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Integrating the study of personality and psychopathology in the context of gene-environment correlations across development

Samantha Perlstein | **Rebecca Waller**

Department of Psychology, University of Pennsylvania, Philadelphia, PA, USA

Correspondence
Rebecca Waller, Department of Psychology, University of Pennsylvania, 425 S University Ave, Philadelphia, PA 19146, USA.
Email: rwaller@sas.upenn.edu

Abstract

Objective: A key principle of individual differences research is that biological and environmental factors jointly influence personality and psychopathology. Genes and environments interact to influence the emergence and stability of both normal and abnormal behavior (i.e., genetic predisposition, *X*, is exacerbated or buffered under environmental conditions, *Y*, or vice versa), including by shaping the neural circuits underpinning behavior. The interplay of genes and environments is also reflected in various ways in which they are correlated (i.e., *rGE*). That is, the same genetic factors that give rise to personality or psychopathology also shape that person's environment.

Methods: In this review, we outline passive, evocative, and active *rGE* processes and review the findings of studies that have addressed *rGE* in relation to understanding individual differences in personality and psychopathology across development.

Results: Throughout, we evaluate the question of whether it is possible, not only to differentiate the person from their problems, but also to differentiate *the person from their problems and their environment*.

Conclusions: We provide recommendations for future research to model *rGE* and better inform our ability to study personality and psychopathology, while separating the influence of the environment.

KEYWORDS

environment, genetics, personality, psychopathology, taxonomy

1 | OVERVIEW

Research has traditionally separated the study of personality (i.e., typical ways of thinking, feeling, and behaving) and psychopathology (i.e., behavioral and psychological dysfunction associated with mental illness) (Stein et al., 2010; Widiger, 2011). Structured trait-dimensional models, such as the Five-Factor Model (FFM; McCrae & Costa Jr., 1997), are the gold standard for personality research, whereas psychopathology is typically represented within categorical taxonomies, including within the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013). Recent research suggests that personality and psychopathology share common aetiologies and operate within a bidirectional pathoplastic relationship (Rosenström et al., 2019). Increasingly, psychopathology is being conceptualized dimensionally (De Fruyt et al., 2017; Trull & Widiger, 2013), including within the Research Domain Criteria (RDoC; Cuthbert, 2014) and Hierarchical Taxonomy of Psychopathology (HiTOP) framework (Kotov et al., 2018; Latzman & DeYoung, 2020). Critically, personality and psychopathology occur within an *environment* with specific physical (e.g., community or home), interpersonal (e.g., relationships with family or peers), and psychological (e.g.,

individual perceptions) attributes. In addition to addressing the question of whether personality can be distinguished from psychopathology (i.e., "can you separate a person from their problems?"), we evaluate here the question of whether a person's *environment* can be distinguished from their personality and psychopathology (i.e., "can you separate a person and their problems from their environment?").

2 | GENE BY ENVIRONMENT (GxE) INTERACTIONS

To disentangle personality from psychopathology, we must first contend with genetics, including the established moderate-to-large heritability estimates for many personality traits and psychiatric disorders (Docherty et al., 2016; Polderman et al., 2015). That is, personality and psychopathology arise from overlapping genetic influences, which interact with environmental inputs to produce individual differences (Kendler et al., 2011). Gene-by-Environment (GxE) interactions represent this interaction, with environmental effects on behavior modeled as contingent on genotype, or vice versa (Hyde et al., 2011). GxE interactions are implicated in the emergence and chronicity of both personality and psychopathology across the lifespan (Plomin, 2014). However, GxE interaction studies are limited because they require large samples to detect small effects and do not adequately address epistasis (i.e., effect of one gene is dependent on that of another), the developmental regulation of genes, or differential susceptibility (e.g., polymorphisms that are disadvantageous in some environments, but advantageous in others) (Hyde et al., 2011). Most critical is the fact that *genetic and environmental influences are not independent,* thus, violating basic statistical assumptions of interaction testing (Wahlsten, 1990). This phenomenon, known as gene-environment correlation (*rGE*), is the process through which genotype is associated with environmental input (Plomin, 2014).

3 | GENE-ENVIRONMENT CORRELATIONS (*rGE*)

Three commonly recognized forms of *rGE* exist (Figure 1). First, *passive rGE* is the nonrandom association between a person's genotype and their environment (Plomin, 2014). The genotype shared by parents and offspring "passively" accounts for observed correlations between the environment and the personality or psychopathology of offspring. For example, although classic research established correlations between child aggression and parental harsh discipline (Gershoff, 2002), findings are confounded by *passive rGE* processes, whereby a parent who uses harsh discipline may have transmitted a genetic liability for aggression to their child (Figure 1a). Likewise, in *evocative rGE*, personality or psychopathology elicits environmental inputs concomitant with genotype (Plomin, 2014). For example, a child with a difficult inherited temperament might inadvertently evoke more negative behavior from a parent who shares the same genotype (Figure 1b). Thus, genetically mediated, child-driven effects increase the likelihood of negative input from the environment, which further exacerbates risk for psychopathology (Knafo & Jaffee, 2013). Finally, *active rGE* processes reflect the propensity of an individual to select environments that are consistent with their genotype (Plomin, 2014) (Figure 1c). For example, children with genetic vulnerability for aggression affiliate with aggressive peers, increasing risk for an escalation in their own aggression (Van lier et al., 2007).

