \sum b_k \times x_k + (1 + v' \times age_{t,p}) \times U_{t,p} \quad .$$

As the v' coefficient is a linear function of age, it reflects the extent to which variance in personality trait (Y) tends to increase or decrease across age. The term 1 is an identification constraint, linking the metric of U to that of Y for individuals at the zero point of the moderator (age). As age is centered at 8 years, this serves to scale U relative to the metric of Y at age 8 years. This identification constraint is directly analogous to the identification constraint that is used in a conventional factor model without moderation effects, in which one loading is fixed to 1.0 (so-called "unit loading identification") in order to identify the metric of the factor. Note that a mathematically equivalent approach would be to freely estimate the parameter, but constrain the variance of the factor to 1.0 (so-called "unit variance identification"). Such an approach is mathematically equivalent because it produces identical variance-covariance expectations, and in the case of the moderation model, it produces identical expectations for age trends in variance.

U represents the combined effects of genetic and environmental variance components. In the preferred *ADE* model, U is decomposed as:

$$U_{t,p} = a \times A_{t,p} + d \times D_{t,p} + e \times E_{t,p} \quad .$$

Combining the above two equations yields:

$$Y_{t,p} = b_0 + \sum b_k \times x_k + (a + a \times v' \times age_{t,p}) \times A_{t,p} + (d + d \times v' \times age_{t,p}) \times D_{t,p} + (e + e \times v' \times age_{t,p}) \times E_{t,p} \quad .$$

Under this model, the effects of age on the *A*, *D*, and *E* effects are proportional to the main effects of *A*, *D*, and *E*, such that the relative contributions of *A*, *D*, and *E* remain invariant over age, even as total variance increases. This approach corresponds to the "phenotypic null hypothesis" (Turkheimer et al., 2014) that effects on personality are best conceptualized as occurring directly on the observable trait, rather than on its biometric components. Again, we emphasize that this hypothesis is premised on the idea that external (e.g., immediate social or general evolutionary) pressures act on observable characteristics, not on unobservable genetic and environmental contributions to these characteristics. We call this the phenotypic variance moderation model. Such a model is a constrained instantiation of a more general biometric age moderation model that allows for individual moderation terms for *A*, *D*, and *E*. Such a "full" age moderation model can be written as:

$$Y_{t,p} = b_0 + \sum b_k \times x_k + (a + a' \times age_{t,p}) \times A_{t,p} + (d + d' \times age_{t,p}) \times D_{t,p} + (e + e' \times age_{t,p}) \times E_{t,p} \quad ,$$

where *a*, *d*, and *e* represent the main effects of *A*, *D*, and *E*, and a', d', and e' represent age moderation of the *A*, *D*, and *E* effects.

Finally, the age moderation model can be simplified to allow for age moderation of only one biometric component at a time:

$$Y_{t,p} = b_0 + \sum b_k \times x_k + (a + a' \times age_{t,p}) \times A_{t,p} + d \times D_{t,p} + e \times E_{t,p} \quad ,$$

$$Y_{t,p} = b_0 + \sum b_k \times x_k + a \times A_{t,p} + (d + d' \times age_{t,p}) \times D_{t,p} + e \times E_{t,p} \quad ,$$

and

$$Y_{t,p} = b_0 + \sum b_k \times x_k + a \times A_{t,p} + d \times D_{t,p} + (e + e' \times age_{t,p}) \times E_{t,p} \quad .$$

We can compare the fit indices of these simpler models to one another and to the full model as a means of isolating the key biometric component that drives age-related trends in the magnitude of observed variance.

Model Fit Comparisons and Parameter Estimates for Parametric Age Moderation Models

We began by inspecting trends obtained from the phenotypic variance moderation model. Parameter estimates from this model are reported in Table 4, and model-implied age trends are displayed in Figure 2. All BFI scales except self-reported openness displayed appreciable age-related increases in observed variance, with the magnitude of increases being more uniform among parent-report measures compared to self-report measures. For child-report measures from age 8 to 18, variance increased by 74% for extraversion, 17% for agreeableness, 30% for conscientiousness, and 28% for neuroticism; variance in openness decreased by 2%. Similarly, for parent-report measures from age 8 to 18, variance increased by 23% for extraversion, 32% for agreeableness, 28% for conscientiousness, 21% for neuroticism, and 46% for openness.

Next, we estimated the full age- moderation models that decomposed variance differences across age levels into additive (*A*) and non-additive (*D*) genetic and non-shared (*E*) environmental components. Parameters from these models are reported in Table 5, and model-implied age trends are displayed in Figure 3 for self-reports and Figure 4 for parent reports; both figures include a panel representing the average trend across traits. The average trends for both self-reports and parent-reports were primarily characterized by increasing non-additive genetic variance with age, although this trend was less pronounced for parent-reports than for self-reports. The trends for the individual traits were generally consistent with the overall trend of increasing non-additive genetic variance with age.

Visual inspection of the results from the full moderation model indicated that increases in non-additive genetic variance primarily drove the increases in observed variance in personality with age;

unlike other age moderation parameters, those for *D* (d′; Table 5) were always positive, although individually they were often not statistically significant (for six out of ten parameters, the magnitude of the parameter was less than twice of its standard error). In order to test whether this pattern also held using a model comparison approach, we fit a series of simplified models in which moderation was only allowed for either *A*, *D*, or *E*. We compared the fits of these models to each other, the full moderation model, the phenotypic variance moderation model, and models that did not allow for moderation (Table 6). In four cases (self-report conscientiousness, extraversion, and agreeableness and parent-reported agreeableness), the lowest AIC values were obtained for the models that allowed for *D* moderation only. In three cases (parent-reported openness, conscientiousness, and extraversion), the lowest AIC values were obtained for the phenotypic variance moderation model; in each of these cases, the *D* moderation only model had the second lowest value. Self-reported openness displayed no variance moderation as indicated by either AIC or BIC. Self-reported neuroticism displayed moderation across all *ADE* parameters (non-proportionally), as indicated by AIC. However, by BIC, the *D* moderation model was preferred, and this model also had the second lowest AIC. Finally, parent-reported neuroticism had the lowest AIC and BIC for the *A* moderation model. In summary, age moderation most commonly occurred in relation to the *D* pathway (8 times; when *ADE*, *D* only or phenotypic variance moderation models were preferred according to AIC), followed by the *A* pathway (5 times; when *ADE*, *A* only or phenotypic variance moderation models were preferred according to AIC), and the *E* pathway was only moderated when all pathways were included, as in the phenotypic variance moderation model or the *ADE* moderation model (4 times). In aggregate, thus, there was most evidence for (non-additive) genetic variance increasing with age.

Table S2 and Figures S1-2 (Online Supplement) present results of a sensitivity analysis whereby the non-additive genetic factor was specified to represent the possibility of epistasis rather than dominance. Fitting the epistasis model (which is less common in behavioral genetics) was justified by

the near-zero DZ twin correlations that we observed. Overall, this alternative specification yielded results consistent with those reported above, indicating that genetic variance, particularly non-additive genetic variance, increased with age.

Non-parametric Age Moderation Models

Results of parametric age moderation models provided relatively consistent evidence that the total between-person variance in both self and parent reports of the Big Five personality increased between ages 8 and 18, and that these age-related increases were predominantly driven by increases in (mostly non-additive) genetic variance. The parametric approach was parsimonious in having a single parameter representing age-related increases in a particular variance component, which was particularly useful for avoiding over-fitting. However, non-linear age moderation effects might also be present in the data, as Mõttus and colleagues (2017) reported non-linear increases in observed variance. We therefore went on to employ on-parametric analyses could be used to gauge whether the shape of the age moderation function might be more complex.

We applied Local Structural Equation Modeling (LOSEM; Briley, Harden, Bates, & Tucker-Drob, 2015). LOSEM accomplishes a similar function as the parametric moderation models described above but, rather than estimating a single interaction parameter for each variance component representing age-related differences (i.e., the a', d', and e' parameters), LOSEM produces local estimates of the focal variance components (i.e., the a, d, and e parameters) continuously across a moderator (here, age). LOSEM is similar to other kernel regression techniques, but is specifically adapted for a structural equation modeling context. For example, LOESS plots (LOcal regrESSion) estimate non-parametric regression lines through a scatterplot based on locally weighted regression (Cleveland and Devlin, 1988). LOSEM applies the same logic to estimate structural equation models

by locally weighting data and continuously shifting target levels of a moderator. The earlier full moderation equation, given above, can be rewritten as:

$$Y_{t,p} = b_0 + \sum b_k \times x_k + a_{[age8 \ldots age18]} \times A_{t,p} + d_{[age8 \ldots age18]} \times D_{t,p} + e_{[age8 \ldots age18]} \times E_{t,p} \quad .$$

The new subscript, [age8 … age18], implies that we estimated locally weighted parameters for each variance component starting at age 8 and shifting continuously up to age 18. We followed the methodological recommendations found in Briley and colleagues (2015) for carrying out the analyses. Importantly, LOSEM is more prone to over-fitting than parametric models (i.e., it may give impressions of complex associations patterns that do not exist in the population) and encourage readers to avoid interpreting small deviations in the trends, particularly at the tails of the age distribution where estimates are based on fewer twin pairs.

