June 20, 2017

Theoretical Concepts in the Genetics of Personality Development

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To appear in:

Handbook of Personality Development
Dan P. McAdams, Rebecca L. Shiner, and Jennifer L. Tackett (Eds.)

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Theoretical Concepts in the Behavioral Genetics of Personality Development

How do genetic and environmental factors combine to give rise to individual differences in personality? How do such factors operate over time to give rise to individual differences in personality development and change? These are some of the most fundamental questions within differential psychology. However, the answers to these questions are not straightforward. Individual differences may arise, persist forward, and decay over time by way of a complex system of endogenous and contextual influences that themselves come to be correlated with and interact with one another over time. In this chapter we review several classes of developmental mechanisms that may influence the course of personality stability and change. We begin by describing behavioral genetic methodology and some of the key descriptive findings that such methodology has produced. We then describe and illustrate several theoretical mechanisms of the genetics of personality development, and consider how such mechanisms may combine and interact.

Developmental Behavioral Genetic Methods and Key Descriptive Findings

Standard behavioral genetic methods provide a descriptive account of the extent to which individual differences in personality are statistically associated with genetic differences between people, the extent to which individual differences in personality are associated with the rearing environment that individuals share with their siblings (the shared environment), and the extent to which individual differences in personality persist after accounting for these two factors (the nonshared environment). When appropriately applied to longitudinal data, behavioral genetic methods can also be used to provide a descriptive account of the extent to which rank-order stability and individual differences in change are associated with genetic, shared environmental, and nonshared environmental factors. Just as is the case for descriptive information regarding phenotypic (i.e. observed) age trends in the mean, variability, and rank-order stability of personality traits, such descriptive information regarding genetic and environmental components of individual differences can be highly valuable for distinguishing between alternative theories of personality development, constraining theories of personality development, and generating new mechanistic theories of personality development. For instance, phenotypic trends have served as the basis for foundational principles in lifespan personality psychology (Roberts, Wood, &

Caspi, 2008), such as the cumulative continuity principle (that the rank-order stability of personality increases with age), the maturity principle (that people, on average, become more dominant, agreeable, conscientious, and emotionally stable, from adolescence forward), and the corresponsive principle (that the typical effect of life experience on personality is to magnify the characteristics that led to those experiences), however, empirical work to identify the precise contexts and life experiences that undergird these processes is still preliminary (Bleidorn, Hopwood, & Lucas, 2016). Similarly, standard behavioral genetic methods provide key information about how latent genetic and environmental sources of variation contribute to individual differences in personality variation, stability, and change that can be used to construct, develop and test theory, but they do not by themselves provide direct empirical information about how specific genes, contexts, or life experiences combine and interact over development to shape personality. As we discuss at the end of this chapter, we believe that a more complete understanding of how individual differences in personality arise and develop over time can be achieved through careful integration of behavioral genetic theory, methods, and results into ongoing theoretical and empirical work in mainstream personality psychology.

Conventional work in the behavioral genetics of personality largely focused on single point estimates of heritability of personality. For instance, point estimates for the heritability of all of the Big Five personality traits have been reported to be approximately .40-.60 (for a review see Bouchard & McGue, 2003), with no consistent differences reported across different Big Five traits (Turkheimer, Pettersson, & Horn, 2014). Evidence for genetic influences on personality are derived from the observation that genetically more related individuals (e.g. identical twins) are more similar in their personality traits than genetically less related individuals (e.g. fraternal twins), even when holding shared rearing environment constant across relationship types. Also of note is that, after accounting for genetic relatedness, individuals reared together are no more similar to one another in their personalities than would be expected for individuals chosen at random out of the population. Nongenetic factors that differentiate individuals regardless of whether their rearing environment was shared with one another are termed the *nonshared environment.* These two important and interesting observations, that the heritability of personality is approximately 40%-60% at the population level and that nongenetic variation in personality is attributable to nonshared environmental factors, are the primary findings from

behavioral genetics used to inform conventional personality theories. Yet, they do not do justice to the important developmental patterns in the genetics of personality.

The relative influence of genetic and environmental effects may shift across the lifespan, rather than remain static. Age trends in the heritability of personality have been reported in quantitative syntheses by Kandler (2012) for Neuroticism and Extraversion, and Briley and Tucker-Drob (2014) for all of the Big Five. In both syntheses, age trends have been very similar across each of the Big Five traits. We therefore consider the aggregate trend reported by Briley & Tucker-Drob (2014). Figure 1 presents the main findings. It can be seen that the heritability of personality (left panel) is highest in early life, decreasing most precipitously in childhood and adolescence, and far more gradually in adulthood, with a countervailing trend for environmentality (right panel). Heritability of personality is estimated at approximately 70% in early childhood, declines to approximately 50% by late adolescence, and subsequently declines to approximately 35% by late adulthood. Nonshared environmentality increases from approximately 30% to 50% to 65% from infancy to late adolescence, to late adulthood. However, at least some of this trend may reflect method bias, as nearly all of the effect sizes for very young children come from parent-reports. These ratings may exaggerate differences between siblings and thus inflate heritability.

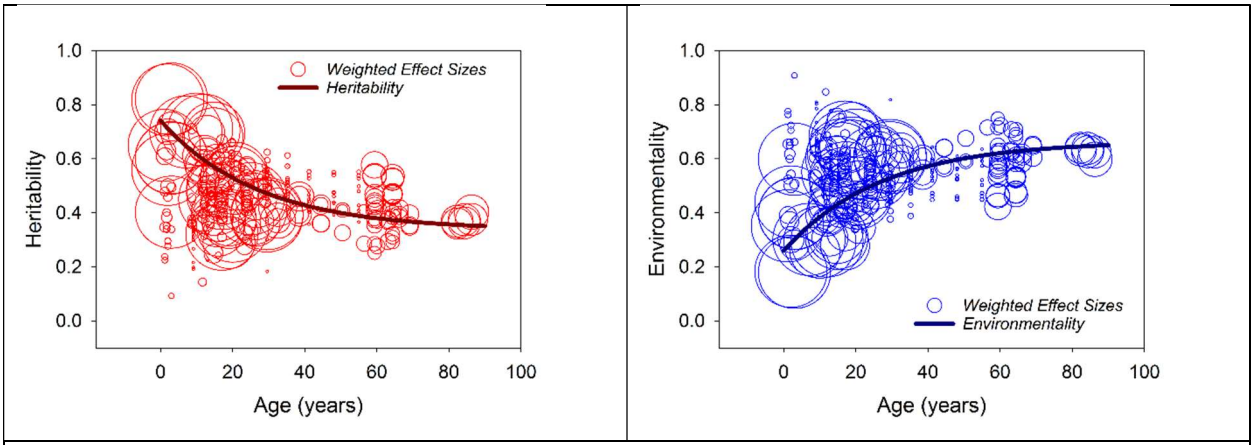

Figure 1. Genetic and (nonshared) environmental influences on individual differences in personality across the lifespan. Shared environmental influences on personality tend to be absent at all ages, and are therefore not modelled. Meta-analytic results from longitudinal behavioral genetic studies. From Briley and Tucker-Drob (2014).

