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Personality (Dys)Function and General Instability

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Abstract

Humans adapt to a dynamic environment while maintaining psychological equilibrium. Systems theories of personality hold that generalized processes control stability by regulating how strongly a person reacts to various situations. Research shows there are higher-order traits of general personality function (Stability) and dysfunction (general personality pathology; GPP), but whether or not they capture individual differences in reactivity is largely theoretical. We tested this hypothesis by examining how general personality functioning manifests in everyday life in two samples (*N*s=205; 342 participants and 24,920; 17,761 observations) that completed an ambulatory assessment protocol. Consistent with systems theories, we found (1) there is a general factor reflecting reactivity across major domains of functioning, and (2) reactivity is strongly associated with Stability and GPP. Results provide insight into how people fundamentally adapt (or not) to their environments, and lays the foundation for more practical, empirical models of human functioning.

Maintaining equilibrium in a dynamic environment is a fundamental feature of self-regulating, goal-directed systems (Powers, 1973; Wiener, 1948). Humans display a range of psychological stability in the face of life challenges. According to dynamic systems theories, personality traits emerge from processes that regulate functioning in distinct affective, behavioral, and social domains, all of which work synchronously to maintain psychological equilibrium (Carver & Scheier, 1982; DeYoung, 2015; Van Egeren, 2009). Theorists thus speculate that a person's overall level of functioning is determined, in part, by domain-general regulatory processes that control stability. Despite being essential to our understanding of human adaptation, the manifestations of such generalized processes in everyday life have not been empirically well-characterized. These superordinate processes have been described as 'constraint' (Depue & Lenzenweger, 2005) or (at the opposite pole) 'impulsive responsivity' (Carver et al., 2017), constructs describing individual differences

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Author contributions:

All authors were involved in the development of the study concept. All authors were involved in parts of the data collection. W.R.R. performed the data analyses with input from A.G.C.W. W.R.R. drafted the manuscript and all authors provided critical revisions. All authors approved the final version of the manuscript for submission.

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in *reactivity*, or the tendency to have controlled responses to situations with(out) regard for long-term consequences. Many systems theories view reactivity as the core indicator of processes that maintain stability, and in this study we sought to directly test this hypothesis.

The idea of superordinate processes that account for general psychological functioning is supported by extensive evidence showing personality is hierarchically organized such that narrow, specific traits are correlated and form increasingly broad, higher-order individual differences. Of the major dimensions of personality, Neuroticism, Conscientiousness, and Agreeableness are most consistently linked to functional outcomes (Ozer & Benet-Martínez, 2006; Roberts et al., 2007) and their correlated variance is thought to reflect regulatory capacities that keep a psychological equilibrium; Neuroticism corresponds to variation in the capacity to regulate emotional reactions, Conscientiousness to impulse control and regulation of goal-directed behavior, and Agreeableness to prosocial tendencies that regulate reactions during social interactions. Indeed, the meta-trait formed by the shared variance of these traits has been termed Stability (DeYoung, 2015), and is almost invariably found in studies of trait structure (DeYoung, 2006; Digman, 1997; Markon et al., 2005). The shared variance of two other major traits, Extraversion and Openness has been referred to as Plasticity, a meta-trait encompassing the capacity to develop new adaptations. These basic functions subsumed by Stability and Plasticity can be viewed as complementary processes that are necessary for adaptation (DeYoung & Krueger, 2018). However, in empirical studies, Plasticity is recovered less consistently and its constituent traits tend to be less predictive of functional outcomes (cf. DeYoung et al., 2008; Tackett et al., 2008). Plasticity likely helps maintain equilibrium by generating novel responses to a changing environment (DeYoung, 2015), but the ability to regulate responses (i.e., Stability) appears to be the main determinant of overall functioning.

Reinforcing the primary role of Stability, converging evidence from clinical psychology has identified psychological *instability* as a shared feature of personality dysfunction. Mirroring research in normative personality, clinical research has found that the shared variance among personality disorder symptoms and maladaptive traits form a higher-order dimension of general personality pathology (GPP) that closely relates to (negative) Stability (Ringwald et al., 2019; Sharp et al., 2015). GPP captures impairments that cut across different manifestations of personality pathology, and is thought to reflect *dysregulation* of affect, behaviors, and social relating—that is, emotional reactivity, impulsivity, or quarrelsomeness that disrupts equilibrium. Unlike normative personality, a maladaptive variant of Plasticity is not reflected in the empirical structure of personality pathology. Instead, pathological variants of Extraversion and Openness share features of dysregulation with other maladaptive traits (e.g., Detachment and Psychoticism, respectively; although the relationship between Openness and Psychoticism is more tenuous; Widiger & Crego, 2019), underscoring the idea that personality dysfunction is more closely related to Stability than Plasticity. The robust evidence for general factors of personality (dys)function from distinct intellectual traditions suggests Stability and GPP are opposite poles of a continuum reflecting the capacity to regulate and maintain stability.