As can be seen in Figure 5, age-related increases in variance were consistent with results based on the parametric approach. In general, each Big Five trait increased in variance across both self-reports and parent-reports, except for self-reported openness. For self-report measures from age 8 to age 18, variance increased by 58% for extraversion, 13% for agreeableness, 37% for conscientiousness, 10% for neuroticism, and 1% for openness. For parent-report measures from age 8 to 18, variance increased by 13% for extraversion, 19% for agreeableness, 17% for conscientiousness, 23% for neuroticism, and 34% for openness.

However, the non-parametric approach identified potential nonlinearities in the age trends. For both self- and parent-reported traits, age-related differences in variance were mostly flat until roughly age 11, except for the majority of the entire increase in variance in conscientiousness happened prior to age 11 (73% and 77% of the total increase, respectively for self- and parent-reports). Interestingly, increases in variance for parent-reports were almost all concentrated between ages 12 and 15, with all traits except neuroticism displaying a plateau in variance during late adolescence. These results were

somewhat similar for child-report. As noted above, increases in variance for conscientiousness were concentrated at younger ages, and agreeableness followed a similar plateau. Neuroticism followed a continued trajectory of increasing variance, similar to parent-report. The primary difference for late adolescence was that child-reported extraversion showed continued and strong increases in variance, while parent-reported extraversion plateaued and then declined in variance.

Next, we used LOSEM to decompose variance in each trait across age. The distinct estimates of additive and non-additive genetic variance using LOSEM exhibited "tradeoffs," sometimes fluctuating in a wave-like pattern, indicating that the *A* and *D* components were difficult to distinguish from one another, with slight shifts in weights associated with the target age producing dramatic shifts between *A* and *D* variance. Therefore, to increase clarity, we combined estimates of additive and non-additive genetic variance for the LOSEM plots. These results are plotted in Figure 6 for self report and Figure 7 for parent report.

For extraversion, neuroticism, and conscientiousness, the LOSEM results were generally similar to the parametric models in that increases in variance were primarily driven by increasing genetic variance. Further, the average genetic variance increase trend for these traits was nearly linear, apart from a slight plateau for older adolescents. However, both self- and parent-reported agreeableness displayed trends that had not been identified in the parametric models: genetic variance increased substantially from ages 8 to 14 and then decreased, while environmental variance decreased from age 8 to 12 and then began to increase subtly. This result implies that the relative stall in age-related observed variance differences at young ages may hide shifts; increases in genetic variance could be offset by decreases in environmental variance. Then, large increases in variance in early to middle adolescence were magnified by stalling declines in environmental variance and continued increases in genetic variance. This sort of inverted-U shape for genetic variance was difficult to detect with the standard

parameterization of the moderation model. For openness, a similar albeit somewhat less pronounced pattern of inverted-U shape for genetic variance appeared in self-reports, although the best-fitting parametric model had indicated no variance moderation. For parent-reported openness, the best fitting parametric model had indicated that variance increased uniformly across genetic and environmental sources of variance, but the non-parametric results identified differences in the rate of variance increases with age. Environmental variance increased slowly and linearly across the entire age range, whereas genetic variance had a punctuated increase in variance between ages 12 and 15, accounting for essentially the entire increase in genetic variance. The average trends across the Big Five traits (bottom-right panels of Figures 6 and 7) were rather similar for self- and parent-reports.

In aggregate, the LOSEM trends were similar to the parametric results, as most clearly displayed in the average trends depicted in Figures 3, 4, 6 and 7. However, they provided further precision, indicating that increases in variance, particularly for parent reports, tended to be concentrated in ages up to 15 years. As a cautionary note, we emphasize the modest magnitude of these nonlinear trends, as well as the potential for imprecision at the either end of the age distribution due to a relatively small sample size for such analyses.

Discussion

The results of the current study indicated that the magnitude of individual differences in youth personality traits tends to increase between ages about 8 and 18 years, with the trend being most consistent until mid-adolescence (about 15 years of age). At the level of observed personality variance, this was a replication of the results by Mõttus and colleagues (2017), underscoring the robustness of this newly-discovered pattern in personality development. We also found that the results generally held regardless of whether youth's self-reports or parent-reports of personality traits were used. Expanding on the previous research, the current results suggest that the observed increases in variance may be

largely driven by influences that genetically similar individuals have in common but that are not stemming from shared environmental experiences. In other words, genetically influenced differences in youth personality tend to become more pronounced with increasing age. In contrast, the magnitude of environmentally influenced personality variance did not appear to systematically change with age.

These results can be informative regarding the mechanisms that contribute to personality development (Tucker-Drob & Briley, in press). Increasing genetic variance in personality with age is consistent with the hypothesis that genetically influenced traits are amplified over time, either because the underlying traits *per se* require time to reach their full extents (akin to height); because the cognitive, affective, or motivational mechanisms through which the traits become observable develop gradually; or both. This hypothesis of the developmental amplification of genetic influences is consistent with the FFT (McCrae & Costa, 2008), but it should be considered in tandem with another FFT-based prediction that environmental (including stochastic) influences diminish with age—as "nature" gradually shines through all other sources of influence. We did not find evidence for decreasing environmental variance (except for agreeableness and self-reported openness at younger ages in the non-parametric results). Thus, our results are only partly consistent with predictions based on the intrinsic maturation hypothesis of the FFT.

We also considered the possibility that increases in personality variance reflect accumulating environmental influences on personality. Children may experiment with new roles and experience a variety of situational influences as they develop, and these experimentations may often happen randomly (Briley & Tucker-Drob, 2017). We drew a parallel with random walk, in which children develop in one direction in the space of personality traits, then try something different and develop towards another direction, and so forth. This hypothesis would have been consistent with non-human behavioral studies showing that even genetically identical organisms placed in identical environments

develop behavioral differences (e.g., Freund et al., 2013; Bierbach et al., 2017) and with the previous meta-analytic finding that the heritability of personality traits tends to decrease through childhood (Briley & Tucker-Drob, 2014; but see also Kandler & Papendick, 2017). However, we did not observe increases in environmental variance in the current study, which (unlike the meta-analysis) employed a single personality measure and constant informant perspectives across the entire age range under investigation. Therefore, the present findings did not support the random walk-like hypothesis.

Another possibility that we considered combines genetic and environmental explanations for the increasing magnitude of personality differences between children. Specifically, genetic influences may be amplified not only because youth traits gradually mature toward their intrinsically determined levels, but also because individuals evoke, select, and create environmental experiences that match and reinforce their pre-existing traits (i.e., the corresponsive principle). That is, genetic amplification may be mediated by individuals transacting with trait-matched environments (Tucker-Drob, in press). Such matching could conceivably occur in at least two ways, typically referred to as evocative and active gene-environment correlation (Plomin et al., 1977). Evocative gene-environment correlation occurs when other individuals adjust their behavior toward a person on the basis of their observable characteristics (e.g., teachers may give extra attention to conscientious students, which reinforces the students' effort). Active gene-environment correlation occurs when individuals create an environment that matches their preferences (e.g., conscientious students may seek out feedback from teachers, which then reinforces their effort). To the extent that these forms of gene-environment correlation apply, it is the genetic component of variance that is expected to increase in magnitude over time, although this increase would not imply that genetic influences *per se* become stronger over time, but that individuals and their environments become increasingly entangled, and thereby *all* influences become increasingly aligned with genetic variance. According to this hypothesis, there is no reason to expect either increases or decreases in the environmental components of trait variance, which is exactly what we

observed. It would be interesting to tease apart the extent to which evocative compared to active gene-environment correlation drives this trend, but this was impossible based on the current data. Future work that explicitly measures environmental evocation and creation across time could answer this question. We note that the intrinsic maturation and corresponsive principle-based (transactional) accounts of personality development are not mutually exclusive. However, lack of evidence for decreasing environmental variance would suggest that intrinsic maturation *per se* is not the dominant developmental mechanism contributing to the children's tendency to grow less alike.

Non-additivity of genetic influences may offer a further clue for how personality develops

Another aspect of our findings that might be informative for theories of personality development is that primarily non-additive components of genetic variance increased with age. In general, the appearance of non-additive genetic variance is consistent with several past studies of personality (Saudino, 1997), including studies of twins reared apart (Tellegan et al., 1998), family studies (Vukasovic & Bratko, 2015), and extended family studies (Boomsma et al., 2017). Such results are also consistent with large-scale molecular genetic studies of unrelated individuals, indicating that additive contributions to personality variation by common genetic variants are lower than typically found in twin and adoption studies (Lo et al., 2017; Penke & Jokela, 2016; Vukasovic & Bratko, 2015). Expanding on this evidence, we observed developmental increases in the magnitude of non-additive variance. At the moment, we do not have a definitive explanation for why intrinsic maturation of a trait should result in amplification of non-additive genetic variation rather than additive genetic variation. But we do have two candidate explanations based on how dynamic transactional processes operate within individuals and/or between individuals and their environments.