Behavioral genetic methods have also been applied to probe the role of genetic and environmental factors in the rank order stability of personality over time. Rank-order stability refers to the correlation between individual differences in personality at one point in time and a later point in time. As patterns for rank-order stability are very similar across Big Five traits, we focus on the aggregate pattern here. Two major research syntheses have described the overall phenotypic pattern of age trends in rank-order stability that behavioral genetic research has gone on to probe. As displayed in the top two panels of Figure 2, results from both Roberts and DelVecchio (2000) and Ferguson (2010) have indicated that the rank-order stability of personality (over an average time interval of approximately 6.6 years) increases relatively monotonically over the first thirty years of life, after which point it remains very high. Roberts and DelVecchio (2000) reported increases in rank-order stability from approximately .35 in infancy to .65 by age thirty years. Ferguson (2010), who disattenuated stability coefficients for unreliability, reported somewhat higher stability coefficients, with rank-order stability increasing from approximately .50 to .90 over this same age range. Briley and Tucker-Drob (2014), Kandler (2012), and Turkheimer et al. (2014) have all meta-analytically examined the role of genetic and environmental factors in rank-order stability. Results from Briley and Tucker-Drob (2014), who provide the most comprehensive treatment of the topic, are represented in the bottom two panels of Figure 2. These plots indicate the genetic (red) and nonshared environmental (blue) contributions to overall phenotypic stability (green) as a function of age, over an average longitudinal time-lag of approximately 5.6 years. First, it can be seen that the overall trend in phenotypic stability closely matches the trend reported by Roberts and DelVecchio (2000), with rank-order stability increases from approximately .35 in infancy to .65 by age 30 years. Behavioral genetic decomposition indicates that stability in infancy is exclusively driven by genetic factors, and that the genetic contribution remains at the same level (~.35) for the entirety of the lifespan. A genetic contribution to stability refers to the extent to which the correlation between the same personality trait across two points in time is statistically accounted for by the influence of overlapping sets of genetic variants on that trait at both time points. (Genetic factors would not contribution to stability, even if personality were heritable at both time points, if different sets of genes were responsible for individual differences in personality at the two time points). Increasing phenotypic stability with age is driven exclusively by an increase in the nonshared environmental contribution to stability from no contribution in infancy to

approximately .35 correlation units by late adulthood, such that the overall phenotypic stability increases from approximately .35 to .70 during this time. A nonshared environmental contribution to stability refers to the extent to which the correlation between the same personality trait across two points in time is statistically accounted for by the influence of overlapping or covarying nonshared environmental factors at both points in time. (If entirely different and uncorrelated environmental factors, e.g. adolescent peer group and adult occupational stress, are responsible for individual differences in personality at two different time points in time, then environmental factors will not contribute to stability, even though they contribute to individual differences at each time point).

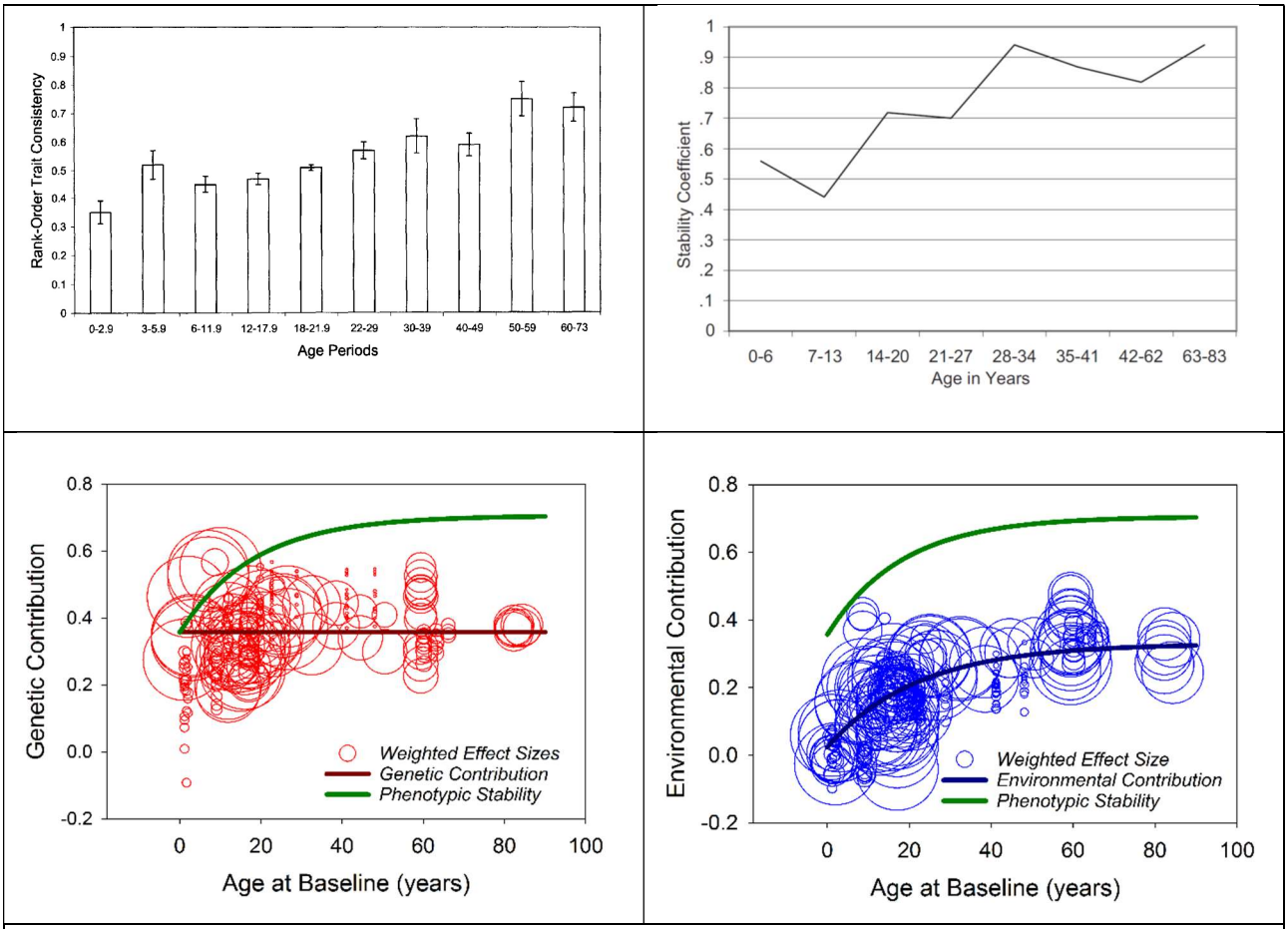

Figure 2. Top left panel: Longitudinal rank order (test-retest) stability of personality from Roberts and DelVecchio (2000) meta-analysis. **Top right panel:** Disattenuated longitudinal rank order (test-retest) stability of personality from Ferguson (20010) meta-analysis. **Bottom left panel:** Genetic contribution to longitudinal rank order (test-retest) stability of personality from Briley & Tucker-Drob (2014) meta-analysis. **Bottom right panel:** Environmetnal contribution to

longitudinal rank order (test-retest) stability of personality from Briley & Tucker-Drob (2014) meta-analysis.

How can a researcher or student without a strong background in behavior genetics make intuitive sense of these findings? For instance, what is the meaning of the finding that the overwhelming majority of stable variance in personality at early ages (e.g. age five years) is driven by genetic factors? One way to describe this finding is that, if each person in the population was raised together with an identical twin (with whom they have 100% of DNA in common), then the level of a personality trait (e.g. extraversion) for a given person at age five would be nearly as predictive as her cotwin's score on that personality trait at age 15 as it was of her own score at age 15. Alternatively, if each person in the population were raised tougher with a fraternal twin (with whom they share only 50% of the DNA variation that differs within humans), then the level of a personality trait for a given person at age five would be less predictive of her cotwin's score on that personality trait at age 15 than it was of her own score at age 15. This knowledge, that stability of earlier personality is mediated by genetic factors, can help us to reduce the possible set of causal mechanisms for personality stability to a subset that is most plausible. For example, on the basis of this result, it might be fruitful to examine how DNA sequence variation relates to the efficiency of hormonal production, brain structure, or some other biological process. It may be similarly fruitful to examine how individuals evoke or actively create environmental experiences that are in some way linked to their temperaments. (We discuss below why this seemingly environmentally mediated process is relevant in the context of large or total genetic influence on stability). However, the finding that environmental factors contribute negligibility to stability of early personality rules out major roles for random sorts of environmental experiences (e.g., car crashes, illnesses, uncontrollable family events) in early life from in the stability of personality in the population at large. Moreover, that the shared environment does not account for personality variation, further reduces the viability of examining differences in experience that cluster within families as systematic correlates of personality variation. In other words, the behavior genetic results constrain the types of likely explanatory mechanisms that warrant further investigation.

