How Do Individual Experiences Aggregate to Shape Personality Development?

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Abstract

Baumert et al. (this issue) have articulated a model of personality development in which chronic, recurrent, and consistent exposures to environmental experience induce enduring changes in personality. Here, I describe how genetic variation may play a central role in the aggregation of experiential effects on personality. First, genetic factors may affect individual responses to experience. Second, genetic factors may drive the type, frequency, consistency, and repetition of exposures to trait-relevant experiences. Both mechanism are expected to result in the differentiation of trait levels by genotype by way of experience.
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Thoughts, feelings, and behaviors fluctuate over time and change in response to experiences. When experiences are discrete, short-lived and isolated, personality traits often return back to pre-existing levels. However, when situational experiences are chronic, recurrent, or consistent in valence across longer periods of time, changes in personality may be more lasting. Short-term variability, individual responses and adaptations to discrete experiences, chronic situational conditions, and stochastic processes aggregate to produce macro-level patterns of relatively stable inter-individual differences and long-term developmental changes in personality. Thus, personality is influenced by an interdependent set of processes occurring at multiple timescales and within multiple embedded systems.

The above account is consistent with own current working view of the complex process of personality development, and in my reading of the target article, this view is largely shared by Baumert et al. (this issue). One major emphasis of the target article is the question of whether the covariance structure of individual differences in personality results from broad psychological factors that reside within individuals, or from the ways in which the sociocontextual and interpersonal pressures external to individuals are themselves organized. This is a theoretically hefty question that has vexed differential psychologists for a long while (Dickens, 2007; Cramer et al., 2012; Cronbach & Meehl, 1955; Thomson, 1916). However, with no intention of de-emphasizing the import of this question, I focus my attention here on what I believe is an equally important question: How do individual experiences aggregate to shape personality development? I suggest that endogenous genetic factors play a particularly central role in the process of experience-dependent personality change. In the remainder of this brief commentary, I discuss several ways that personality psychologists might conceptualize the symbiotic role of genetics and experience in personality development (for more comprehensive discussions of these ideas, see Briley & Tucker-Drob, 2014, Briley & Tucker-Drob, 2017, Tucker-Drob, in press; and Tucker-Drob & Briley, in press).
Perhaps the most classical conception of the role of genotype in personality variation is as a set point (Conley, 1984). This view holds that constitutional differences between individuals are the primary determinant of individual differences in personality. According to this view, experiential effects on personality are small and short-lived. Discrete experiences are thought to have ephemeral effects on personality, and individuals are expected to adapt relatively quickly to chronic situational conditions. Under a moving set point model (Costa & McCrae, 2006), age-related trends in personality represent developmentally programmed changes in endogenous equilibrium levels of personality traits. As neither a genetic set point nor a moving set point model allows for meaningful effects of experience on personality, I believe that these models are by themselves inadequate to account for accumulating empirical evidence for experiential effects on personality (Bleidorn, Hopwood, & Lucas, 2016).

A more sophisticated view of the role of genotype in personality variation is that of a reaction norm (Figure 1, top panel). Whereas the genetic set point perspective treats experiences as impotent and ephemeral, a reaction norm view (Dobzhansky, 1955; Gottesman, 1963) allows for highly potent and lasting experiential effects on personality. Key, however, to the reaction norm concept is that genetic differences between people relate to the magnitude of personality change in response to the experience, both in the immediate term, and during the process of acclimation and fade-out (Tucker-Drob & Briley, in press). In this context, genetic influences on personality traits occur by way of an interaction between individuals and their environments: Environmental experiences cause changes in trait levels and genotypes affect the magnitude of the experience-driven change. Interestingly, some authors (e.g. Denissen & Penke, 2008; Nettle, 2009) have suggested that personality traits themselves are best conceptualized as reaction norms.

Finally, although much theoretical work in personality development has focused on the role that individuals play selecting, evoking, and creating their experiences (Buss, 1987; Caspi, Roberts, & Shiner, 2005), there has been less of an appreciation within personality psychology for the role of genetics in such processes. When genetically-influenced motivations, aptitudes, and proclivities lead individuals to expose themselves to different sorts of trait-relevant experiences at different levels of frequency, duration, and
intensity, this can lead to genetic influences on the traits themselves by way of environmental experience (Figure 1, middle panel; Bouchard, 1997; Hayes, 1962; Johnson, 2010; Scarr & McCarntey, 1983).

Baumert et al. argue that, with the exception of rarer traumatic or otherwise transformative experiences, “mostly, development is triggered and perpetuated by repeated experiences and enduring changes in those patterns.” In other words, individual effects of discrete experiences typically fade over time, and it is the repeated exposure to the same or similarly valenced experiences that builds up temporally robust changes in personality. What, then, causes environments to be consistently and repeatedly experienced to shape personality on a lasting basis? Chance, happenstance, or arbitrary experiences may certainly shift the assortment experiences to which an individual is later exposed. Here I have described two additional mechanisms that may be particularly relevant to the aggregation of experiential effects on personality. First, genetic factors may affect individual responses to the experience. Second, genetically driven “tastes” and “appetites” (Rimfeld et al., 2016) for particular classes of experiences may drive the consistent, repeated, selection and evocation of experiences. In a sense, then, genetic factors may act as set points, not for trait levels themselves, but for equilibrium levels of the types, quantity, duration, and consistency of experiences that an individual selects and evokes. I believe that both mechanisms are likely at play: genetic factors affect individual response to the experiences to which they are differentially exposed on the basis of those same genetic factors. The combined result of these mechanisms is the differentiation of trait levels by genotype (Figure 1, bottom panel).
References

Baumert et al. (this issue). Integrating personality structure, personality process, and personality development. European Journal of Personality.


Figure 1. Three stylized representations of individual differences in engagement with trait-increasing experiences. Each colored line represents the trajectory for a given individual. In all three panels, an individual’s trait level increases upon exposure to an experience and then begins to return to pre-exposure levels after the offset of the experience. The top panel represents a reaction norm scenario. All individuals experience the same frequency and duration of exposure but differ with respect to the rate at which their trait levels increase in response to each experience, and the rate at which their trait levels return to baseline after experience offset. The middle panel represents a transactional, gene-environment correlation, scenario. All individuals react equivalently to each experience, and their trait levels decay at equivalent rates post offset. However, individuals differ in the frequency at which they select and evoke experiences and in the duration of exposure to each experience. The bottom panel represents a combined scenario in which individuals vary in both in their response to each experience and in their pace of exposure to experience. When genetic variation is related to the magnitude of response, the pace of exposure, or both, this translates to genetic influences on the traits themselves.