What appears as non-additive genetic variance in behavior genetic models reflects the tendency for observable similarity between individuals to increase disproportionally with their genetic similarity.

Put differently, non-additive genetic variance implies that even relatively small genetic differences between individuals result in disproportionally large observable differences. One way that this could occur is that the observable traits are influenced by, or consist of, several more specific components that interactively influence each other (Cramer et al., 2012, Mõttus & Allerhand, 2017). Even if these causally connected components themselves and/or the links between them are under strict additive genetic influence (Cramer et al., 2011), the products of these causal connections may result in non-additive variance because they depend on combinations of genetic variants (Mõttus & Allerhand, 2017)[2]. Thus, if personality is influenced by, or indeed is, a combination of subcomponents that influence each other over prolonged periods of development, we might expect increases in non-additive genetic effects on personality.

As another possibility, individuals may transact with their environments in somewhat idiosyncratic ways, seeking environments that match some of their personality traits but not necessarily the others. Specifically, it is possible that not all traits are equally important for individuals, with some traits being more central than others (Costantini et al., 2015), and it may be particularly important for individuals to find or create environments that match their most central traits. For example, someone high in excitement-seeking (a central trait for *this* person) may end up in the company of individuals who match their high level of this trait (e.g., due to shared activities or preference for like-minded people), regardless of how self-conscious, orderly, irritable, or politically liberal these individuals are. Alternatively, an individual with excitement-seeking as a central trait may be prone to experience non-social environments matching this particular trait (e.g., a diverse range of risky activities) regardless of his or her other trait levels. More genetically related individuals (e.g., MZ twins compared to DZ twins)

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[2] For example, a component (x) can indirectly contribute another component (y) via a third component (z), but the realization of this indirect contribution depends on the genetic influences on all connections between the components (between x and z as well as between z and y). There may be numerous indirect associations between personality components, raising the possibility that a substantial proportion of genetic variance is non-additive. The results of these interactions may accumulate over time, appearing as an increase in non-additive genetic influences.

may be disproportionally more likely to share their central traits than do less genetically related individuals, because even otherwise relatively similar individuals (e.g., DZ twins) may differ in which particular traits are most central for them. If the process of seeking out and evoking personality-relevant experiences over time is particularly influenced by the central traits, this may lead to increasing dissimilarity of less genetically related individuals and to maintained, or even amplified, similarity of more related individuals. Therefore, such processes may also lead to the emergence and amplification of non-additive genetic effects with age.

We note, however, that lack of increasing observed variance would not have ruled out the pertinence of such processes, because personality characteristics may compete against each other such that increases in some of them (e.g., as a result of person-environment transactions) may entail decreases in others, so that, on average, people do not gravitate towards extreme trait levels. It is possible, for example, that such within-individual competition between characteristics may become more prevalent over time (e.g., due to increasing social constraints), contributing to the observed plateauing of the magnitude of individual differences.

Plateauing of genetic variance

In the non-parametric analyses, increases in genetic variance were more robust from about age eight until mid-adolescence. By and large, this observation is consistent with previously reported increases in observed variance from age three to early adolescence (Mõttus et al., 2017). What could explain such a curvilinear trend, in addition to the possibility of within-individual competition between causally connected characteristics? One explanation is that person-environment transactions amplify genetic variance to an asymptotic level as individuals approach to or reach an equilibrium state with respect to their personality trait levels and environment (Mõttus et al., 2017). Similarly, it is plausible that the further an individual's traits are pulled from their genetically influenced baseline as a result of

transactions with environments, the harder it may become to pull them yet further. The same would happen if extremes of any trait tended to be (socially) less adaptive than trait levels closer to population means. This can be conceptualized as a form of gene-environment interaction: An environment that has been sought out or created to facilitate the manifestation of pre-existing genetic dispositions can only do this up to a certain level, after which the genetic dispositions become less sensitive to further environmentally driven change. Alternatively, to the extent that increases in variance reflect intrinsic maturation, it could be that the genetically influenced trait levels, or their manifestations, have typically developed to their full degrees by mid-adolescence.

It is also possible that the plateauing of variance may be explained by processes that suppress individuation catching up with processes that contribute to people "walking their own way." For example biological changes during puberty may lead to increasing sensitivity to reward (e.g., Steinberg, 2010; Harden & Mann, 2015), and social shifts may give adolescents increasing opportunities to pursue rewards. What specifically is rewarding for any given person likely depends, in part, on genetically influenced characteristics, and purportedly increasing sensitivity to and perusal of rewarding activities may contribute to increasing (genetic) personality variance. However, at later stages of development, more slowly developing self-regulation (e.g., De Luca, Wood, Anderson et al., 2003; Harden & Tucker-Drob, 2011) or increasing socialization pressures may cap further individuation resulting from reward sensitivity, or reward sensitivity itself may plateau. This possibility is consistent with the dual-systems perspective on socioemotional and cognitive development (e.g., Shulman, Smith, Silva et al., 2016).

Finally, it is also possible that trait measures only capture a limited range of how any trait can become manifest. If so, the observed plateauing of variance may simply reflect methodological artefacts.

Considering non-dynamic explanations and the need for longitudinal data

In the preceding sections, we largely discussed dynamic processes of personality development that unfold within people or between people and their experiences. However, our results are also consistent with a more "static" account of development whereby genetic variance in personality simply emerges during certain periods. Put differently, there may be stable sources of genetic variance that persist across time (e.g., some of the genetic variance at age eight will be the same at age 18), but all of the increases in genetic variance are entirely unrelated to past genetic influences and result from novel genetic factors "turning on." It is notable that the identified age trends in variance for parent-reported personality tend to track with times of dramatic shifts in pubertal development. Variance increases in child-reported personality, while less clearly concentrated during a specific transition, may reflect children's more nuanced perception of their own physiological and social changes that co-occur with puberty. Dramatic changes in psychological, social, and physiological process occur during puberty (e.g., Del Giudice, 2014; Harden, 2014; Mendle, 2014). Our results are therefore compatible with such a transition activating previously inactive genetic variants. If these potentially hormone-relevant variants are dependent on one another or other psychological/social characteristics, novel non-additive genetic variance would be produced. However, we note that similar increases in observed variance were documented throughout childhood by Mõttus and colleagues (2017)–much earlier than in puberty.

Longitudinal data will be able to discriminate between these two patterns of results. Such models can identify whether increases in genetic variance are shared with earlier time points (i.e., amplification) or are unique to later time points (i.e., innovation). Briley and Tucker-Drob (2013) demonstrated that increasing genetic variance in cognitive ability primarily results from amplification processes, consistent with dynamic models of person-environment transactions. How similar is personality in this respect? The best piece of information currently available in this respect is the

genetic correlation between measures across time (Briley & Tucker-Drob, 2017). Genetic stability for cognitive ability is very high at early ages, reaching a nearly perfect correlation by age 10. This level of stability implies that any increases in variance must occur through amplification processes, or else stability would be lower. On the other hand, the genetic stability of personality is lower than that of intelligence and increases more slowly across age (Briley & Tucker-Drob, 2017). During the developmental period currently under investigation, one would expect genetic stability of approximately .65 to .85 (Briley & Tucker-Drob, 2014; Kandler & Papendick, 2017). Although this level of stability is certainly high, it leaves open the possibility of variance increases resulting from novel sources of genetic variance. Of course, stability could also be less than perfect because earlier genetic influences decay across time, allowing for any increases in genetic variance to be driven by more stable genetic pathways.

As emphasized throughout this article, descriptive information concerning development can constrain theories and point toward potential mechanisms of personality development. To better understand which theory or model is most plausible, longitudinal, genetically informative samples are necessary. Given the fairly early and rapid shifts in variance (Mõttus et al., 2017), it would be particularly interesting if such data were collected with relatively short re-test intervals and with information concerning social experiences and hormone production. As personality can change rapidly under some circumstances (e.g., Roberts et al., 2017), it is possible that the smooth trends identified in the current sample actually reflect punctuated change among some individuals, possibly in response to a universal transition (e.g., puberty). If the timing of this event differs somewhat across individuals, potentially for genetic reasons (e.g., Moore, Harden, & Mendle, 2014), this differential experience and the concomitant social repercussions could explain our results. Thus, genetically informative, longitudinal studies with intensive sampling across this period of accelerated change will be necessary to document personality formation and maturation.

Does heritability increase or decrease over childhood and adolescence?