Now, let's turn to another classic empirical problem: what drives personality development during emerging adulthood? The meta-analytic findings described earlier indicate

that the nonshared environment plays an emerging and increasing role in personality stability in adolescence through middle adulthood. In other words, superimposed on a backdrop of genetically-mediated stability, the nonshared environment plays an increasing role in the longitudinal stability of personality traits over the life course. This finding suggests a number of promising sources of stable variation in personality in adolescent and young adulthood warranting further investigation. For instance, researchers may do well to examine effects of peer groups, social clubs, occupational roles, and the general social niches that individuals adopt as they become adults. As we discuss later, the nonshared environment also includes potential interactions between environmental experiences and genetics. For instance, genetic differences between people may predict how they respond to unique environmental experiences. For instance, it is possible that some individuals may experience social rejection by withdrawing from further social interactions, while others may redouble their efforts to integrate themselves socially. Drawing these sorts of insights is not obvious or straightforward from the results presented above. We suggest that, before such quantitative genetic results can be integrated more fully into personality theory, it is useful to articulate the sorts of empirical patterns that would result from different mechanisms of personality development.

Mechanisms of Personality Development

Several theoretical perspectives have proposed developmental processes that give rise to individual differences in personality and their stability and change over time. Here we attempt to articulate the sorts of empirical patterns that each of several archetypical theoretical processes would be expected to produce.

Genetic Set Point

One of the most longstanding perspectives on the basis of individual differences of personality holds that endogenous genetic factors determine each individual's level of each personality trait, and that experiential factors have only transient, short-lived, effects on personality. Under this perspective, perturbations in trait levels result from exposure to novel environmental experiences, but these perturbations rapidly decay over time such that individual personality levels regress back toward their person-specific genetic set points. For example, someone may act more open to new experiences when traveling abroad for a summer, but levels of openness to experience would be expected to return to a set-point shortly after returning.

Under this *genetic set point* hypothesis (Figure 3, left panel), even if exposure to the novel environment persists over time—either through repeated exposure, or via permanent wholesale change in environmental context—individuals acclimate to the novelty and traits regress back to their genetic set points. For instance, face with the challenge of making new friends, an individual may increase in extraversion shortly after permanently moving to a new city. However, after a few months of establishing a routine, the genetic setpoint would predict that her extraversion levels would return back to a pre-existing setpoint.

According to early theoretical work (e.g. Waddington, 1942), individuals evolved to have buffered responses to environmental variability such that each genotype would produce a relatively constant ultimate phenotype. Conley (1984) hypothesized that "the heritabilities of intelligence and personality would produce innate individual differences which could to a greater or lesser degree resist the destructuring, randomizing influence of the environment" (p. 22). Scarr (1992) speculated that "ordinary differences between families have little effect on children's development, unless the family is outside of a normal, developmental range" (p. 15), and that "Fortunately, evolution has not left development of the human species, nor any other, at the easy mercy of variations in their environments. We are robust and able to adapt to wide-ranging circumstances… If we were so vulnerable as to be led off the normal developmental track by slight variations in our parenting, we should not long have survived" (p. 16). Indeed, some degree of *penetrance* of genetic variability into phenotypic variability is necessary for natural selection to occur (Falconer & Mackay, 1996).

Support for a genetic set point understanding of personality comes from several sources. Theoretically, genetic set points are included in many models of personality development. For example, Fraley and Roberts (2005) include a form of a genetic set point as a "constancy factor" due to the fact that DNA sequence variation remains unchanged across the lifespan (importantly, the authors build several other developmental processes on top of this set point). In the behavior genetic literature, a classic piece of evidence comes from the Minnesota Study of Twins Reared Apart (Bouchard et al., 1990). This study tracked identical and fraternal twins put up for adoption and placed in different homes weeks after their birth. Apart from their time together in the womb, these twins shared none of the same upbringing, but they did share DNA. Bouchard and colleagues (1990) reported, remarkably, that the reared apart identical twins correlating at .50, only slightly lower than the expected differential stability of personality in adulthood. More

recently, Kandler et al. (2010) used a sophisticated longitudinal behavior genetic design to test for a genetic set point. Their impressive model incorporated three waves of self- and informant-reports of personality to alleviate concerns of measurement error. Although their results were complicated (we will return to this study below), they found some support for a genetic set point. Specifically, variance that was stable across all three waves of data collection was solely associated with genetic effects, and they found that variance specific to a measurement occasion decreased, consistent with the idea that individuals shift in personality but return to a genetically influenced set point.

Moving Set Point (Genetic Influences on Developmental Change)

A slightly more nuanced version of the genetic set point hypothesis does not require that person-specific genetic set points remain fixed over development. Proponents of this hypothesis have suggested that developmental changes in genetic set points may result from "biologically based intrinsic maturation" (Costa & McCrae, 2006). Under this *moving set point* hypothesis (Figure 3, right panel), genetic factors are thought to determine the longitudinal trajectory of trait levels over development, such that each person's set point moves with age, and environmental experiences are expected to have short-lived effects, such that trait levels regress to the person-specific age-specific set point post initial exposure. Such an elaboration helps to account for the sizable and consistent developmental trajectories of mean levels of personality traits (Roberts, Walton, & Viechtbauer, 2006; Srivastava, John, Gosling, & Potter, 2003). The moving set point hypothesis is also somewhat consistent with findings of moderate-to-large genetic influences on individual differences in long-term (e.g. five or more year) changes in personality traits (e.g. Harden, Quinn, & Tucker-Drob, 2011; Bleidorn et al., 2009), although in its purest form it would incorrectly predict very minimal environmental influences on such changes. Indeed, although Kandler et al. (2010) found some evidence for genetic influences on personality maturation (consistent with a moving set point), they also found substantial environmental variance in personality development, much of which was stable and carried forward across time (contrary to a strict interpretation of a moving set point). The moving-set point hypothesis is also consistent with the moderate heritability and robust genetic contributions to longitudinal stability of personality traits across the lifespan. Environmental influences on personality are expected to occur at all phases of development, but expected to be short-lived over time: New environmental influences are expected to arise as old environmental influences dissipate. Thus the (moving)

genetic set point hypothesis is by itself not well-equipped to account for decreasing heritability and increasing environmentality of personality with age, or the increasing contribution of environmental factors on longitudinal stability of individual differences in personality across the lifespan.

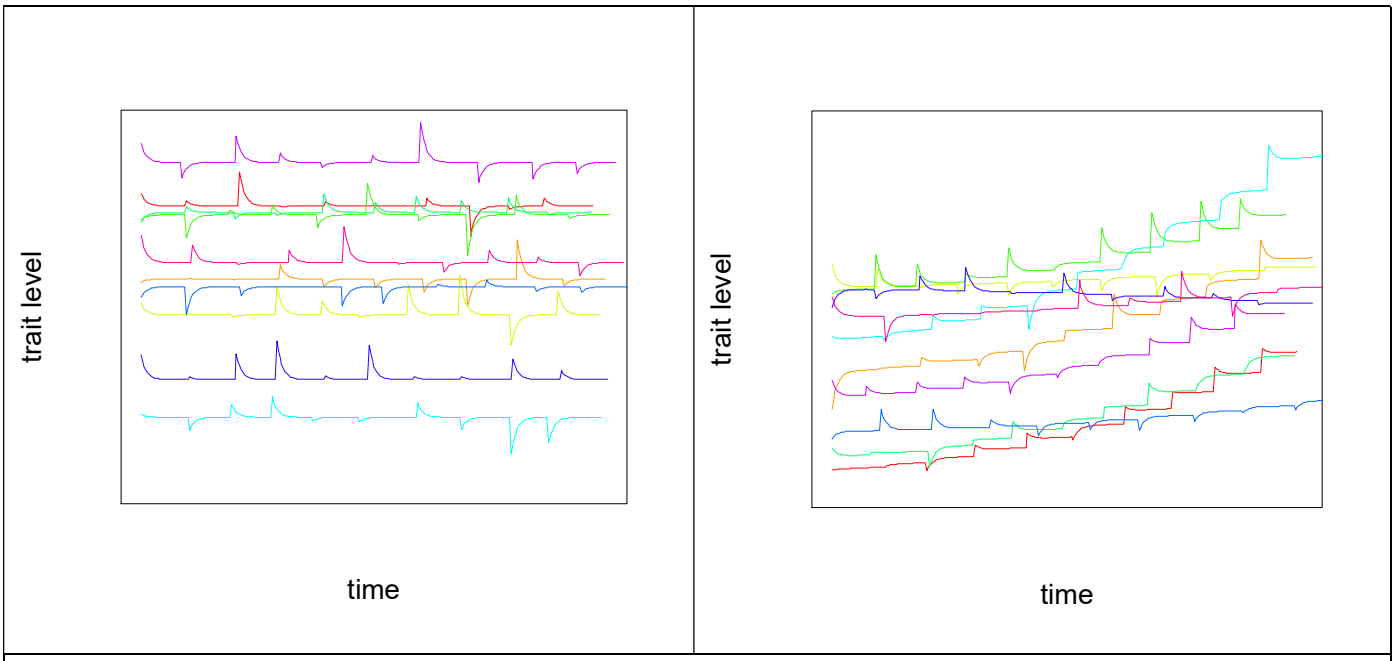

Figure 3. Genetic Set Point (left) and Moving Genetic Set point (right). Randomly experienced environmental events cause short-term perturbations in trait levels that decay over time, such that individuals return to their genetic set point. **Left panel:** The set point remains constant over development for each person. **Right panel:** Genetically-influenced individual differences exist in both the initial set point and the linear rate of change in the set point over development. On average, the genetic set point for the trait increases with development. Each individual randomly experiences different short term events that perturb trait levels. Following perturbations, trait levels regress back to the person-specific age-specific set point.