Before personality, psychopathology, and the environment can be disentangled, it is critical to accurately characterize and measure these different *rGE* processes. That is, because *rGE* implies that the same genes that give rise to psychological outcomes also give rise to specific environments, major confounds emerge from a sole focus on direct associations between environmental inputs and measures of personality or psychopathology (Knafo & Jaffee, 2013). Indeed, *rGE* processes explain why many environments associated with psychopathology, such as social support, family environment, divorce, and trauma exposure, appear to be moderately heritable (Kendler & Baker, 2007). Several study designs allow some separation of genetic and environmental effects, including adoption designs where children are raised by parents who are genetically unrelated to them, twin studies that exploit differential relatedness of monozygotic (MZ) and dizygotic (DZ) twin pairs, and candidate gene studies, genome-wide association studies (GWAS), or epigenome-wide association studies (EWAS) that establish correlations between genetic variation and personality, psychopathology, or the environment. Studies using these designs have established that passive, evocative, and active *rGE* processes influence personality and psychopathology across development (Knafo & Jaffee, 2013) and provide insight into how to separate a person and their problems from their environment, highlighting the value of this endeavor from both a research and translational perspective (Avinun, 2020).

4 | ADOPTION DESIGNS AND *rGE* PROCESSES

Adoption studies largely eliminate passive *rGE* processes when measuring associations between adoptive parents and children because they are genetically unrelated. Genetic main effects can be estimated by examining whether biological parent personality or psychopathology is associated with child

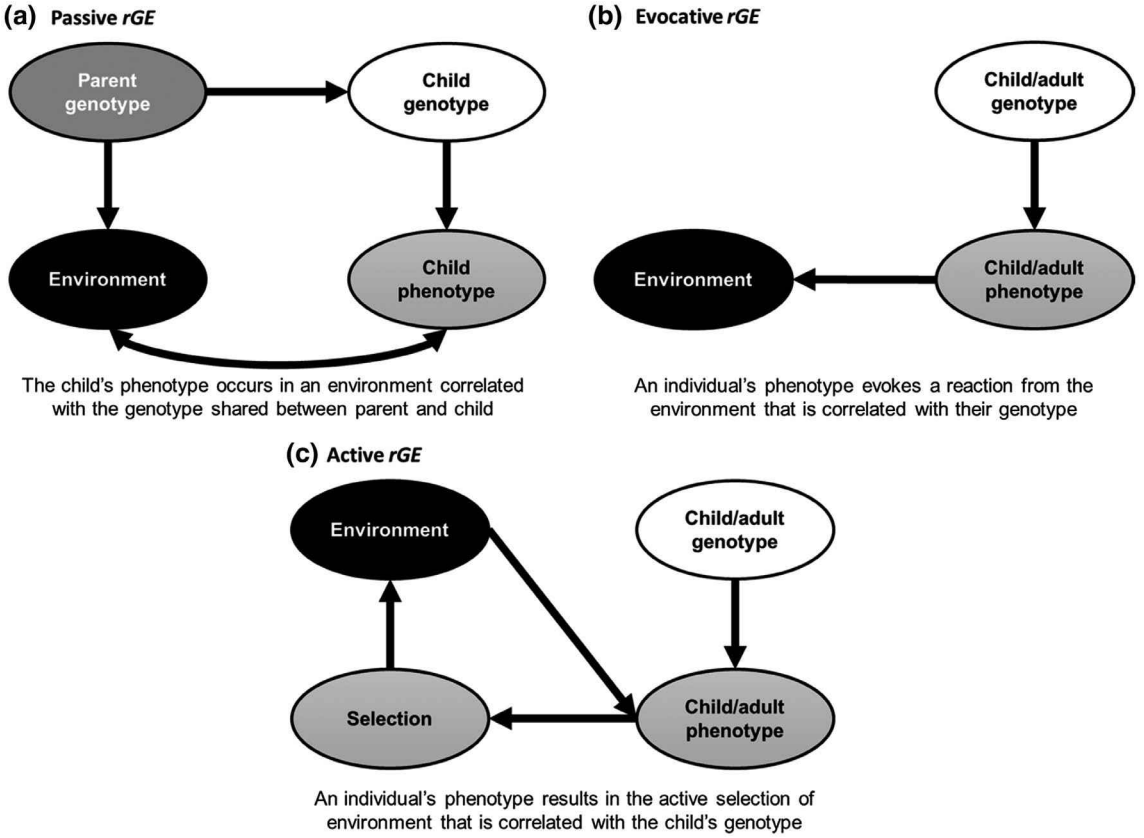

FIGURE 1 Three type of gene-environment correlations (*rGE*). (a) Passive *rGE* processes occur when shared genetic material between the parent (or another family member) and the child accounts for observed correlations between partially heritable traits and the child's environment. (b) Evocative *rGE* processes occur when genetically mediated traits elicit certain responses from others or experiences creating a nonrandom environmental exposure. (c) Active *rGE* processes occur when individuals select experiences consistent with their genotypes creating nonrandom environmental exposures