A previous meta-analysis reported that heritability estimates of personality traits tend to decrease across childhood and adolescence (Briley & Tucker-Drob, 2014), whereas the present findings suggest the opposite, as does the more recent meta-analysis by Kandler and Papendick (2017). These discrepancies could be because the heritability estimates for different age groups were often based on different personality instruments in the meta-analyses. Moreover, in Briley and Tucker-Drob (2014), effect sizes pertaining to earlier ages were mostly based on parental ratings, whereas studies tended to rely on self-ratings in middle childhood and beyond. Different tests or sources of ratings could be differentially sensitive to genetic and environmental influences, or parent-ratings may be more reliable than adolescents' self-ratings, yielding artificially lower heritability estimates for older age groups. Indeed, when effect sizes based on parent-reports were excluded from the meta-analysis age trends in heritability became non-significant (Briley & Tucker-Drob, 2014). And the meta-analysis that reported increasing heritability estimates with age (Kandler & Papendick, 2017) relied mostly on self-reported personality traits for the focal age groups, although the typical heritability estimates were not lower than those based on parent-reports. Another reason for the discrepant findings across the two meta-analyses might may have been differences in the sampled constructs: Briley and Tucker-Drob (2014) sampled a wide range of constructs, whereas Kandler and Papendick (2017) only focused on the Big Five traits. The present research relied on a single personality instrument (and thereby the same constructs) and constant rating perspectives throughout the studied period, as could future studies on the topic. Although further research is required, the present findings may be more in line with those of Kandler and Papendick (2017).

Qualitative changes

The study is based on the assumption that personality traits are qualitatively similar throughout the considered age range. By and large, this assumption is justified, as the adult-like Big Five personality traits can be used to describe differences even in very young children (Soto & John, 2014; Soto, 2016). However, there is also some evidence for personality traits becoming increasingly differentiated as children develop (Rothbart, Ahadi, Evans, 2000; Tackett, Slobodskaya et al., 2012). Future studies could assess whether intra-individual differentiation of personality, to the extent that it exists, results from genetic or environmental influences, or both.

Relatedly, we also note that personality measures generally lack appropriate levels of measurement invariance across age groups (Mõttus et al., 2015; Soto, 2016). We did not specifically test for measurement invariance in the present study, but the same was almost guaranteed to apply. This could mean that age trends in personality scores, in means or variances, are specific to particular items of the scales rather than uniform across the items of the same constructs. However, given the overall consistency of the findings across the constructs, this would have been unlikely to alter our overall conclusions regarding the increasing magnitude of genetically influenced individual differences. In previous research, the trends for increasing observable variance have generally also applied for individual test items (Mõttus et al., 2017).

Strengths and limitations

One of the strengths of the current study is the use of a large and diverse sample of twins, which speaks to the relative generalizability of the findings, at least within a Northern American context. Furthermore, the appearance of the main findings in both self- and parent-ratings, and across traits, bolsters the robustness of the findings. Each single rating perspective is subject to substantial method effects (McCrae, 2015), so findings based on any one of them alone could be misleading, whereas the convergence of the findings provides non-trivial evidence for quasi-replication. Also, the use of the

same personality measure throughout the addressed developmental period reduced the likelihood of method-specific biases. Additionally, it is important to keep in mind that we controlled for acquiescence bias, which is associated with age, and could therefore have confounded the results.

A major limitation of the study is its cross-sectional design, which prevents us from making direct inferences about within-person changes over time. Strictly speaking, we studied age differences in the magnitude of genetic and environmental variance rather than developmental changes *per se*. Likewise, the study only relied on a relatively brief Big Five personality instrument and did not address developmental patterns in more specific personality traits such as facets or nuances (Mõttus et al., 2017). Finally, due to very few twins being younger than 8 years, we could not test whether variance differences across childhood observed by Mõttus and colleagues (2017) reflected environmental or genetic influences, or both.

Conclusions

We found evidence that children became increasingly less alike in personality across age, both based on their self-descriptions and personality ratings provided by their parents. Specifically, genetically influenced personality variance increased in magnitude, whereas environmentally influenced personality variance neither increased nor decreased. These observations are consistent with the possibility that pre-existing genetic influences become amplified over time as people evoke and select environments aligned with these influences. That the increases in variance appeared primarily to result from genetic influences that operate in a non-additive manner could reflect developmental unfolding or accumulation of trait-trait interactions, a form of person-environment transactions whereby genetically similar people are disproportionally likely to experience similar environments, or all of them. Another possibility could be direct dominant genetic influences that become activated at specific points in development. These descriptive findings are likely to have important implications for

theories of personality development, although further longitudinal studies are required to tease apart the possible explanations outlined in this study.

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Table 1. *Unstandardized parameter estimates for effects of covariates on self- and parent-reported BFI scales.*

	Age	Age^2	Age^3	Sex	Age × Sex
Self-Reports					
Openness	0.143 (0.061)	-0.037 (0.012)	0.002 (0.001)	-0.154 (0.075)	0.015 (0.012)
Conscientiousness	0.039 (0.053)	-0.033 (0.011)	0.002 (0.001)	-0.113 (0.071)	0.006 (0.011)
Extraversion	**0.223 (0.063)**	**-0.051 (0.013)**	0.003 (0.001)	-0.094 (0.070)	0.020 (0.012)
Agreeableness	0.085 (0.058)	-0.029 (0.012)	0.002 (0.001)	-0.133 (0.070)	0.013 (0.012)
Neuroticism	-0.053 (0.061)	0.005 (0.012)	0 (0.001)	-0.030 (0.072)	**-0.048 (0.012)**
Parent-Reports					
Openness	-0.014 (0.031)	-0.010 (0.008)	0.001 (0.001)	-0.075 (0.061)	-0.009 (0.011)
Conscientiousness	0.022 (0.027)	-0.005 (0.007)	0 (0)	-0.060 (0.064)	-0.024 (0.011)
Extraversion	-0.016 (0.019)	-0.013 (0.005)	0.001 (0)	-0.008 (0.058)	0.005 (0.010)
Agreeableness	0.010 (0.026)	-0.008 (0.007)	0 (0)	-0.063 (0.054)	0.026 (0.010)
Neuroticism	-0.020 (0.032)	0.003 (0.008)	0 (0.001)	-0.030 (0.062)	-0.011 (0.012)

NOTE: Standard errors provided in parentheses. Bold-face indicates that the parameter was significant at $p < .001$.

Table 2. *Unstandardized parameter estimates for biometric portions of main effects ADE, ACE, and AE models.*

	ADE Model			ACE Model			AE Model	
	a	*d*	*e*	*a*	*c*	*e*	*a*	*e*
Self-Reports								
Openness	0.117 (0.651)	**0.564 (0.148)**	**0.795 (0.025)**	**0.543 (0.038)**	0 (0)	**0.819 (0.024)**	**0.543 (0.038)**	**0.819 (0.024)**
Conscientiousness	0 (0)	**0.559 (0.044)**	**0.804 (0.029)**	**0.500 (0.045)**	0 (0)	**0.842 (0.026)**	**0.500 (0.045)**	**0.842 (0.026)**
Extraversion	0 (0)	**0.605 (0.038)**	**0.777 (0.028)**	**0.536 (0.041)**	0 (0)	**0.827 (0.026)**	**0.536 (0.041)**	**0.827 (0.026)**
Agreeableness	0 (0)	**0.557 (0.039)**	**0.816 (0.027)**	**0.509 (0.039)**	0 (0)	**0.846 (0.026)**	**0.509 (0.039)**	**0.846 (0.026)**
Neuroticism	0 (0)	**0.517 (0.043)**	**0.834 (0.028)**	**0.454 (0.045)**	0 (0)	**0.870 (0.027)**	**0.454 (0.045)**	**0.870 (0.027)**
Parent-Reports								
Openness	0 (0)	**0.701 (0.036)**	**0.693 (0.031)**	**0.658 (0.040)**	0 (0)	**0.737 (0.031)**	**0.658 (0.04)**	**0.737 (0.031)**
Conscientiousness	0 (0)	**0.509 (0.056)**	**0.849 (0.030)**	**0.426 (0.061)**	0 (0)	**0.893 (0.027)**	**0.426 (0.061)**	**0.893 (0.027)**
Extraversion	0 (0)	**0.491 (0.065)**	**0.853 (0.036)**	**0.339 (0.081)**	0 (0)	**0.923 (0.031)**	**0.339 (0.081)**	**0.923 (0.031)**
Agreeableness	0.385 (0.215)	0.452 (0.206)	**0.808 (0.034)**	**0.573 (0.042)**	0 (0)	**0.824 (0.029)**	**0.573 (0.042)**	**0.824 (0.029)**
Neuroticism	0 (0)	**0.602 (0.050)**	**0.803 (0.031)**	**0.552 (0.056)**	0 (0)	**0.837 (0.029)**	**0.552 (0.056)**	**0.837 (0.029)**

NOTE: Standard errors provided in parentheses. *a* = additive genetic effect; *c* = shared environmental effect; *d* = non-additive genetic effect; *e* = non-shared environmental effect. Bold-face indicates that the parameter was significant at $p < .001$.