Random Walk

Random walk mechanisms (Figure 4) contrast starkly with genetic set point mechanisms, in that they predict that trait level change in response to environmental experiences persist forward in time such that trait levels at a given point in time constitute a random deviation from the trait level at the immediately preceding point in time. This can occur either because environmental events are randomly experienced at each point in time, or because trait levels

change as the result of randomly behaving intrinsic processes. In either respect, these trait levels are not expected to regress to a person-specific set point, and changes are instead expected to haphazardly build on one another over time. Taking a step back from formalized mechanisms, a random walk approach to personality has intuitive aspects. How many life experiences have you had that seemed entirely random but affected you for better or worse? Some of the greatest scientific discoveries have occurred through such random events. Taking just one example, penicillin was discovered due to a messy laboratory and a holiday break (Fleming, 1929). More generally, some of life's most important moments appear to happen seemingly at random: the chance encounter with a stranger who turns into a spouse, being in the wrong place at the wrong time while a drunk driver is on the road, or saying just the right thing in a job interview that speaks to the interviewer's past. Each of these random coincidences has the possibility to influence personality development. Using the behavior genetic framework, we can anticipate the expected effects of random walks for empirical findings.

Random walk models provide some appealing mechanistic accounts of certain developmental patterns. For instance they easily account for the increasing nonshared environmentality of personality with age, in that accumulating variation resulting from random walks-if truly random- will be unrelated to either genetic variation or the family environment. As Turkheimer (2000) wrote, "nonshared environmental variability predominates… because of the unsystematic effects of all environmental events, compounded by the equally unsystematic process that expose us to environmental events in the first place" (p. 163). Additionally, under simple random walk models, between-person variability of personality would be expected to increase with age, the stability of personality would be expected to increase with age (because of increasing between-person variability with age, the same magnitude of random deviation from the previous trait level will shift the rank order of individuals to a lesser extent with increasing age), and the increasing stability with age would be expected to be mediated by the nonshared environment. Empirical evidence supports each of these expectations. The stability of personality increases across the lifespan (Roberts & DelVecchio, 2000) primarily due to the nonshared environment (Briley & Tucker-Drob, 2014). Further, the variance of personality also increases in childhood and early adolescence (Mõttus, Soto, & Slobodskaya, in press). Random walk processes will, of course yield incomplete accounts of personality development, insofar as a) personality is genetically influenced, b) environmental experiences are nonrandomly experienced

on the basis of pre-existing individual differences (an issue that we return to in subsequent sections), and c) even those environmental experiences that are initially experienced for random reasons tend to recur, or shape the profile of other future environments that an individual experiences.

One interesting elaboration of the random walk process would hold that the range of possible changes (i.e. the variance the random walk distribution from one time point to the next) decreases over development as individuals form social, educational, and professional niches, individuals form roles and identities, and personality traits crystallize and become resistant to change (Caspi, Roberts, & Shiner, 2005; McCrae & Costa, 1994). This elaboration is not necessary to account for increasing nonshared-environmentally mediated rank-order stability with age, but it would accentuate this process. It would also predict a slowing of increases in between-person variability in personality with age.

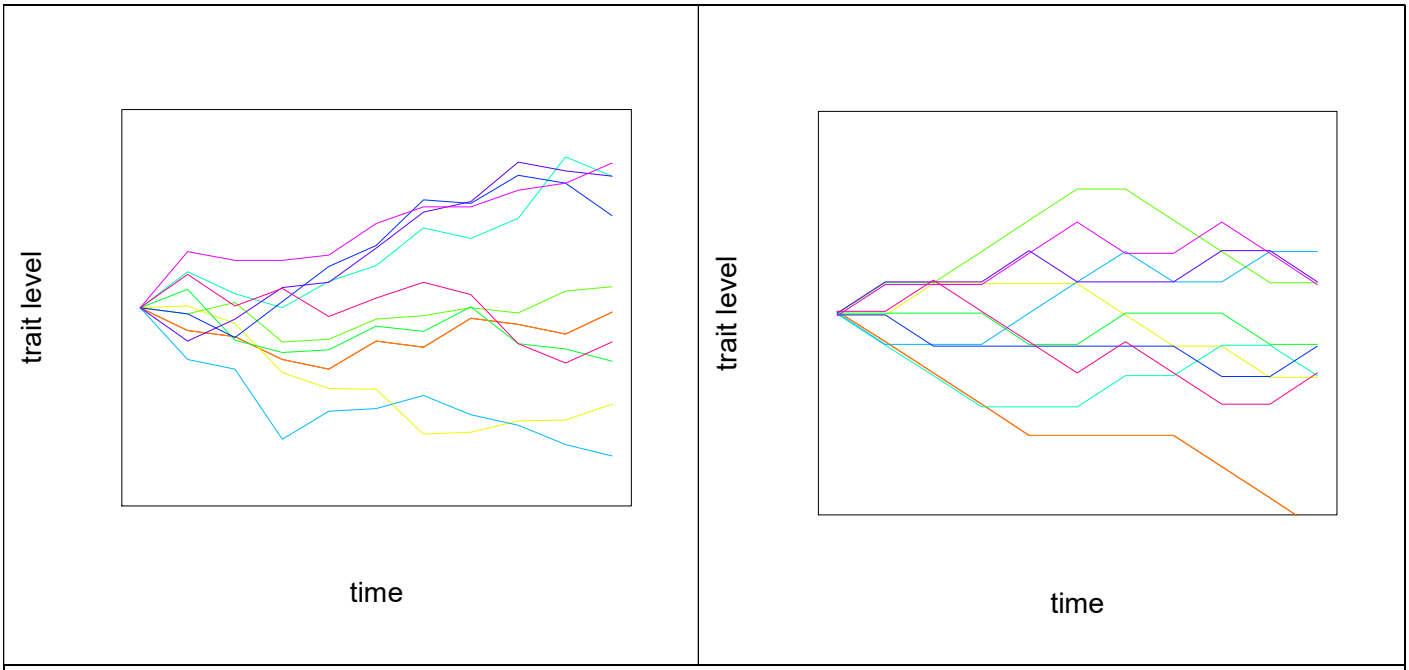

Figure 4. Random walk. Each participant's trait level at a given time point is a random deviation from his or her trait level at the preceding point in time. In the left panel, the deviation is randomly drawn from a continuous normal distribution. In the right panel, the deviation is drawn from a two-trial binomial distribution, generating three discrete levels (increase, decrease, stay).

Heterogeneity in (Near-Universal) Transition Points

An additional mechanism that may contribute to the differentiation of individual differences in personality traits with age involves social or biological transitions that are nearly universally experienced, but experienced according to different developmental schedules. Even in a simplified scenario in which each transition has the exact same effect on personality traits for each individual (Figure 5), variability in the timing of developmental milestones across individuals will result in same-age individuals being at different developmental "stages," and thus evincing different trait levels. Examples of near-universal social transitions include leaving the parental household, completing education, and establishing an occupation, for which timing varies markedly across individuals and has been implicated in personality development (e.g. Bleidorn et al., 2013). Another example is the pubertal transition, for which individual differences in timing are also marked (Mendle et al., 2010) and implicated in the development of both personality and psychopathology (Harden et al., 2015; Kretsch, Mendle, & Harden, 2016; Mendle et al., 2016). More generally, individual differences in timing and pace of psychological and social development has been a focus of considerable attention in life-history theory, a "theory from evolutionary biology that describes the strategic allocation of bioenergetic and material resources among different components of fitness" (Figueredo et al., 2006, p. 244).