behavior. Any associations between adoptive parental traits, or the rearing environment they provide, and child behavior reflects either "true nonheritable" parenting effects or evocative *rGE* processes. The power of an adoption design for informing our understanding of *rGE* processes is exemplified by a wealth of published findings generated by The Early Growth and Development Study (EGDS), an ongoing study of 561 adopted children and their adoptive and biological parents. For example, biological parent antisocial behavior directly predicted increases in negative parenting by adoptive fathers of children aged 18–27 months, consistent with evocative *rGE* effects (Klahr et al., 2017). Moreover, biological mother symptoms of attention-deficit/hyperactivity disorder were related to more hostile parenting in adoptive mothers via greater impulsivity of adopted children (Harold et al., 2013). Finally, reciprocal associations were found between harsh parenting of adoptive parents and increases in adopted child callous-unemotional behaviors from 2.5 to 4.5 years old (i.e., lack of empathy and guilt), while callous-unemotional behaviors simultaneously led to increases in adoptive parental harshness over time (Trentacosta et al., 2019). In each of these studies, pathways between children's behavior and the rearing environment are not accounted for by shared genetic risk. Thus, each provides compelling evidence that genetic influences shape children's environments, including in ways that are concomitant with their emergent personality and psychopathology.

5 | TWIN STUDIES AND *rGE* PROCESSES

Twin studies also highlight how *rGE* processes lead to reciprocal associations between environment, personality, and psychopathology across the lifespan (Jaffee & Price, 2007; Scarr & McCartney, 1983). Classic twin designs leverage the fact MZ twins share nearly 100% of their genetics versus the 50% shared by DZ twins. Greater similarity within MZ twin pairs implies heritability, whereas differences between MZ twin pairs suggests shared and nonshared environmental influences (Jaffee, 2016). Nuclear twin family designs allow researchers to model genetically informative data within an extended circle of family members, including parents and non-twin siblings

(see Keller et al., 2009 for a review). Using these designs, twin studies have established that exposure to specific environments is heritable (Jaffee & Price, 2007). For example, exposure to parental warmth and harsh discipline in early childhood is influenced by passive and evocative *rGE* processes (Rutter & Silberg, 2002). Later in life, other environmental factors, including marriage (Johnson et al., 2004), divorce (Jocklin et al., 1996), and social support (Bergeman et al., 1990), are influenced by evocative and active *rGE* processes. Moreover, specific aspects of personality influence the relative heritability of different environments (Jaffee & Price, 2007; Saudino et al., 1997). For example, personality traits moderated the genetic and environmental influences on the quality of the parent–adolescent relationship, indexed via regard, conflict, and involvement (South et al., 2008). Among adult twin pairs, the relationship between personality traits and major life events, including controllable (e.g., marriage), desirable (e.g., meeting someone new), and undesirable (e.g., death of a child) events, was also accounted for by genetic influences, although only in women (Saudino et al., 1997). Finally, among adolescent twins, the heritability of parental negativity and negative life events was correlated with adolescent oppositionality, delinquency, aggression, depression, and anxiety (McAdams et al., 2013). That is, adolescents with more psychopathology either provoked or were subject to more negative environmental inputs. Together, these studies establish that personality and psychopathology are reciprocally related to the environment via both passive and evocative *rGE* processes.

Twin studies also provide support for active *rGE* processes. For example, in an extended twin family study that investigated individual differences in political interest and participation, shared environmental effects were stronger in adolescence, whereas genetic effects were stronger in early adulthood (Kornadt et al., 2018). That is, active *rGE* influences increased over time, with genetically mediated aspects of personality shaping the environment. In support of this finding, genetic effects on political orientation were not significant among 17-year-olds, but became significant by age 23, indicating a shift to active *rGE* processes across early adulthood (Hufer et al., 2020). This finding exemplifies a well-recognized phenomenon that the greater independence from parents that begins during early adulthood heralds a shift away from passive and evocative *rGE* influences toward active *rGE* processes (Scarr & McCartney, 1983). These changing influences are evident in greater heritability estimates with increasing age, including for prosocial behavior (Knafo & Plomin, 2006) and cognitive ability (Briley & Tucker-Drob, 2013). Overall, early genetic influences on personality are amplified when children inadvertently shape and actively choose their environments, thus, exposing them to specific inputs that reinforce the same underlying genetic predisposition for personality and psychopathology.