Table 3. *Model fit indices for main effects biometric models.*

	Chi Square	*df*	*p*	Scaling Factor	RMSEA	CFI	TLI	AIC	BIC
Self-Reports									
Openness									
ADE	28.630	36	0.804	1.150	.000	1.00	1.05	**7648.502**	7695.603
ACE	31.608	36	0.678	1.185	.000	1.00	1.03	7653.010	7700.111
AE	32.485	37	0.681	1.153	.000	1.00	1.03	7651.010	**7692.878**
Conscientiousness									
ADE	54.513	36	0.025	1.076	.033	0.90	0.90	**7639.576**	**7686.677**
ACE	65.199	36	0.002	1.077	.042	0.84	0.85	7651.107	7698.208
AE	67.010	37	0.002	1.048	.042	0.83	0.85	7649.107	7690.974
Extraversion									
ADE	50.280	36	0.057	1.137	.029	0.89	0.90	**7651.505**	**7698.606**
ACE	66.584	36	0.001	1.138	.043	0.77	0.79	7670.083	7717.184
AE	68.434	37	0.001	1.107	.043	0.77	0.79	7668.083	7709.951
Agreeableness									
ADE	31.153	36	0.698	1.119	.000	1.00	1.06	**7689.824**	7736.925
ACE	37.413	36	0.404	1.120	.009	0.98	0.98	7696.865	7743.966
AE	38.452	37	0.404	1.090	.009	0.98	0.98	7694.865	**7736.733**
Neuroticism									
ADE	83.658	36	< .001	1.178	.054	0.77	0.79	**7672.817**	**7719.918**
ACE	91.047	36	< .001	1.174	.058	0.74	0.76	7681.149	7728.250
AE	93.576	37	< .001	1.143	.058	0.73	0.76	7679.149	7721.017
Parent-Reports									
Openness									
ADE	37.442	36	0.403	1.134	.010	0.99	0.99	**6799.473**	**6845.709**
ACE	49.805	36	0.063	1.140	.030	0.93	0.94	6813.801	6860.037
AE	51.189	37	0.060	1.109	.030	0.93	0.94	6811.801	6852.899
Conscientiousness									
ADE	56.443	36	0.016	1.048	.037	0.75	0.77	**6920.317**	**6966.553**
ACE	68.013	36	0.001	1.046	.046	0.60	0.64	6932.320	6978.556
AE	69.903	37	0.001	1.018	.046	0.59	0.64	6930.320	6971.418
Extraversion									
ADE	69.342	36	0.001	1.092	.047	0.71	0.73	**6895.274**	**6941.509**
ACE	83.930	36	< .001	1.086	.056	0.58	0.61	6910.676	6956.912
AE	86.261	37	< .001	1.057	.056	0.57	0.61	6908.676	6949.775
Agreeableness									
ADE	41.062	36	0.258	1.097	.018	0.95	0.95	6952.233	6998.469
ACE	41.022	36	0.260	1.135	.018	0.95	0.95	6953.762	6999.998
AE	42.161	37	0.258	1.105	.018	0.94	0.95	**6951.762**	**6992.861**
Neuroticism									
ADE	43.722	36	0.176	1.170	.023	0.87	0.88	**6955.814**	7002.049
ACE	49.053	36	0.072	1.169	.029	0.79	0.80	6962.017	7008.253
AE	50.416	37	0.070	1.137	.029	0.78	0.80	6960.017	**7001.116**

NOTE: *df* = degrees of freedom; RMSEA = Root Mean Square Error of Approximation; CFI = Comparative Fit Index; TLI = Tucker Lewis Index; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion. Lowest AIC and BIC values for each BFI scale are bolded.

Table 4. *Unstandardized parameter estimates from biometric portion of trait variance moderation models.*

	Interaction Term (v')	Main Effects			
		a	*d*	*e*	Factor Variance
Self-Report					
Openness	-0.001 (0.005)	0.113 (0.678)	**0.569 (0.151)**	**0.800 (0.034)**	0.977
Conscientiousness	0.014 (0.006)	0 (0)	**0.513 (0.045)**	**0.746 (0.037)**	0.820
Extraversion	**0.032 (0.007)**	0 (0)	**0.497 (0.038)**	**0.667 (0.034)**	0.692
Agreeableness	0.008 (0.006)	0 (0)	**0.530 (0.040)**	**0.782 (0.037)**	0.892
Neuroticism	0.013 (0.008)	0 (0)	**0.478 (0.045)**	**0.781 (0.045)**	0.838
Parent-Report					
Openness	0.021 (0.007)	0 (0)	**0.636 (0.037)**	**0.628 (0.034)**	0.799
Conscientiousness	0.013 (0.005)	0 (0)	**0.478 (0.054)**	**0.799 (0.032)**	0.867
Extraversion	0.011 (0.006)	0 (0)	**0.461 (0.062)**	**0.810 (0.039)**	0.869
Agreeableness	0.015 (0.006)	0.360 (0.199)	0.417 (0.192)	**0.754 (0.035)**	0.872
Neuroticism	0.010 (0.007)	0 (0)	**0.570 (0.044)**	**0.768 (0.039)**	0.915

NOTE: Standard errors provided in parentheses. *a* = additive genetic effect; *d* = non-additive genetic effect; *e* = non-shared environmental effect; Factor Variance = $a^2 + d^2 + e^2$ (i.e., trait variance). Because age was centered at 8 years, the main effects represent effects at age 8 years. Bold-face indicates that the parameter was significant at $p < .001$.

Table 5. *Unstandardized parameter estimates from biometric portion of full age moderation models.*

	a	*a′*	*d*	*d′*	*e*	*e′*
Self-Report						
Openness	0.418 (0.235)	-0.036 (0.026)	0.340 (0.237)	0.031 (0.025)	**0.847 (0.045)**	-0.009 (0.007)
Conscientiousness	0 (0)	0 (0)	**0.423 (0.067)**	0.023 (0.010)	**0.795 (0.047)**	0.002 (0.008)
Extraversion	0.238 (0.182)	-0.035 (0.026)	0.227 (0.106)	**0.063 (0.014)**	**0.791 (0.049)**	-0.004 (0.009)
Agreeableness	0 (0.002)	0 (0)	**0.391 (0.084)**	0.029 (0.013)	**0.854 (0.055)**	-0.007 (0.009)
Neuroticism	**0.493 (0.09)**	**-0.051 (0.012)**	0.027 (0.154)	**0.070 (0.020)**	**0.845 (0.060)**	-0.004 (0.011)
Parent-Report						
Openness	0.259 (0.234)	-0.040 (0.038)	**0.568 (0.105)**	0.024 (0.016)	**0.645 (0.056)**	0.010 (0.009)
Conscientiousness	0 (0)	0 (0)	**0.434 (0.099)**	0.014 (0.015)	**0.822 (0.048)**	0.006 (0.008)
Extraversion	0 (0)	0 (0)	**0.345 (0.103)**	0.028 (0.016)	**0.863 (0.047)**	-0.002 (0.008)
Agreeableness	0.332 (0.185)	0.010 (0.014)	0.364 (0.180)	0.019 (0.014)	**0.786 (0.042)**	0.004 (0.008)
Neuroticism	0.097 (0.128)	-0.068 (0.037)	**0.465 (0.108)**	0.008 (0.036)	**0.844 (0.060)**	-0.008 (0.010)

NOTE: Standard errors provided in parentheses. *a* = additive genetic effect; *d* = non-additive genetic effect; *e* = non-shared environmental effect. ' indicates the moderation terms for *a*, *d*, and *e*. Bold-face indicates that the parameter was significant at $p < .001$.