Individual differences in timing of transitions may not, by itself, be particularly useful for accounting for established patterns of personality development. Such mechanisms may, however, be a key element in the wider constellation of co-occurring processes that underlie personality development. They are particularly valuable for highlighting that, regardless of the extents to which variation in timing are themselves attributable to genetic and environmental factors, there may be some circumstances in which individual differences in personality traits are better conceptualized as reflective of a particular point in a maturational process, rather than as a characteristic or chronic trait level.

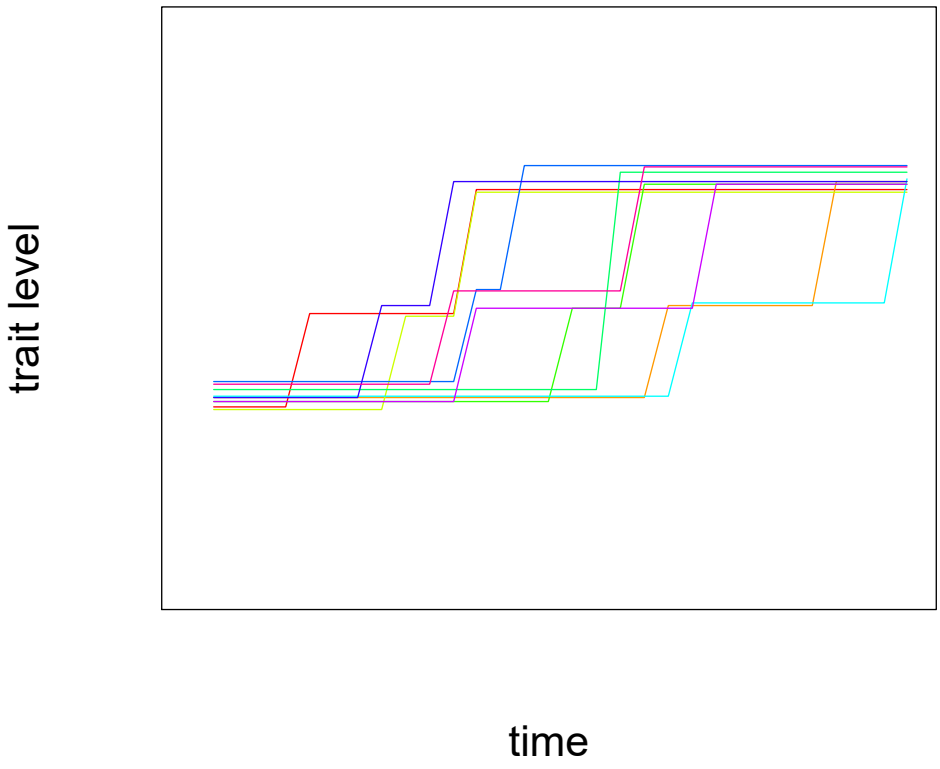

Figure 5. Heterogeneity in Transition Points. In this stylized example, all individuals go through two developmental transitions. Each transition has the exact same influence on personality trait levels for all people, but individuals differ from one another in the timing of these transitions (some individuals may even experience both transitions concurrently). Heterogeneity in timing could be attributable to differences in genetically programmed maturational processes (e.g. pubertal development) and/or to differences in the timing of social transitions (children leaving their parent's home, marriage, parenthood). We allow for individual differences in pre-existing trait levels.

"Learning" Curves with Decay (Genetic Reaction Norms to Environmental Experiences)

So far, we have been discussing stylized examples of processes in which personality trait changes in response to environmental experience are either entirely random (set point theory and random walk) or entirely fixed (transition point). Our examples have also treated experience-

related changes in personality traits as either entirely ephemeral (set point theory) or entirely lasting (random walk and transition point). In reality, however, individuals may systematically differ in how their personality trait levels change in response to environmental experiences. Moreover, the durability of these changes may be intermediate along the continuum from short-lived to permanent, with individuals potentially differing from one another in trait change durability.

The panels in Figure 6 represent stylized versions of a very general pattern that has been observed in many different realms of psychological research, in which levels of a psychological, behavioral, or biological phenotype change (in this case, increase) systematically over time in response to the initial introduction of an environmental experience, after which point, levels recover to some degree, regressing partly back to their pre-exposure levels either as a result of adaptation to the previously novel situation, removal of the experiential stimulus, or some combination of the two. Lucas et al (2003), for instance, characterized individual differences in life satisfaction over the period leading up to and following marriage, in terms of a baseline phase, a reaction phase, and an adaptation phase. The overall trend generally resembles aggregate trends observed in Figure 6, in which life satisfaction increases leading up until marriage, and then partly regresses back to lower levels after marriage, with individual differences in both the reaction and adaptation changes. Lucas et al. (2007) surveyed several experiences that conform to this general pattern, including divorce, widowhood, unemployment, and disability (for which life satisfaction decreases during the reaction phase and then partly regresses upward to baseline levels during that adaptation phase), with the specific shape of the response pattern differing both as a function of the specific event under study, and individual differences. Another, very different example of the general pattern observed in the panels of Figure 6, is the salivary cortisol response to a challenging situation or stressor, such as the Trier Social Stress test. Cortisol is a stress hormone that is produced in response to psychological and physiological threats to homeostasis, and which has been postulated to influence behavior, including personality and psychopathology (Tucker-Drob et al., 2017). In stressful situations (such as public speaking under high pressure evaluative situations), cortisol levels increase substantially, and shortly after removal of the stressful situation, begin to return back to basal levels. Ram and Grimm (2007) modeled cortisol levels according to baseline, production/response, and dissipation phases, with the overall profile closely paralleling that

observed in Figure 6. They further documented individual differences in changes across each of the phase transitions. Finally, the general patterns observed in panels in Figure 6 are consistent with the widely known patterns observed in cognitive and educational psychology (Ebbinghaus, 1885), in which material is learned with continued studying, repetition, or practice over time, and is partly forgotten over time.

The top two panels of Figure 6 represent examples in which individuals differ from one another in their response to and recovery from an experience. In the upper left panel of Figure 6, individuals differ from one another in the magnitude of initial response to the experience. When between-person heterogeneity in response to environmental input varies as a systematic function of between-person genetic variation, this phenomenon is termed gene-by-environment interaction (G×E). Put differently, individual differences in development may emerge from a genetically influenced "norm of reaction" to the environment (Dobzhansky, 1955; Gottesman, 1963; Turkheimer & Gottesman, 1991). Using occupational stress as an example, the upper left panel of Figure 6 could represent increases in neurotic thinking and behaving following a harsh interaction with a supervisor. All individuals in this hypothetical example experience the same event, but some individuals react to the experience more dramatically than others. In the upper right panel of Figure 6, everyone initially responds identically to the experience, but some individuals are better able to cope and show greater recovery to baseline levels than others. In both of these situations, individuals follow different reaction norms due to genetic differences in sensitivity to the environment.

In conceptualizing the relations between differential response curves and lifespan behavioral genetic trends in personality, it is important to consider the nature of the relevant environments in question, and whether they are necessarily objectively shared by twins within a given pair. For instance, if the G×E is not explicitly modeled, a standard behavioral genetic model will attribute individual differences in response to an objectively shared family-level environment to the genetic factor (Purcell, 2002; Turkheimer, 2000). This is because, in the context of such a G×E, the shared environment will differentiate the phenotypes of children raised together as a function of their genetic relatedness. However, if individuals respond differentially to an objectively nonshared environment, and the G×E is not explicitly modeled, a standard behavioral genetic model will attribute this interaction to the nonshared environmental factor (Purcell, 2002; Turkheimer, 2000). This is because when individuals differentially respond

to different experiences as a function of genotype, their phenotypes will become differentiated, even if they share the same genotype.