6 | MOLECULAR GENETICS STUDIES AND *rGE* PROCESSES

Since the advent of psychiatric genetics in the late 1980s and early 1990s, researchers have linked genetic variation to personality and psychopathology (Ginsburg et al., 1996; Plomin & McGuffin, 2003). Candidate gene approaches focus on single nucleotide polymorphisms (SNPs; variation in a single base pair) that are identified a priori. In contrast, genome-wide association studies (GWAS) analyze the entire genome for common genetic variation among groups of individuals defined by a given phenotype or disorder (Plomin, 2014). GWAS support the creation of polygenic risk scores (PRS) composed of multiple SNPs that are combined into a single measure of genetic predisposition (Plomin, 2013). The use of these approaches provides further insight into the correlations between personality, psychopathology, and environment.

First, *passive rGE* processes have been documented via candidate gene studies. In two samples of 3-year-olds, children with the short allele of the serotonin transporter gene (i.e., 5-HTTLPR) gene experienced more maternal hostility specifically when mothers reported experiencing abuse from their own mothers (Kopala-Sibley et al., 2017). Similarly, in a twin study of 3-year-old boys, variation in 5-HTTLPR was associated with lower child self-control, which in turn predicted less positive parenting from mothers (Pener-Tessler et al., 2013). In a study of 6-year-olds, children with a 9-repeat variant in the *DAT1* gene expressed more negative affect toward parents, which in turn was related to more hostile parenting and less engagement (Hayden et al., 2013). Finally, genetic variation in the oxytocin receptor gene among children was linked to harsh (Brody et al., 2017) and warm parenting (Feldman & Bakermans-Kranenburg, 2017; Feldman et al., 2012), as well as deviant peer affiliation (Fragkaki et al., 2019; Poore & Waldman, 2020). In sum, candidate gene studies are suggestive of genetically driven links between a person and their environment in ways that amplify risk for psychopathology.

Second, PRS studies based on GWAS provide further evidence for overlap between personality, psychopathology, and the environment. For example, among children assessed at ages 5–6 and 11–12, several PRS derived from GWAS of schizophrenia, major depressive disorder, Neuroticism, and well-being were associated with multiple risky environmental inputs, including passive *rGE* processes that began prior to birth (e.g., maternal prenatal anxiety, smoking, and alcohol use), and evocative *rGE* processes after birth (e.g., maternal distress) (Ensink et al., 2020). Using a PRS approach,

evocative *rGE* processes have also been identified later in childhood and adolescence. For example, among children assessed between ages 5–13 and again a year and a half later, PRS scores (derived from prior studies of genetic risk for impulsivity, sensation-seeking, reward dependence, and low behavioral inhibition) were related to child impulsivity in middle-childhood, which in turn predicted less parental monitoring in early adolescence, and ultimately greater affiliation with substance-using peers in middle adolescence (Elam et al., 2017). Finally, in a study of 12- to 17-year-olds, higher genetic risk for depressive symptoms indexed via a PRS score was related both to lower parental knowledge and less adolescent Agreeableness, which in turn predicted greater risk for major depressive and conduct disorder symptoms (Su et al., 2018).

Together, these PRS studies chart evolving *rGE* processes leading to developmental cascades where genetically driven child temperament and personality features influence environmental inputs, increasing risk for psychopathology. PRS studies also aggregate the effects of genetic variants to estimate heritability and infer genetic overlap between personality and psychopathology. However, PRS present new challenges to disentangling *rGE* processes. For example, PRS underestimate total genetic effects because they are limited to the additive effects of *common* variants that are discoverable only via adequately powered GWAS (Krapohl et al., 2017). To address this limitation researchers have called for more sophisticated methods to analyze GWAS data, including via functionally coherent subnetworks analysis (see Taşan et al., 2015 for a review of this method). With the growing revolution of "Big Data" in genetics research, we can better determine shared genetic influences on personality and psychopathology.