Empirical support for a systems theory interpretation of the general factors is limited, however, because the regulatory processes thought to explain general functioning unfold

in *dynamic* transactions with the environment that are not directly sampled by the cross-sectional assessments used to measure Stability and GPP. Ambulatory assessment (AA), on the other hand, directly samples people's responses across situations in their natural environment, but this research has remained almost entirely separate from research on these general factors (cf. Ringwald et al., 2020; 2021). A major advantage of AA is the measurement of within-person *variability* which gives an index of reactivity or how much a person's affect or behavior fluctuates from situation-to-situation. Variability is not a direct measure of regulatory processes, but it is considered a strong indicator (Ebner-Priemer et al., 2009): higher variability suggests a tendency toward intense reactions to everyday situations and lower variability suggests more controlled reactions.

Although it could be speculated that variability is an indicator of appropriate, flexible responding (and potentially, Plasticity), a large body of AA research suggests that it more likely maps onto maladaptive, dysregulated processes described by systems theories. Higher affective variability is associated with depression, social anxiety, bulimia, bipolar disorders, and personality disorders as well as non-clinical markers of lower well-being (Crowe et al., 2019; Houben et al., 2015; Houben & Kuppens, 2020; Lamers et al., 2018; Mneimne et al., 2018; Santangelo et al., 2014; Snir et al., 2017; Sperry & Kwapil, 2020; Trull et al., 2008). Although less researched than affective variability, variability in other domains such as interpersonal behavior, perception of others, and self-esteem is also related to psychopathology (Farmer & Kashdan, 2014; Ringwald et al., 2020; 2021; Russell et al., 2007; Santangelo et al., 2017; Zeigler–Hill & Abraham, 2006). These findings suggest variability is an indicator of functioning that cuts across diverse psychological problems, but the evidence does not bear on whether variability in different domains reflects independent or *shared* processes. A related line of work shows that variability in positive and negative affect is heritable, despite average levels of affect having divergent genetic correlations, suggesting the regulatory processes (not one's typical emotional valence) comprise a substantive individual difference (Jacobs et al., 2013; Zheng et al., 2016). However, this research similarly has not tested if variability across valence systems have a shared basis; that is, whether the same people who are variable in negative affect tend to be variable in positive affect or whether they reflect separable regulatory processes.

Most AA research has focused on single disorders and personality traits as predictors of variability in circumscribed domains (e.g., only negative affect or dominant behavior), but the principal claim of systems theories is one's overall level of functioning emerges from generalized regulatory capacities that impact most domains. Thus, despite indirect evidence, there are two basic but untested hypotheses that follow from a systems conceptualization of personality: (1) affective, behavioral, and interpersonal variability will strongly covary from person-to-person and form a general variability factor if there are superordinate processes that influence reactivity across domains, (2) Stability and GPP will be correlated (negatively and positively, respectively) with a general variability factor.

Towards the goal of understanding the relationship of self-reported meta-traits to affective and behavioral dynamics in everyday life, we brought together research on Stability and GPP with AA methods to test these two hypotheses based on systems theory. We examined these questions in a sample enriched with people diagnosed with borderline personality

disorder (henceforth referred to as the clinical sample), a diagnosis typified by emotional and behavioral dysregulation. We also replicated all analyses in a community sample to establish whether our findings would generalize across the range of personality functioning.

Methods

Our analyses were not formally preregistered. De-identified data for both samples and code needed to reproduce our results, along with supplementary information, are posted on the Open Science Framework at: https://osf.io/dgu6s/

Participants and Procedures

The data for our study came from two independent samples representing a range of personality functioning, including a clinical sample enriched for personality pathology and a community sample. The clinical sample was drawn from a longitudinal study examining suicidal behavior and consisted of 153 participants diagnosed with borderline personality disorder and 52 that did not meet criteria for any lifetime psychiatric diagnoses (N = 205). Participants in the clinical sample were located in the surrounding area of Pittsburgh, Pennsylvania. The community sample consisted of 342 participants. Participation in the community sample was completed online and data was not collected on their geographic location. Demographic information for both samples is provided in Table 1. Informed consent in both samples was obtained in accordance with approved protocol guidelines of the University of Pittsburgh Institutional Review Board.

In both samples, personality traits were assessed at a baseline session and functioning in everyday life was assessed by an AA protocol. Surveys for the AA protocol were delivered via push notifications on study-provided smartphones (clinical sample) or the participant's personal smartphones (community sample). Participants in the clinical sample completed an average of 5.9 surveys per day for 21 days. A total of 24,920 surveys were used in the analyses, all of which assessed affect and 8,950 included reports on interpersonal interactions. Participants in the community sample completed an average of 6.1 surveys per day for ten days. A total of 17,761 surveys used in the analyses assessed affect with 11,478 of those including ratings of interpersonal interactions. Details about sampling, participant characteristics, and study protocols are in the supplementary materials.