More concretely, let's assume that the effect of parental warmth on child agreeableness differs across children on the basis of their genotypes: some children become more agreeable when treated warmly by their parents (as we might intuitively expect), whereas others respond to parental warmth by becoming less agreeable (perhaps they feel smothered). Because these responses are associated with genetic differences between people, identical twins (who share all of their DNA) will respond more similarly to warm parents than will fraternal twins (who share only 50% of DNA that varies within humans). As heritability is estimated as the extent to which more genetically similar individuals are more similar on the phenotype, this gene-by-shared environment interaction will be reflected in the heritability estimate for agreeableness. Now, let's complicate this example by no longer assuming that parents have a set level of warmth that is applied equally across siblings. Parents can play favorites. This elaboration moves in the direction of gene-by-nonshared environment interaction. Again, children may respond differently to levels of warmth based on their genetically influenced levels of agreeableness. To make things easier, we can consider two types of parental warmth applied to siblings, consistent across siblings and inconsistent. Here, because we are focusing on the nonshared aspect of the interaction, it is most relevant to consider the inconsistent parenting type. Identical twins would respond similarly if they received similar parenting, but if parenting differs across identical twins, this may further exaggerate dissimilarity because the difference in environmental treatment is magnified by the genetically influenced difference in response. When differences between identical twins are maintained or magnified, the result is nonshared environmental variance.

At first glance, this sort of logic may seem a bit strange. Why can we make such broad statements about psychological development just by looking at similarity among twins? Behavior geneticists are trained to think in terms of sibling comparisons or other family-based contrasts, whereas typical psychological scientists are trained to think about how individuals or groups behave. The same sorts of mechanisms described above are certainly at work outside of twin pairs; the rationale can be drawn out for single individuals. Assuming complete knowledge of the genetic architecture of agreeableness, researchers would be able to create agreeableness profiles for individuals based solely on their DNA sequence. Then, one could test whether individuals

that score a standard deviation above the mean on this genetic-agreeableness scale respond more positively to parental warmth. This sort of interaction and study design is common and the logic straightforward. Twin and family based studies use a slightly more complicated rationale to get to the same inferential end point.

Ideally, a researcher would be interested in comprehensively modelling all these sorts of interactions using measured variables, so as to produce an accurate representation of genetic and environmental influence across all observed levels of all moderators. In reality, however, if G×E interactions occur pervasively, in response to daily interactions with a nearly infinite range of different environmental experiences, comprehensively modelling all possible G×E interactions may be an unobtainable goal. Indeed, the capability of behavioral genetic modeling to represent latent variance components representing the total aggregation of all genetic and environmental effects may be viewed as an advantage, so long as the variance components are properly interpreted.

An additional consideration concerns the durability of the interaction effect on behavior. Trait levels may respond instantaneously to an environmental experience, but quickly and entirely return to the origin point (as in the genetic set-point model). Alternatively, some portion of the environmental effect (and the differential response) may persist across time. Returning to our earlier example of parental warmth and child agreeableness, durability of the effect entails whether warm parenting instills a lasting sense of positivity and friendliness or fades away as soon as the child encounters a stressful social experience in school. The durability of the genetically-differentiated response may also differ as a function of age (e.g., Fraley & Roberts, 2005) and history of previous experiences. Thus, many of the same key issues surrounding the action of environmental main effects also apply to G×E interactions. If interactions between genetic factors and unique life experiences have lasting effects, these could serve as the basis for the increasing nonshared environmentality and increasing contribution of the nonshared environment to stability with age. However, to our knowledge, such interactions have yet to be consistently tested. Thus the roles of G×E interaction in lifespan trends in personality development are therefore currently unclear and in need of further investigation.

The magnitude and timescale of the environmental exposure is also important to consider. If the stylized curves depicted in the top panels of Figure 6 are taken as representing reactions to major life changes with consequences for long-term quality of life (e.g. marriage,

divorce, job loss), it is plausible for their effects on personality to be appreciable and lasting. However, if the curves are taken as representing reactions to a single seemingly trivial experience (e.g. a negative interaction at the office), the psychological reaction may likely receive the label of "state" rather than "trait" as in our Figure 6. Indeed, personality trait development is conventionally thought to be slow, gradual, and not due to "one-off" environmental exposures. However, it is important to keep in mind that the lifespan is composed of small units of time that sum to the whole. It may be short-term psychological reactions to seemingly trivial experiences, when taken in aggregate, provide the foundation for lasting personality change (Roberts & Jackson, 2008; Wrzus & Roberts, 2016). Indeed, this basic principle can be clearly observed in the work on multitrial learning dating back to the late 19th century (Ebbinghaus, 1885). As displayed in Figure 7, information retention after single initial learning trial quickly fades over time, with ultimately little appreciable effect. However, with each successive trial, the rate of decay becomes shallower, such that after a sufficiently large number of trials, information is retained at near maximum for very long periods of time. We suspect that this pattern may not only apply in cognitive and educational contexts, but to a broad array of contexts in which behavior repertoire (i.e. personality) is affected by environmental experience. Moreover, as we discuss next, individually varying endogenous factors may guide the timing and duration of personality-relevant environmental exposures, such that exposures differentially aggregate across individuals over time.

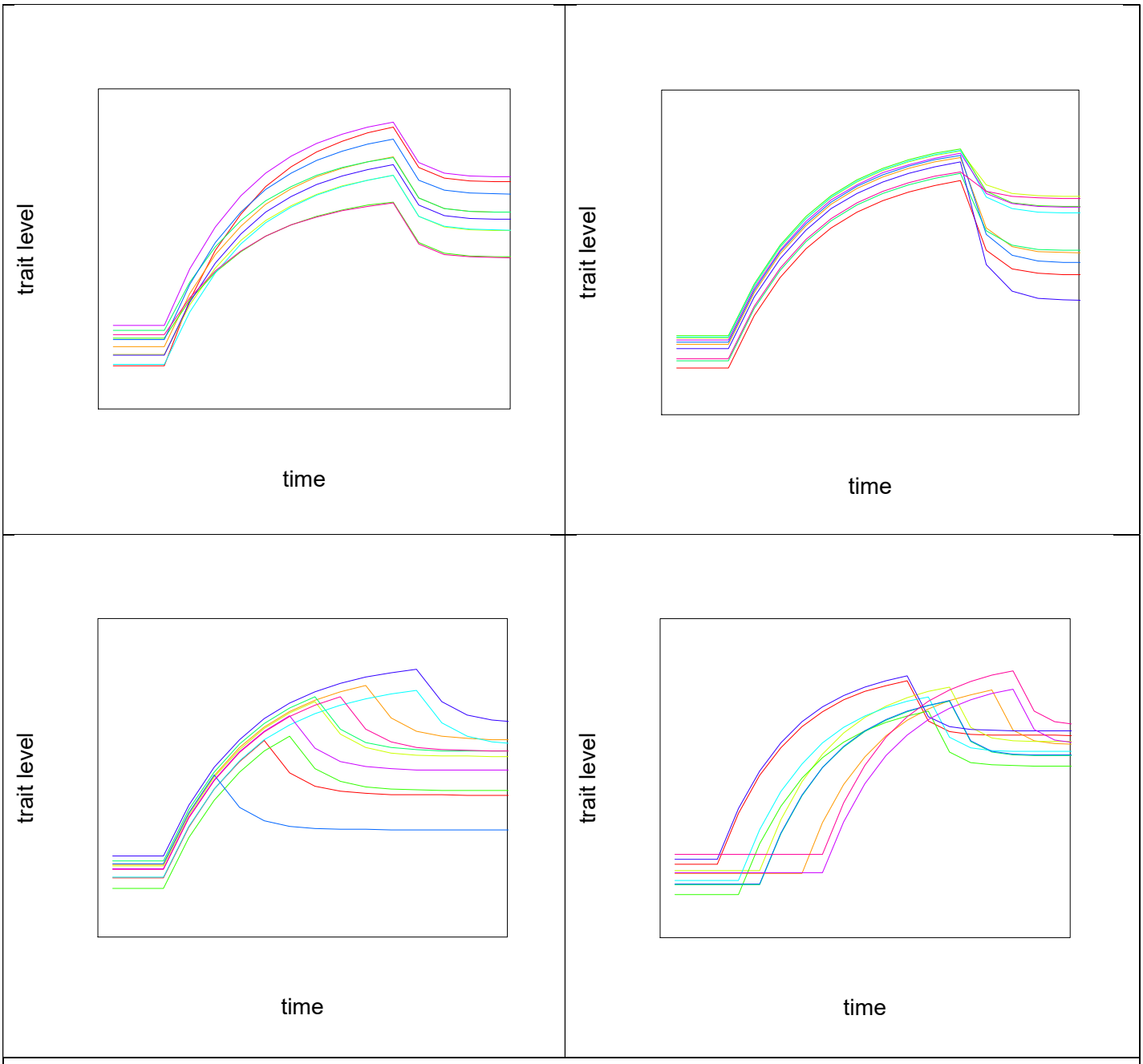

Figure 6. (Genetically-influenced) individual differences in response to experience, and post experience decay/acclimation. Upper left panel: Individual differences in response to experience. Decay parameters held constant. **Upper right panel:** Individual differences in and post-experience decay. Response parameters held constant. **Lower left panel:** Individual Differences in timing of experience offset. Learning and decay parameters held constant. **Lower right panel:** Individual differences in timing of experience onset. Learning and decay parameters held constant. In all panels, we allow for pre-existing individual differences in trait levels.