7 | SEPARATING PERSONALITY, PSYCHOPATHOLOGY, AND THE ENVIRONMENT

The evidence reviewed herein reflects the large body of research that has leveraged *rGE* processes to explore how shared genetic risk shapes a person's environment in ways that are concomitant with individual differences in their personality and psychopathology (Bleidorn et al., 2020; Wagner et al., 2020). However, fewer studies have truly separated the environment from personality and psychopathology in the context of known *rGE* confounds. In considering a roadmap for future studies to examine the potentially separable influence of the environment, while accounting for *rGE* processes, a number of caveats warrant consideration. First, extant literature does not rule out nonheritable influences of the environment, including truly independent environmental exposures that amplify risky pathways from personality to psychopathology (Knafo & Jaffee, 2013). For example, prospective associations between loneliness and suicidal ideation were exacerbated in the context of sexual assault (Chang et al., 2019). Likewise, higher disinhibition predicted substance use disorder in the context of poor neighborhood quality (Ridenour et al., 2009). Second, there are important parallels between the measurement of genes and environments with *polygenic* effects (i.e., many genes affecting the expression of a trait) echoed in *polyenvironmental* effects (i.e., many environmental factors affecting the expression of a trait) (Plomin, 2013). Relatedly, as evidenced through epistasis, environmental influences operate at multiple interacting levels, with the impact of some serving to buffer or exacerbate the effects of others. Third, genetic pleiotropy (i.e., variation in a single gene or PRS score affecting many traits) is echoed in the finding that variation in one or two specific environmental risk factors (e.g., maltreatment or abuse) have pervasive but highly varied effects on personality and psychopathology (Green et al., 2018). Finally, environments necessarily change across development (e.g., warm parenting differs for a 2-year-old vs. 15-year-old) (Barber et al., 2005) and between cultures (Claes et al., 2003). Notably, while there have been measurement advances within psychiatric genetics in the last few decades to assess genetic risk, there have been comparably fewer breakthroughs in novel characterizations of the "E" portion of GxE interactions or *rGE* processes (Plomin, 2013). With these caveats in mind, we offer a number of recommendations for future research to disentangle the person from their problems and their environment (Table 1).

7.1 | Evaluate the environment within experimental designs

Within traditional personality and psychopathology research, the majority of studies that have explored the environment have employed observational designs (Coolican, 2017). Even when these designs include prospective assessments over many years, they cannot determine causality, nor truly separate personality, psychopathology, or the environment. In contrast, experimental designs that enact quantifiable change to the environment facilitate firmer conclusions about cause and effect. Decades of research has already featured experimental manipulations of the environment in randomized controlled trials (RCTs) of behavioral interventions where specific aspects of an environment are changed in a treatment group relative to a no treatment, or treatment as usual control condition, allowing for strong conclusions to be drawn about environmental influences on personality or psychopathology (Barlow, 2014). For example, Multisystemic Therapy (MST) adopts a multifaceted approach to modify the family, home, and parenting environment, and has been

TABLE 1 Different approaches for future studies to better separate personality, psychopathology, and the environment

Approach	Description	Advantages	How best to leverage to separate a person, problems, and their environment
1. Randomized Controlled Trials (RCTs)	• Test effectiveness of behavioral or pharmacologic treatment on personality, psychopathology, and environment relative to a no treatment or treatment-as-usual condition	• Strong conclusions about cause and effect • Potential to isolate influence of the environment on personality or symptom changes	• Use **dynamic network models** to isolate individual mechanisms of change and model concomitant and temporal changes in nodes representing personality, psychopathology, and environment separately
1. Novel Measures of Environment	• **Dimensional models** capture environmental continuum by identifying dimensions that share common features across many inputs • **Environmental Taxonomies** catalog multilevel dimensions of environmental inputs (e.g., **DIAMOND or CAPTION**)	• Richer, more complex, multilevel measurement of the environment • Greater statistical power	• Within **multivariate twin models,** quantify shared versus unique genetic associations for specific environmental measures, personality traits, and psychopathology • Assess within **RCTs** pre, mid and posttreatment • Use **dynamic network modeling** to determine change pathways and isolate the specific environmental inputs that cause, and are caused by, changes in personality and psychopathology
1. HiTOP	• Dimensional classification system of personality and psychopathology • Overarching higher-order factor and categories underneath that gradually increase in specificity	• Emphasis on continuum between personality and psychopathology	• Test alongside **environmental taxonomies** to advance the development of an integrative model and classify distinct versus shared features of the environment, personality, and psychopathology • Use **genetically informed study** and **RCT designs** to further disentangle causal pathways, including genetically mediated effects
1. Epigenetics	• Gene expression is modulated with no change to underlying DNA sequence • Biochemical signature of environmental experience	• Indexes the interplay of genes and the environment • Strengthens causal conclusion about environmental inputs	• Incorporate **EWAS** into **twin study designs** to separate nonheritable environmental influences while accounting for genetic factors • Use **EWAS** within **RCT designs** and collect epigenetic markers pre and posttreatment to establish mediating effects of the environment on personality or symptoms

Abbreviations: CAPTION, complexity, adversity, positive valence, typicality, importance, humor, and negative valence; DIAMOND, duty, intellect, adversity, mating, positivity, negativity, deception, and sociality; EWAS, epigenome-wide association studies; HiTOP, hierarchical taxonomy of psychopathology; RCT, randomized control trail.

shown to effectively reduce conduct problems (Henggeler & Sheidow, 2012). Similarly, Family Focused Therapy (FFT; Falloon et al., 1985; Miklowitz & Goldstein, 1990) is an effective treatment for bipolar disorder that reduces expressed emotion (i.e., excessive criticism, hostility, or emotional overinvolvement of caregivers), teaches communication skills, and educates families on preexisting vulnerabilities that increase risk for symptoms (Miklowitz et al., 2009; Miklowitz & Chung, 2016).