Table 6. *Model fits for alternative age moderation models.*

	Self-Report					Parent-Report				
Model	df	LL	Scaling Factor	AIC	BIC	df	LL	Scaling Factor	AIC	BIC
Openness										
None	9	-3815.251	1.205	**7648.502**	**7695.603**	9	-3390.737	1.160	6799.473	6845.709
Trait	10	-3815.219	1.209	7650.458	7702.773	10	-3381.553	1.233	**6783.106**	**6834.479**
ADE	12	-3814.081	1.123	7652.161	7714.963	12	-3381.117	1.335	6786.234	6847.882
A	10	-3815.097	1.190	7650.194	7702.528	10	-3383.819	1.286	6787.639	6839.011
D	10	-3815.179	1.206	7650.358	7702.693	10	-3382.342	1.232	6784.684	6836.057
E	10	-3815.109	1.210	7650.219	7702.553	10	-3383.269	1.217	6786.539	6837.911
Conscientiousness										
None	9	-3810.788	1.011	7639.576	7686.677	9	-3451.159	1.060	6920.317	6966.553
Trait	10	-3807.039	1.038	7634.077	7686.412	10	-3447.580	1.052	**6915.159**	**6966.532**
ADE	12	-3805.929	0.937	7635.858	7698.659	12	-3447.399	0.968	6918.798	6980.445
A	10	-3808.800	1.036	7637.601	7689.935	10	-3449.849	1.073	6919.698	6971.070
D	10	-3805.955	1.009	**7631.910**	**7684.245**	10	-3447.687	1.059	6915.374	6966.747
E	10	-3808.145	1.040	7636.290	7688.625	10	-3447.945	1.048	6915.891	6967.264
Extraversion										
None	9	-3816.753	1.059	7651.505	7698.606	9	-3438.637	0.945	6895.274	6941.509
Trait	10	-3798.962	1.098	7617.924	7670.259	10	-3435.778	0.973	**6891.556**	6942.929
ADE	12	-3792.276	1.086	7608.552	7671.354	12	-3434.643	0.896	6893.287	6954.934
A	10	-3798.417	1.060	7616.834	7669.169	10	-3437.680	0.995	6895.361	6946.733
D	10	-3792.735	1.084	**7605.470**	**7657.805**	10	-3434.664	0.963	6889.328	**6940.701**
E	10	-3804.033	1.102	7628.066	7680.401	10	-3436.335	0.973	6892.671	6944.044
Agreeableness										
None	9	-3835.912	1.011	7689.824	**7736.925**	9	-3467.117	1.203	6952.233	6998.469
Trait	10	-3834.530	1.051	7689.059	7741.394	10	-3461.944	1.203	6943.888	6995.261
ADE	12	-3832.324	0.973	7688.648	7751.450	12	-3461.153	1.133	6946.306	7007.954
A	10	-3834.480	1.038	7688.961	7741.295	10	-3461.937	1.152	6943.875	6995.247
D	10	-3832.802	1.018	**7685.604**	7737.939	10	-3461.472	1.159	**6942.943**	**6994.316**
E	10	-3835.217	1.065	7690.434	7742.769	10	-3463.203	1.207	6946.405	6997.778
Neuroticism										
None	9	-3827.409	1.040	7672.817	7719.918	9	-3468.907	1.209	6955.814	7002.049
Trait	10	-3823.808	1.200	7667.615	7719.950	10	-3466.481	1.320	6952.962	7004.334
ADE	12	-3818.972	1.174	**7661.944**	7724.745	12	-3462.369	1.478	6948.738	7010.385
A	10	-3823.629	1.197	7667.257	7719.592	10	-3462.786	1.486	**6945.571**	**6996.944**
D	10	-3821.770	1.315	7663.540	**7715.874**	10	-3464.573	1.356	6949.146	7000.519
E	10	-3824.589	1.203	7669.178	7721.512	10	-3467.446	1.298	6954.892	7006.264

NOTE: Bold-face represents best fit statistics. df = degrees of freedom; AIC = Akaike Information Criterion; BIC = Bayesian Information Criterion. Lowest AIC and BIC values for each BFI scale are bolded. Model fit indices that are derived from comparing model-implied covariance matrices to those from a fully saturated model (e.g. Chi Square, RMSEA, CFI, TLI) are not available because, rather than assuming a single covariance

matrix, age moderation models allow for the covariances to vary across the age range. None = no age moderation allowed; Trait = phenotypic variance moderation model; ADE = full age moderation model. A = Additive genetic variance only moderation model; D = Non-additive genetic variance only moderation model; E = Non-shared environmental variance only moderation model.

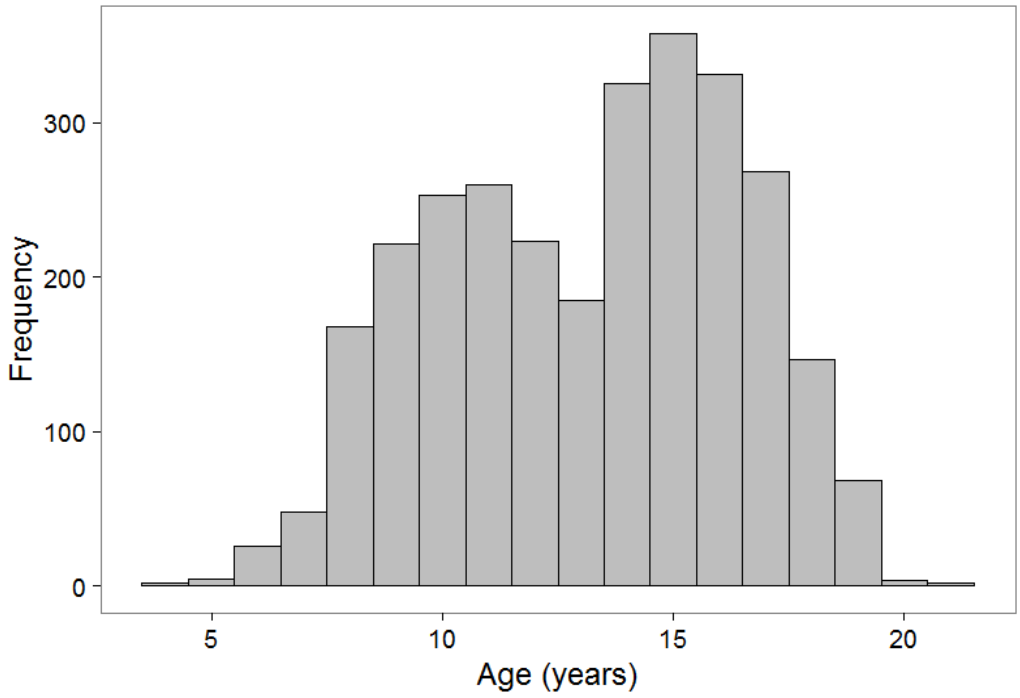

Figure 1. *Histogram of age distribution.*

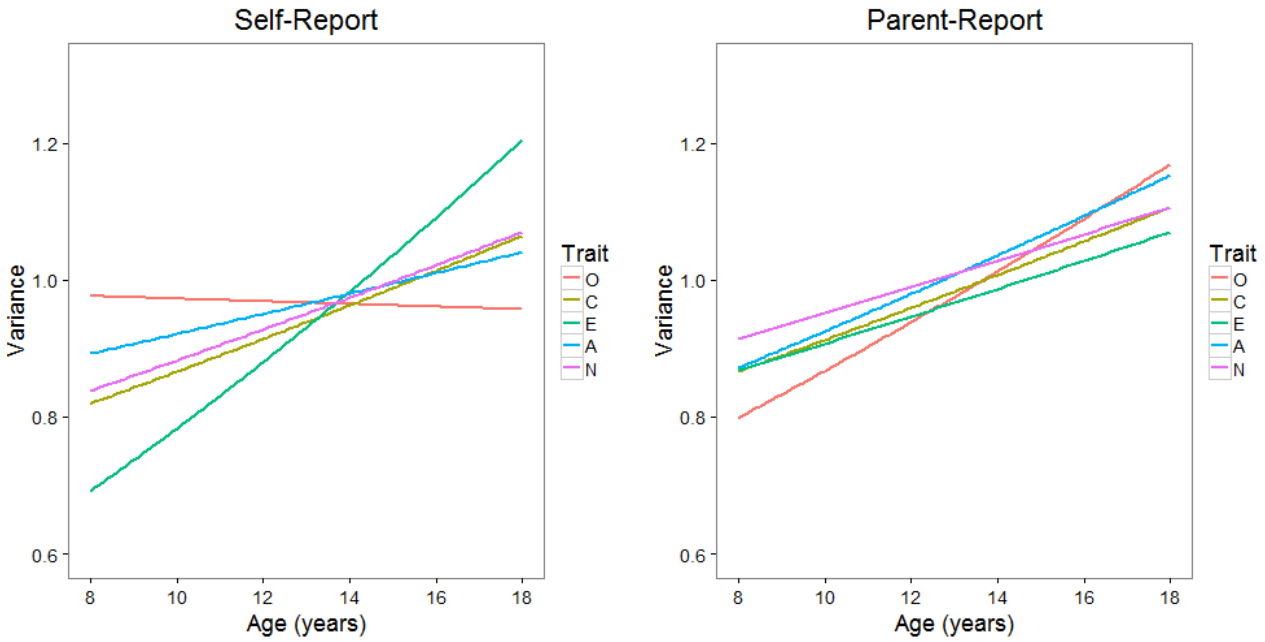

Figure 2. *Linearly estimated age trends in phenotypic variance in self- and parent-reports of Big Five traits (O = openness; C = conscientiousness; E = extraversion; A = agreeableness; N = neuroticism).*