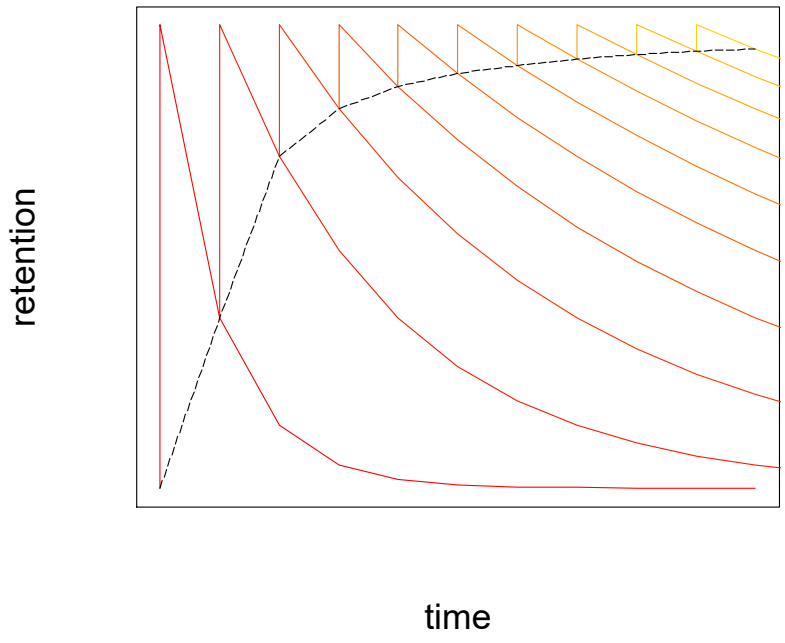

Figure 7. Ebbinghaus (1885) learning and decay curve over repeated trials of exposure to a learning experience. Retention decays post exposure, but overall memory trace increases with repeated exposures such that the rate of decay becomes shallower with increasing repetitions of exposure. The dashed line represents retention after the constant interval between exposures, after which the subsequent repetition commences. Colors represent different trials of exposure for a single individual.

Selecting and Sorting Experiences (Gene-Environment Correlations)

Gene-environment correlation (rGE) refers to the tendency for individuals to non-randomly encounter environmental experiences as a systematic function of their genetically-influenced characteristics (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983).

Evidence for rGE comes from subjecting measures of the environment to behavioral genetic models, as if they were phenotypes. For instance, just as a behavioral genetic model of personality infers heritability from determining whether (holding shared rearing experience constant) more genetically related individuals tend to be more similar in their personality than less related individuals, rGE can be inferred from determining whether more genetically related individuals tend to experience more similar environments than less genetically related individuals. Active rGE occur when individuals actively select their experiences on the basis of their genetically influences interests, proclivities, personality, and aptitudes. Evocative rGE occurs when individuals are selected into different environments on the basis of others' observations of their genetically influenced traits. For example, active rGE occurs when individuals interested in pursuing ideas have greater motivation to pursue postsecondary education, and evocative rGE occurs when colleges select individuals for admission on the basis of their high school performance (which is itself influenced by traits such as, organization, self-discipline, and intelligence).

Putting these textbook definitions of rGE aside, a vivid experimental demonstration of rGE can be found in the VH1 show Twinning. The conceit of the show is to take a group of identical twins and separate each pair into separate, mirror image houses. The show is a behavior geneticist's dream: place incredibly complex human beings into a social situation to see what happens; then do it again with genetically identical individuals with no knowledge of what happened. Similarities in behavior abound. The somewhat nerdy twins both fall into socially awkward positions of trying to figure out if boys and girls are sleeping throughout the house or are segregating by gender. Subsequently, a different twin pair independently bullies each of the nerdy twins. The muscle-bound twins each notice the gym equipment immediately upon entering the house and comment identically. Most interestingly, peer groups form based on the same group members while performing the same behaviors. In one house, four men take turns playing pool and commenting on how crazy the situation is. One twin hypothesizes that their co-twins are probably having the same conversation. Cut to the other house where, as predicted, the co-twins of the four men are all standing around the pool table discussing their co-twins. This is rGE in action. The somewhat diminished social graces of the nerdy twin pair evokes a similar response from the environment in the form of bullying. Meanwhile, other twins actively form similar peer groups likely because each individual looks for somewhat similar qualities and

attributes in potential friends. Of course, some video editing magic may have enhanced these similarities.

Returning to empirical work with somewhat larger sample sizes, rGE for important environmental experiences, such as stressful life events and social support, appears to be widespread; genetic factors have been estimated to account for a relatively large portion of variation in these experiences (roughly 30%; Kendler & Baker, 2007). The empirical implications of this finding for personality development are displayed graphically in the bottom two panels of Figure 6. In the lower left panel, all individuals encounter an environmental experience at the same point in time, but some individuals leave the environment more quickly. In the context of the harsh supervisor example, the lower left panel of Figure 6 predicts that everyone will get in an argument at some point in time, but that some individuals are able to resolve the disagreement more quickly than others. These more conciliatory workers may therefore experience a less steep increase in neurotic behaving and thinking in response to the experience. In the lower right panel, individuals differ in the timing of the event. Some workers may be more pleasant or diligent in their work, pushing back a negative encounter with their supervisor for a longer period of time. Again, this process means that measures of personality capture a cross-section of people at different points of the dynamic interplay between genetic influences, environmental experiences, and the mutual control of the expression of one over the other. If genetically influenced traits guide the timing, duration, and frequency of small neuroticism-inducing experiences on an everyday basis, these individually-trivial encounters may aggregate over time to have lasting effects on personality.

Gene-environment correlations have potentially widespread theoretical implications for personality development following environmental exposures and life events. As Lüdtke, Roberts, Trautwein, and Nagy (2011, p. 622) have written, "life events were originally construed as random—a position that has long been abandoned given that putatively random life events are both heritable and partially explained by personality traits." To the extent that life events are heritable and have causal effects on personality development, this process should lead more genetically related individuals to be more psychologically similar than less genetically related individuals. Put differently, genetic effects on personality may not exclusively flow through purely inside-the-skin biological pathways, but may also occur through outside-the-skin environmental pathways. For example, if bullying or peer groups have some causal effect on

personality development, then the results of VH1's experiment with Twinning indicates that such rGE may not just be a consequence, but a mechanism of genetic influences on personality. Genetic effects on personality may be environmentally-mediated. More generally, evidence of gene-environment correlation should temper strong interpretations of heritability as supporting a purely biological model of personality development.

The existence of rGE implies that individuals tend to select or evoke environments on the basis of genetically influenced characteristics, such that the effects of environmental experience magnify the initially small genetic influences (Briley & Tucker-Drob, 2013; Dickens & Flynn, 2001). If gene-environment correlation guides personality development, then one would expect increases in heritability and that phenotypic stability would be increasingly mediated by genetic variance components. However, the lifespan behavioral genetic trends highlighted in Figures 1 and 2 are inconsistent with this prediction. It may be the case that, absent rGE genetic effects on personality would fade even more precipitously with age, and that rGE largely maintains, but does not strengthen, genetic influences on personality. It is also possible that rGE processes operate in conjunction with other processes, e.g. random walk processes, such that any magnification of genetic influences that are produced are diluted by variation that arises over development through nongenetic pathways.