Behavioral interventions have similarly been shown to produce changes in personality by targeting specific behavioral processes (Chapman et al., 2014; Magidson et al., 2014). For example, Behavioral Activation (BA) therapy teaches clients to change behavior to mold their own environment and create opportunities for personality change (Magidson et al., 2014). System-level interventions also bring about personality change, including when enacted at the neighborhood level or via early school and education programs (Chapman

et al., 2014; Heckman, 2006). RCTs of behavioral interventions can also determine whether environmental change, broadly defined, leads to change in personality *via reductions in symptoms of psychopathology*. For example, a meta-analysis of 207 studies established marked changes in personality following clinical intervention, with the largest effects for emotional stability and Extraversion (Roberts et al., 2017). In addition, individuals with internalizing disorders showed the most change in personality traits, although the type of treatment (e.g., cognitive-behavioral vs. pharmacological) was not differentially related to personality changes. Together, these behavioral interventions inherently address multiple *rGE* processes by targeting different portions of the environment to disrupt potentially risky pathways between environment, psychopathology, and personality.

Nevertheless, no studies to date have actually quantified the specific environmental mechanisms that contribute to personality or psychopathology change following behavioral intervention (Roberts et al., 2017). Addressing this question through careful assessment of the environment (i.e., home, relationships, and perceptions of daily experiences) can help us to differentiate between a person, their problems, and their environment and determine which treatments, for which individuals, and under which set of circumstances confers the largest reductions in symptoms of psychopathology or personality change. In particular, research is needed to establish concomitant "environmental change" that occurs following either psychotherapy or pharmacological treatment. One promising method to isolate mechanisms of change and structurally model the relationship between personality, psychopathology, and the environment within the context of an RCT involves the use of dynamic network models (Hofmann et al., 2020). These models estimate individual temporal and contemporaneous network structures to explore relationships between personality, psychopathology, and the environment over time (Epskamp et al., 2018). Variables of interest are represented as *nodes* and relationships between nodes are represented as *edges*, creating a network structure (Hofmann et al., 2020). Through the use of time series data, such models identify the within- and across-time relationships of different nodes (Hofmann et al., 2020). Dynamic network models can be a valuable tool for clinicians to conceptualize and plan treatments (Hofmann et al., 2020). Importantly, these models do not seek to simply overcome *rGE* processes, but actively use them to inform more effective treatments by establishing specific relationships between personality, psychopathology, and the environment.

7.2 | Leverage environmental dimensional models and taxonomies

A related method to improve our ability to separate reciprocal associations between personality, psychopathology, and environment, whether in an RCT or genetically informed study design, is to revolutionize how we measure the environment. The past several decades have heralded major advances in our ability to assess individual differences in personality and psychopathology, including through new technologies, including whole-genome and epigenome sequencing (De Fruyt et al., 2017; Plomin, 2013). However, methods to robustly assess variability in individuals' environments have relatively lagged behind (Oreg et al., 2020). A major challenge is that we have traditionally assessed only single or specific environmental inputs in relation to personality or psychopathology, and have failed to fully and simultaneously capture the environment of a person in all its complexity and richness (Cantor et al., 1982; Edwards & Templeton, 2005; Parrigon et al., 2017). For example, typical "specificity models" focus only on the effects of environmental exposures defined as if they are discrete, including physical and sexual abuse, neglect, parental death, parental divorce, or poverty. However, these models fail to fully capture lived experiences arising from supposedly single or discrete events (McLaughlin et al., 2020). For example, two children who both experience poverty may have varying lived experiences associated with different effects of poverty that might arise and interact differently across many levels, including family stress, parenting practices, neighborhood influences, and school quality (McLaughlin et al., 2020). Thus, we need to incorporate multilevel dimensional models and taxonomies to assess the environment.

One approach to modeling the environment involves the identification of underlying dimensions of environmental experience that share common features across numerous single environmental events (McLaughlin et al., 2020). For example, researchers have distinguished between the dimension of threat (e.g., experiences of direct harm or threat of harm to survival) and the dimension of deprivation (e.g., experiences involving the absence of expected inputs) (McLaughlin et al., 2014). These dimensions encompass different "single" environmental insults, including abuse, domestic violence, and food insecurity, with higher scores reflecting greater severity and chronicity in exposure to either threat or deprivation (McLaughlin et al., 2014). Evidence to support the utility of this approach comes from studies showing differential predictions of psychopathology by environmental experiences involving threat (e.g., posttraumatic stress disorder) versus deprivation (e.g., reactive attachment disorder) (King et al., 2019; McLaughlin et al., 2020). The benefit of this approach for characterizing the environment is that we can identify specific environmental dimensions implicated in personality and psychopathology, including by deriving "dose response curves," which can be used to inform treatment.