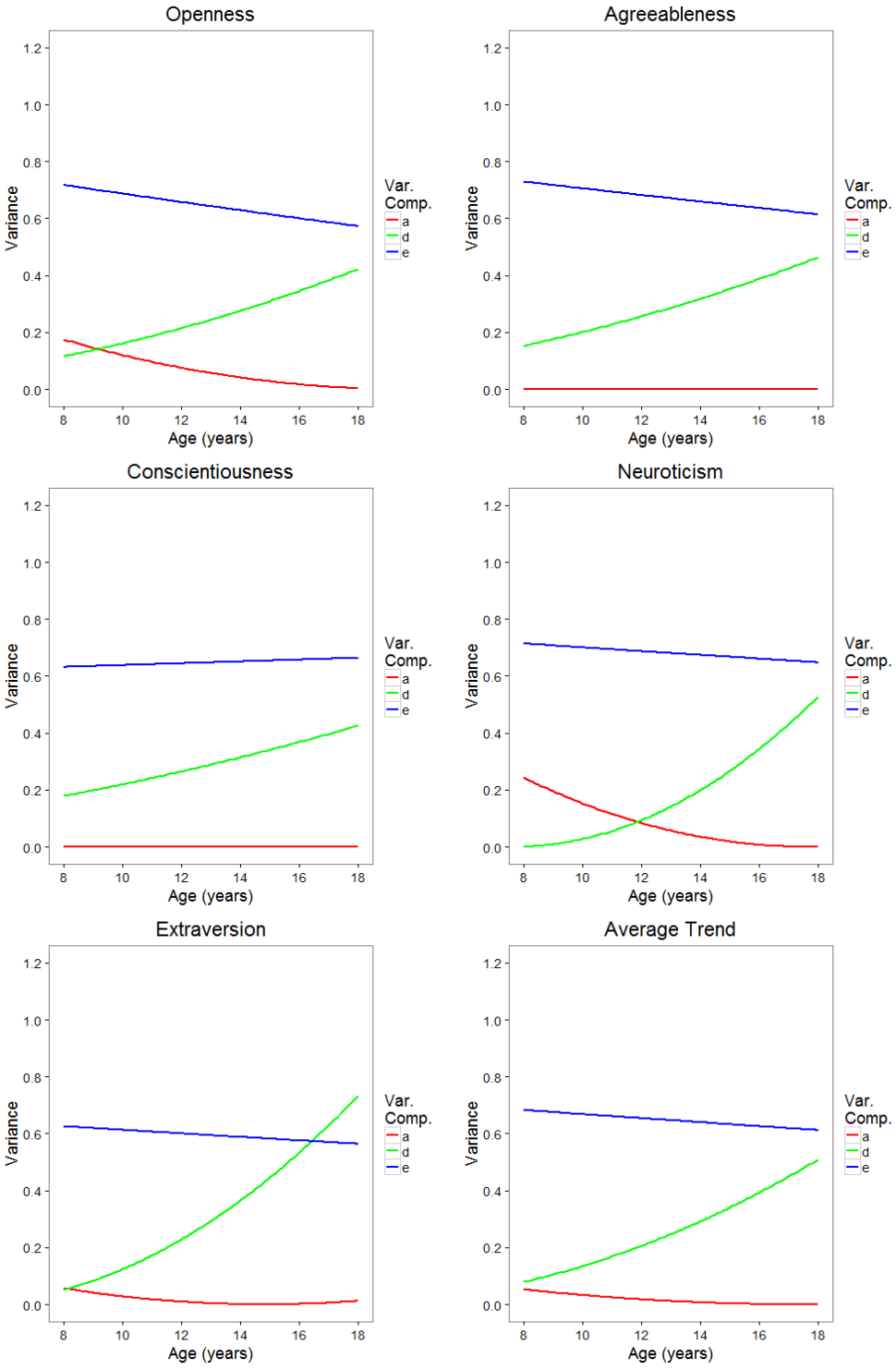

Figure 3. *Age trends in the variance of additive (a) and non-additive (d) genetic and non-shared environmental (e) components in self-reported Big Five scores.*

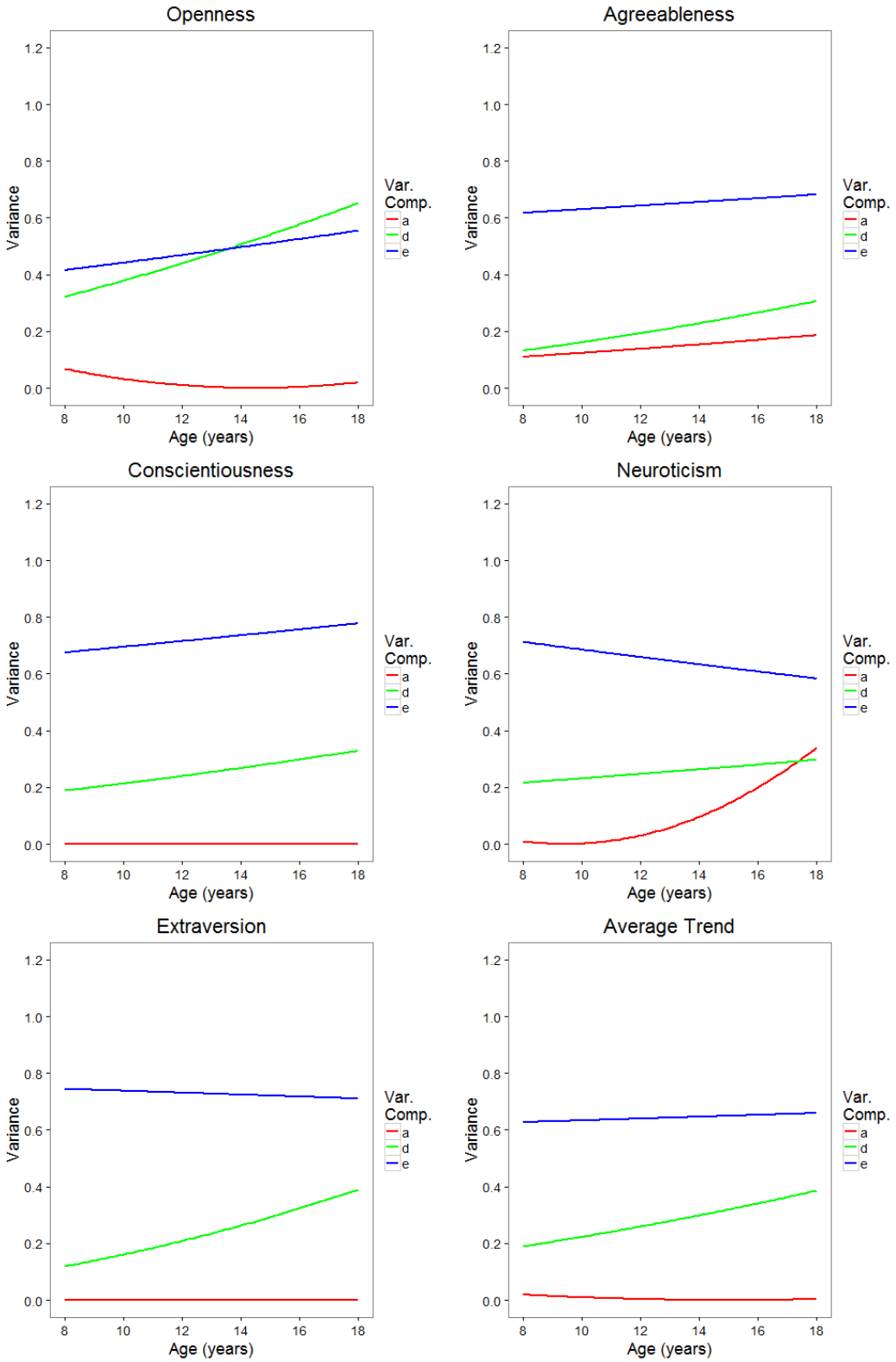

Figure 4. *Age trends in the variance of additive (a) and non-additive (d) genetic and non-shared environmental (e) components in parent-reported Big Five scores.*

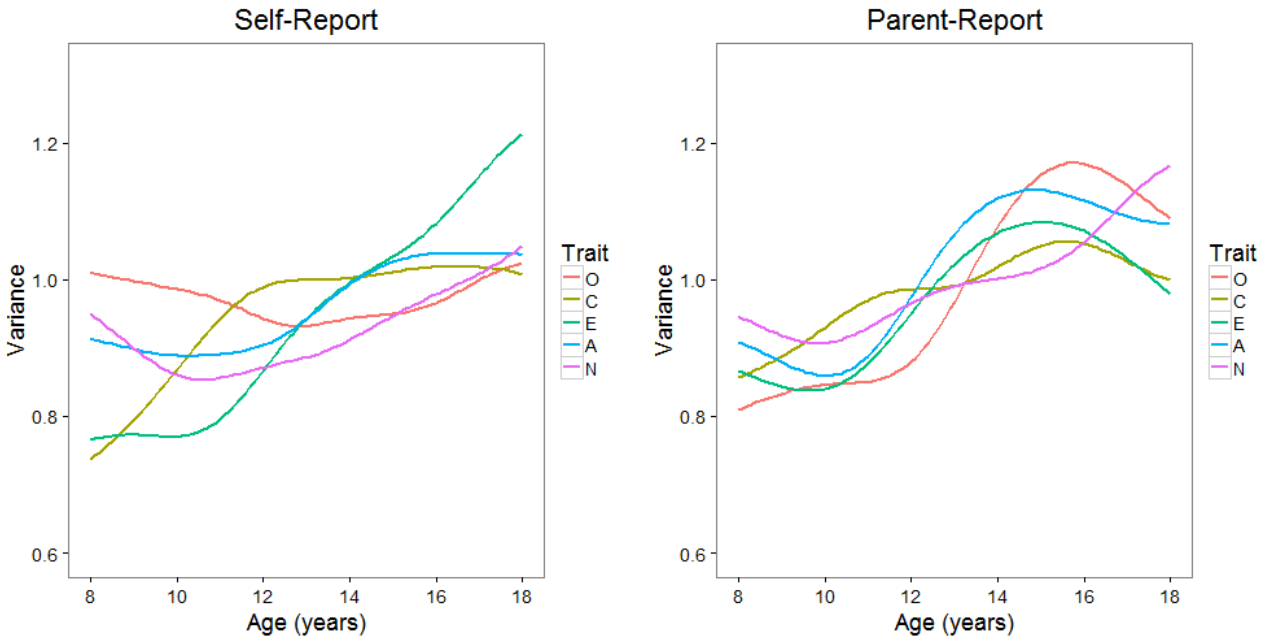

Figure 5. *Non-linear age trends in phenotypic variance in self- and parent-reports of Big Five traits (O = openness; C = conscientiousness; E = extraversion; A = agreeableness; N = neuroticism).*