Putting it all together

In the previous sections, we pulled apart many different mechanisms of personality development to identify their unique components individually. However, many of these different mechanisms may simultaneously act to shape personality development. Rather than seeking to identify the "true" or "more correct" mechanisms of personality development, we believe that ongoing work in personality development research will do well to empirically delineate the circumstances under which aspects of each of the aforementioned are most relevant. This involves identifying specific measurable experiences that are relevant for personality change, measuring them intensively and repeatedly over time, and charting how they dynamically relate to the development of different personality traits. In this final section, we outline some important unanswered questions and reflect on why the questions have remained unanswered throughout the history of personality psychology.

Personality psychology has undergone several historical transitions, from periods in the early twentieth century during which personality was viewed as fixed and highly relevant to an individual's behavioral interactions and social and economic standing, to periods in the 1970's during which it was viewed as impotent and easily overwhelmed by the demands of situational contexts. In the 1990's the pendulum largely swung back, and personality was again seen as a relatively stable, enduring feature of an individual's psychology that was largely untouched by the external world. This aspect of personality was particularly useful from a persuasion perspective, because it allowed personality researchers to argue in favor of causality. Under this thinking, if personality was correlated with some outcome, it must be the case that personality is the cause. However, as personality psychology has again come to be accepted within the mainstream, current personality psychology may be going through some of the pains of a shift in thinking on these issues. For example, growing evidence is emerging that cross-cultural differences in social roles shape trajectories of personality development. For instance, Bleidorn et al. (2013) reported that in cultures where people tend to take on occupational roles at an earlier age, the normative age trends in conscientiousness and neuroticism are accelerated relative to cultures in which social roles are adopted later. These broadband culture effects may act on individuals by way of day-to-day social influences that aggregate over time. Other work has identified more specific social pressures, such as joining the military (Jackson, Thoemmes, Jonkmann, Lüdtke, & Trautwein, 2012) and preparing for transition to postsecondary education (Bleidorn, 2012). These studies have been instrumental in establishing that enduring changes in social roles and environmental contexts can have appreciable effects on personality development, presumably because they gradually and incrementally accrue over time. Recent work, however, suggests that personality changes may, in some cases, be more abrupt (as in Figure 5 above). Roberts et al. (2017) document large (d = .37) changes in personality development over a short period of time (4 weeks) in response to an intervention. These changes held in observational and experimental studies and lasted over long periods of time. The results raise the intriguing possibility that classic reports of slow, steady changes in personality over the entire lifespan (e.g., Roberts et al., 2006) may in fact smooth over exaggerated and discontinuous periods of personality change occurring at the individual level.

There are a number of central questions in personality development that remain unanswered. We view the following questions as most pertinent:

How do people engage the environment? The assortment of specific environmental experiences known to correlate with personality development is rather limited. Part of the issue may reflect our lack of knowledge concerning why people enter into certain environments, such as stressful life events. To what extents do specific life events occur at random, occur via social stratification, occur via active selection and evocation on the basis of genetically influenced factors, or occur as consequences of past personality development? Others factors that seem logically related to personality maturation, but typically go unmeasured, include details on the intensity, frequency, duration, timing, and consistency of exposures across time. One of the best examples of work toward answering these questions comes from Kandler, Bleidorn, Riemann, Angleitner, and Spinath (2012). The study was a genetically informative, three wave longitudinal study measuring both personality and life events. This design allowed for disentangling the extent to which individuals selected into environments on the basis of genetically influenced characteristics and whether the life events, in turn, had effects on personality development. Further, the authors distinguished between controllable and uncontrollable life events, positive and negative life events, and measured the intensity of the experience. However, the temporal resolution of the design, roughly 5 years between waves, leaves uncertain the specific engagement of the environment (e.g., would the effect have been different if the participant was a year younger? Or if the participant was fired from work before or after having a child?).

What is the shape and time course of environmental effects on personality? Studies of personality tend to focus on enduring effects. For example, a strength of the 5-year interval between assessments in the Kandler et al. (2012) study is that associations between a life event and personality are known to endure over a long period of time. However, it may be that after a five year interval, the effect of life events on personality has already decayed somewhat, and may be near a lower asymptote. Shorter interval, higher frequency, longitudinal approaches may offer opportunities for mapping dynamic changes of the sort represented by the stylized reaction curves in Figures 6 and 7.

Do individuals differ in their response to the environment on the basis of genotype? A defining feature of modern conceptions of personality is that some dimensions confer differential response to the same environment (Denissen & Penke, 2008). An environment that is stimulating to someone high on extraversion may be overwhelming to someone low on extraversion. Given this, it is surprising that there are relatively few examples of studies examining G×E interaction

in the development of personality. Some evidence comes from Krueger, South, Johnson, and Iacono (2008) who demonstrate shifts in heritability and environmentality across different levels of parenting variables. However, this finding has not been replicated to the best of our knowledge, and follow-up work for other sorts of environmental variables is lacking. One explanation is that researchers are looking in the wrong place for such interactions. We highlighted two ways that genotype could interact with environmental experiences: by moderating the initial response to the environment or by moderating the recovery from the response (Figure 6, top two panels). Some teens may respond well to instances of parental control whereas the same experience may lead others to rebel. Or perhaps all teens respond initially to the enforcement of parental control and reduce risk taking, but for some this effect is only short lived while setting others on the straight and narrow path. Ultimately, if these sorts of parent-child interactions are stable and recurrent, then one might expect this phenomenon to accumulate in systematic G×E interaction measurable using aggregate measures of parenting and adolescent behavior, thus partially obviating the need to measure specific instances of parent-child interaction. That said, much may be learned from the measurement of differential responsivity to specific episodes of experience, such as whether and how individual responses aggregate over time.

How does personality development build on itself? Moving beyond specific behavioral instances, it will also be important to accumulate information about how previous developmental history might also impact the response or decay of environmental effects. More generally, it may not be necessary to delineate how differential responses relates to both genotype (as in the previous question) and environmental history. It is possible that differential response is best accounted for by individual differences in the observable personality phenotype, such that identifying separate sources of variance may not be necessary (Turkheimer et al., 2014). Of course, whether developmental links between personality, genetic variation, and environmental variation occurs through a unified (i.e., phenotypic) path or a specialized (i.e., showing preference for genetic or environmental processes) path is an empirical question yet to be sufficiently addressed.

Several existing personality theories may provide useful conceptual frames for ongoing empirical research to address these questions. For example, the sociogenomic model of personality (Roberts & Jackson, 2008) articulates how small scale changes in states, such as

those depicted in Figure 6, can accumulate into personality development. Similarly, Whole Trait Theory (Fleeson & Jayawickreme, 2015) is premised on the idea that individual differences in personality constitute differences in density distributions of behaviors that are elicited from continuously changing situations. Finally, the network perspective on personality (Cramer et al., 2012) moves away from the latent trait concept in favor of personality dimensions as systems of discrete, causally interconnected behaviors. Each model has it strengths. The sociogenomic model highlights ways in which genes and environments are mutually interdependent; Whole Trait Theory draws on the most compelling work on short term fluctuations in behavior (e.g., Fleeson, 2004; Fleeson & Gallagher, 2009); the network perspective refines the locus of personality development away from the latent trait toward specific, narrow aspects of personality which more accurately reflect how the environment affects behavioral development (e.g., is it more plausible that getting a promotion at work activates general conscientiousness, including whether or not one's desk is clean, or more specifically achievement striving?).

In conclusion, we anticipate a bright future for genetically informative research in personality. We believe that we have the tools necessary to make substantial progress on how genetic and experiential inputs combine and interact over development to affect individual differences in repertoires of thinking, feeling, and behaving.

Acknowledgements

During the time of writing this chapter, Tucker-Drob's research was supported by National Institutes of Health (NIH) grants R01HD083613 and R21HD081437. Tucker-Drob is a member of the Population Research Center at the University of Texas at Austin, which is supported by NIH grant R24HD042849. During the time of writing this chapter, Briley's research was supported by a grant from the John Templeton Foundation (JTF58792).

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