A second new approach to modeling the environment comes from efforts to develop environmental taxonomies. For example, Rauthmann et al. (2015) proposed eight dimensions

to characterize individual differences in the dynamic interactions between a person and their environment: Duty, Intellect, Adversity, Mating, Positivity, Negativity, Deception, and Sociality (DIAMONDS) (De Fruyt et al., 2017). Similarly, Parrigon and colleagues (2017) proposed the CAPTION model, an integrative taxonomy of psychological situation characteristics (Complexity, Adversity, Positive Valence, Typicality, Importance, Humor, and Negative Valence) (Parrigon et al., 2017). Strong convergence exists between the CAPTION and DIAMOND scales (Parrigon et al., 2017; Rauthmann & Sherman, 2018). The CAPTION dimensions have also been linked to various personality traits. For example, Complexity was related to Extraversion and Openness to Experience, while Adversity was related to Neuroticism and lower Agreeableness (Parrigon et al., 2017).

By including these dimensional measures (e.g., DIAMOND, CAPTION, threat, or deprivation) within genetically informed and experimental (i.e., RCT) studies of personality and psychopathology, future research can better separate a person and their problems from their environment. For example, via evocative and active *rGE* processes, individuals high on Extraversion or Openness to Experience may engage in more novel or risky behaviors (i.e., evocative and active *rGE*) that result in the experience of Complexity (Woo et al., 2014). Via passive *rGE* processes, individuals higher on Neuroticism may "inherit" difficult environments from their parents (Elam et al., 2017), and might also perceive and rate their environmental experiences more negatively (Oreg et al., 2020; Parrigon et al., 2017) (Table 1).

Multivariate twin models could further quantify the extent to which the associations between specific environmental inputs, personality traits, and psychopathology are accounted for by common genetic influences. Such models would be better powered through the use of dimensional measures of the environment (including derived dimensions of threat or deprivation or the DIAMOND and CAPTION dimensions) than prior approaches that relied on binary representations of the environment (Verhulst, 2017). For example, a longitudinal multivariate twin model could disentangle the environmental dimensions of threat and deprivation, early temperament, and later child psychopathology. In such a model, shared additive genetic paths would be estimated between twin scores measured over successive prospective assessments to decompose unique versus shared genetic variances of each construct, highlighting potential *rGE* processes. If the different environmental dimensions (i.e., threat vs. deprivation) evidenced shared genetic variance with different outcomes (e.g., anxiety vs. conduct problems), this would represent strong evidence for their discriminant and predictive validity. Likewise, if different aspects of temperament evidenced shared genetic variance with different environmental dimensions or outcomes, this would provide evidence for the unique contributions of each specific environmental, personality, and psychopathology factors, while simultaneously modeling *rGE* process. Such models could also decompose the *nonheritable variance* shared between environmental dimensions, personality, and psychopathology, thus, providing evidence for the ways in which personality or psychopathology shape the environment and vice versa.

Finally, within RCT designs, researchers could utilize rich taxonomies to characterize multiple dimensions of environmental experience to assess participants' environments pre- and postintervention. Through the use of dynamic network analysis researchers could be better positioned to not only isolate the specific dimensions of the environment that cause, or are caused by, changes in personality and psychopathology, but also at what level of that environmental dimension change occurs. Together, these approaches highlight that careful cataloging, or dimension reduction, of environmental experiences into an integrative taxonomies or underlying dimensions can advance our understanding of how personality and psychopathology are intertwined, while simultaneously determining which environmental targets to manipulate within novel treatments that maximize symptom reduction, accounting for *rGE* processes are considered.

7.3 | Utilize the HiTOP framework within genetically informed study designs

Recent research has also emphasized an integrative model of personality and psychopathology, with psychopathology conceptualized as an extreme on the same continuum as personality (Widiger, 2011), which has important implications for separating the environment from the person and their problems. This integrative model addresses concerns regarding traditional systems of diagnostic categorization (i.e., the DSM) that exhibit within-diagnosis heterogeneity, excessive comorbidity, and low diagnostic reliability (Kotov et al., 2017). Moreover, there is often high comorbidity between different forms of psychopathology, which has led to the characterization of a higher-order latent "*p* factor" that represents general psychopathology susceptibility that has not been well represented in traditional diagnostic classification systems (Caspi et al., 2014). To capture the full spectrum of personality and psychopathology, the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov et al., 2017) classification system includes an overarching higher-order latent factor (similar to the general *p* factor), with various categories underneath that gradually increase in specificity.

The HiTOP model was derived from quantitative nosology studies and has the potential to improve the reliability, validity, and utility of diagnostic classifications in research and clinical settings (Conway et al., 2019). Critically, the HiTOP model holds potential to advance research on the relationship between a person, their problems, and the environment.