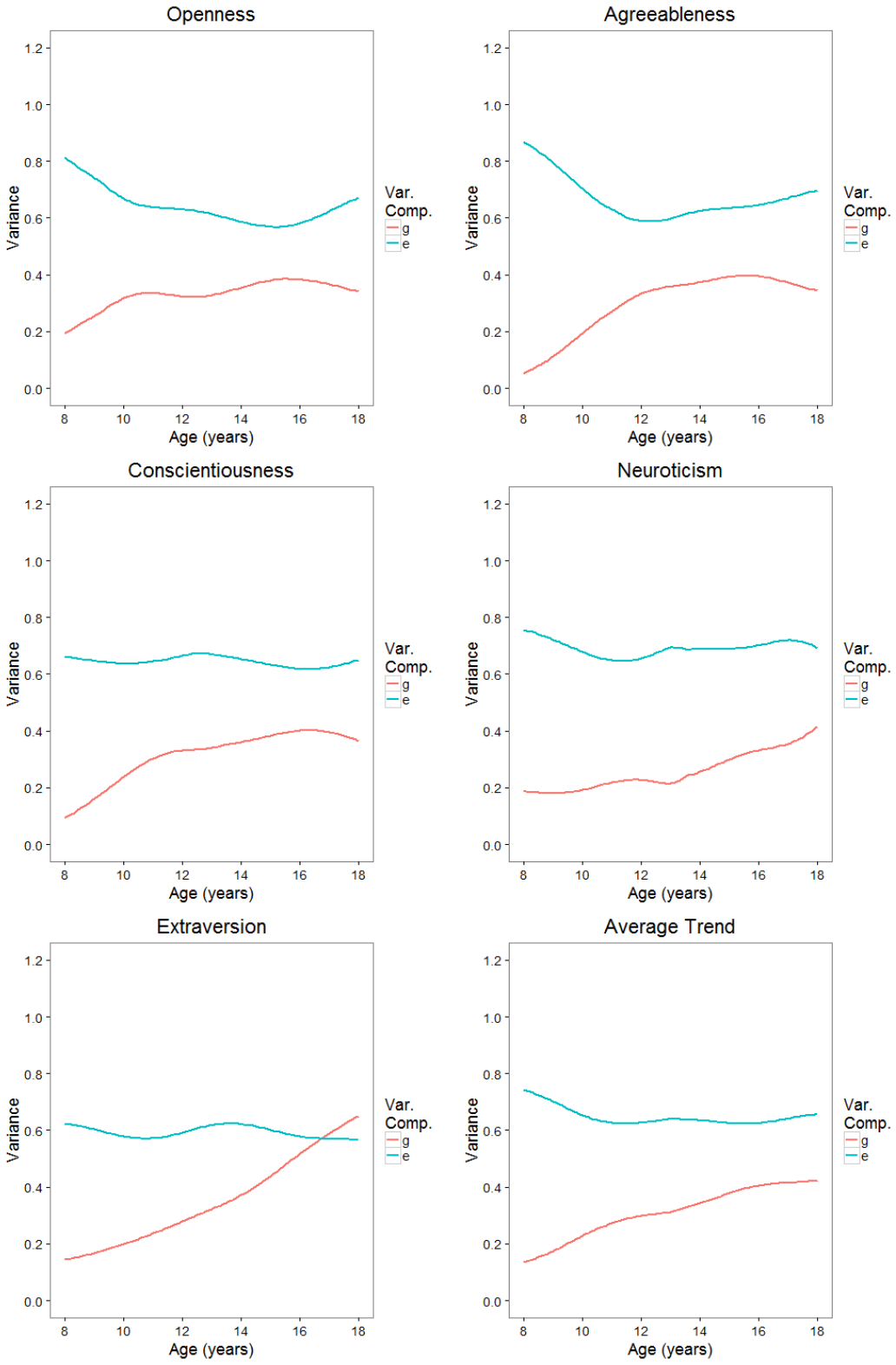

Figure 6. *Age trends in the variance of genetic (combined additive and non-additive effects; g) and non-shared environmental (e) components in the self-reported Big Five scores.*

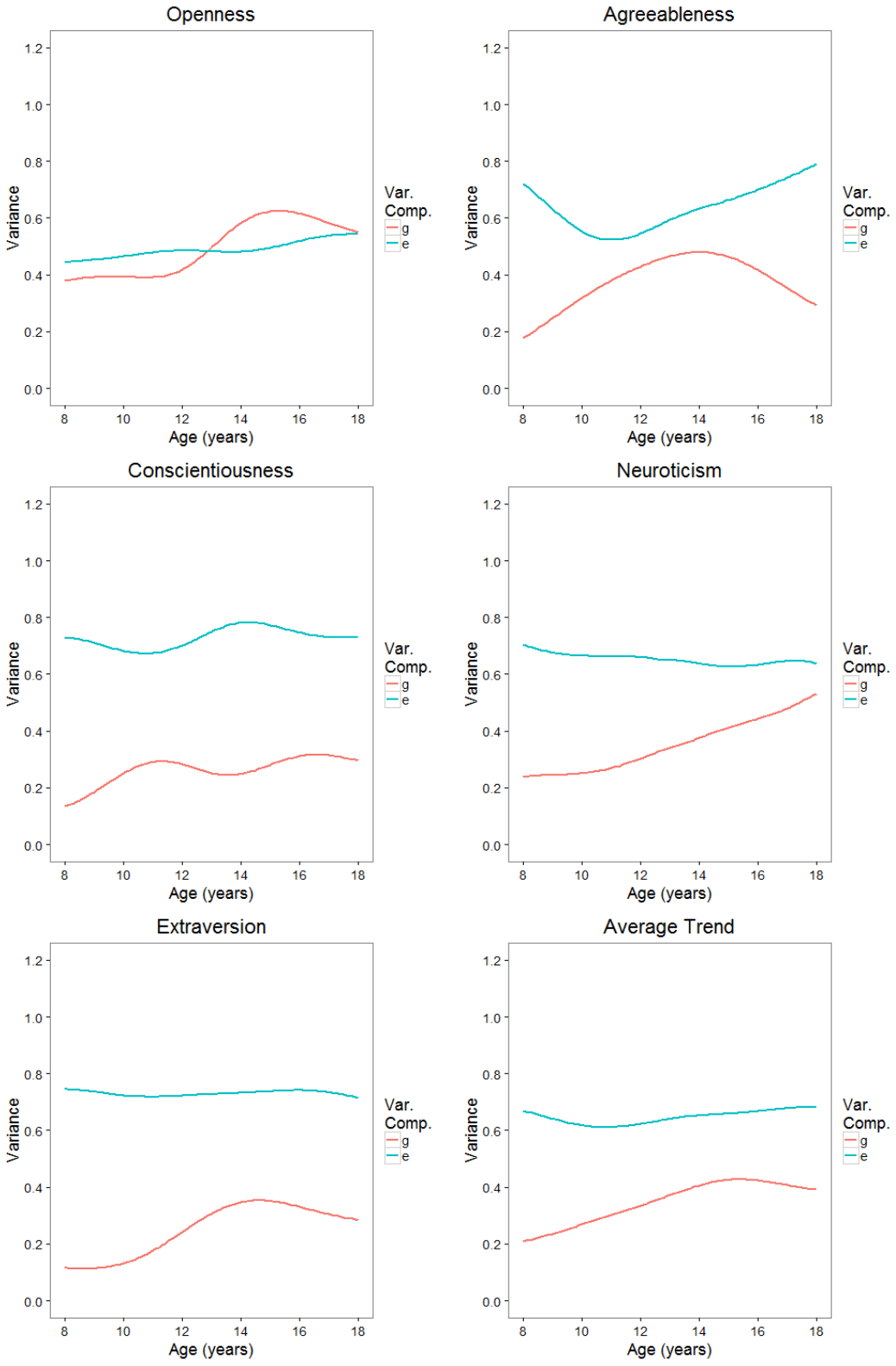

Figure 7. *Age trends in the variance of genetic (combined additive and non-additive effects; g) and non-shared environmental (e) components in the parent-reported Big Five scores.*