For example, future studies could integrate the HiTOP model with environmental taxonomies, such as the DIAMOND or CAPTION, which would advance the development of an integrative model to chart pathways between specific dimensions of environment, personality, and psychopathology. This endeavor would be especially valuable if relationships between the HiTOP and environmental taxonomies were tested within genetically informed designs. Finally, studies using dynamic network models could explore and validate the HiTOP and/or genetically informed environmental taxonomies within the context of RCTs to further establish change processes that jointly consider a person, their problems, and their environment. This approach would allow researchers to derive greater specificity vis-a-vis modifiable and separable environmental mechanisms that result in improvements in mental illness and changes in personality over time (Table 1).

7.4 | Incorporate epigenetics into study designs

Finally, beyond the identification of genetic influences on the environment, research has begun to show that the environment directly shapes genetic expression. The study of epigenetics offers another opportunity to disentangle the environment from personality and psychopathology. Epigenetics is a collection of mechanisms whereby gene expression is modulated with no change to the underlying DNA sequence, representing a biochemical signature of environmental input (Meaney, 2010; Plomin, 2014; Rutter et al., 2006). Epigenetic processes have been documented in personality and psychopathology, including in attentional problems (Chen et al., 2018), susceptibility to stress (Hamilton et al., 2018), drug addiction (Mews et al., 2018), stress response (Chen et al., 2012; Prados et al., 2016), and risk taking (Kaminsky et al., 2008). Studies have also shown that environmentally induced epigenetic changes persist over a lifetime and, in some cases, are transmitted to subsequent generations (Bohacek et al., 2013). Thus, extant literature on epigenetics highlights the bidirectional interplay of genes and environment, which informs our understanding of *rGE* processes.

This interplay is exemplified in a recent theoretical framework of personality that specified interactions between environmental and genetic influences on personality, while accounting for *rGE* processes (Wagner et al., 2020). In this framework, genetic influences shape both personality and the environment via largely stable individual differences in gene expression, protein synthesis, morphological structures, and functioning of the nervous and endocrine systems (Wagner et al., 2020). Environment influences include both stable (e.g., cultural and social opportunities) and unstable (e.g., traumatic experiences) characteristics, which influence personality development, as well as neural responsivity, hormonal activity, and gene regulation and expression. Personality was theorized to influence the environment via characteristic patterns of behavior, which can increase the probability of exposure to specific environmental inputs (Wagner et al., 2020). Ultimately, by integrating this model with epigenetics, neuroimaging, and physiological methods, we will be able to separate environmental influences on personality and psychopathology mechanisms, while accounting for *rGE* processes and establishing mediating biological mechanisms.

Finally, to separate the environment from genetic influences, future studies could combine twin and epigenetic modeling (Bell & Saffery, 2012; Craig, 2013; Li, 2020). Epigenetic markers that are discordant between MZ twins represent a powerful indicator of nonheritable environmental influences. One approach to investigate discordance between twins is within epigenome-wide association studies (EWAS) (Craig, 2013; Li, 2020) (Michels et al., 2013). Studies have begun to explore DNA methylation (i.e., the biological process by which methyl groups are added to the DNA molecule altering gene expression) as an epigenetic marker using an EWAS approach in relation to psychopathology within twin designs, including ADHD in children (Walton et al., 2017) and adults (van Dongen et al., 2019), depression (Byrne et al., 2013), and schizophrenia (Dempster et al., 2011). The power of leveraging twin designs and epigenomic data holds significant promise to better identify biomarkers of true environmental influences that are not confounded by *rGE* processes (Bell & Saffery, 2012; Craig, 2013). At the same time, dynamic changes in epigenetic markers are adaptive and occur normally throughout development, making the interpretation of findings from EWAS challenging, and certainly more challenging than interpreting findings from GWAS (Michels et al., 2013). To address this challenge, future studies can incorporate EWAS techniques within experimental designs, including in the context of RCTs of psychological or behavioral interventions where epigenetic markers can be collected before and after treatment, and coupled with careful assessment of personality, psychopathology, and the environment (Kumsta, 2019). This approach would allow us to isolate the mechanisms of effective behavioral and psychological interventions that alter the environment, while simultaneously generating information about how genetic factors impact personality and psychopathology (Table 1).

8 | CONCLUSIONS

This review has highlighted that a person and their problems are not easily separable from each other, or the environment. Passive, evocative, and active *rGE* processes illustrate that the same genetic factors that give rise to personality or

psychopathology simultaneously shape a person's environment, often in ways that further elicit behaviors or environmental inputs that are consistent with those personality traits or that exacerbate pathways to psychopathology. However, we have outlined exciting new methodological advances that can address these challenges and ultimately can help to disentangle a person and their problems from their environment. For example, future studies can combine genetically informed designs, such as twin or adoption studies, with recent advances in GWAS and EWAS, dynamic network modeling within RCTs of treatments, or with taxonomies of the environment. Leveraging these approaches can generate a richer understanding of the interrelations of a person, their problems, and their environment, as well as helping to improve and personalize the targets of behavioral and psychological interventions.

ORCID

Samantha Perlstein https://orcid.org/0000-0002-6584-0786
Rebecca Waller https://orcid.org/0000-0002-5069-1183

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