Our sample sizes were not predetermined for the specific analyses used in the current study as both samples were collected for parent studies conducted prior to the formulation of our research questions. For the clinical sample, power calculations were based on goals of the parent study to detect the cross-level interaction of group as a moderator of within-person associations between AA variables (see supplementary materials for additional information about groups in this sample). Informed by comparable AA studies, it was determined that the sample size achieved would result in a power of >.99 to detect small, between-group effects. For the community sample, sample size was determined primarily by the desire to achieve stable estimates of effects rather than power to detect any specific effect size in the population, as well as the ability to detect small effects that are consistent with the average effect in the published personality psychological literature. Recent work showing that correlation estimates of this size begin to stabilize when sample sizes approach N

= 250 (Schönbrodt & Perugini, 2013). Therefore, a minimum sample size of 250 was selected for the most conservative modeling situations of interest, which was establishing between-person associations. To adjust for expected exclusion due to low participation rates in ~10% of participants, the target sample size was $N > 300$. In the current study, we also did not aim to detect a specific effect size; however, our samples sizes were much larger than typical AA samples and both were adequate (community sample) or close to adequate (clinical sample) to achieve stable estimates of effects.

Measures

McDonald's omega (ω) was used to assess reliability of our scales. Complete reliability results are reported in the supplementary materials.

Personality.—Basic personality traits were assessed by self-report using the 120-item International Personality Item Pool representation of the NEO-PI-R (IPIP-NEO-120; Johnson, 2014) in the clinical sample and by the 60-item Big Five Inventory – 2 (BFI-2; Soto & John, 2017) in the community sample. For both instruments, participants rated the extent to which a characteristic applies them (e.g., "I am someone who is outgoing"). In the IPIP-NEO-120, items were rated on a Likert scale from "Very Inaccurate" (1) to "Very Accurate" (5). In the BFI-2, items were rated on a Likert scale from "Disagree Strongly" (0) to "Agree Strongly" (4). Items were averaged to produce facet scales and the mean of corresponding facet scales were used to calculate trait scores of Neuroticism, Agreeableness, Conscientiousness, Extraversion, and Openness in the NEO-PI-R, and Negative Emotionality, Agreeableness, Conscientiousness, Extraversion and Open-Mindedness in the BFI-2. We used the general labels of Neuroticism and Openness in this study. Reliability for the trait scales in both samples was high (mean ω = .86).

General personality pathology.—Maladaptive personality traits were self-reported using the 220-item Personality Inventory for the *DSM-5* provided by the American Psychiatric Association (PID-5; Krueger et al., 2012) in the clinical sample. In the community sample, the 100-item PID-5 short form was used (PID-5-SF; Maples et al., 2015). For both versions of the PID-5, participants rated how well a series of statements described them (e.g., "I feel like I act totally on impulse") on a Likert scale from "Very False/ Often False" (0) to "Very True or Often True" (3). Items were averaged to create facet scores, then trait scores were calculated by averaging three corresponding facets (APA, 2013). The traits and corresponding facets used in this study were Antagonism (Manipulativeness, Deceitfulness, Grandiosity), Detachment (Withdrawal, Anhedonia, Intimacy Avoidance), Disinhibition (Irresponsibility, Impulsivity, Distractibility), Negative Affectivity (Anxiousness, Emotional Lability, Separation Insecurity), and Psychoticism (Unusual Beliefs, Eccentricity, Perceptual Dysregulation). Reliability for the trait scales in both samples was high (mean ω = .88).

Positive and negative affect.—In both samples, affect was self-reported at random intervals throughout the day during the AA protocol. Affect was rated using emotion adjectives derived from the Positive and Negative Affect Schedule – Expanded Form (PANAS-X; Watson & Clark, 1994) along with an additional adjective ('Content') that is not

included in the PANAS-X. Questions were adapted for AA and asked: “How ADJECTIVE do you feel right now?” Each adjective was rated on a Likert scale from “Very Slightly” (1) to “A Great Deal” (5) in the clinical sample and on a slider scale from “Not at All” (0) to “Extremely” (100) in the community sample. Positive affect was assessed with the adjectives Happy, Proud, Confident, Excited, and Relaxed in the community sample and Happy, Excited, and Content in the clinical sample. Negative affect was assessed with the adjectives Ashamed, Nervous, Hostile, Angry in the community sample and Nervous, Sad, Angry, and Irritated in the clinical sample. Reliability for the affect scales in both samples was high (mean $\omega_{\text{between-person}} = .89$).

Interpersonal behavior of self and others.—In both samples, for AA surveys that participants indicated that an interpersonal interaction had occurred, they reported on their own behavior and the behavior of the person they interacted with. The item prompts were: “Please rate YOUR BEHAVIOR toward the other person during the interaction” and “Please rate how the OTHER PERSON BEHAVED toward you during the interaction.” For both self and other, dominance was rated from “Accommodating/Submissive/Timid” to “Assertive/Dominant/Controlling” and warmth was rated from “Cold/Distant/Hostile” to “Warm/Friendly/Caring” on 101-point slider scales (−50 to +50).

Analytic Plan

We used multi-level structural equation modeling (MSEM) to investigate associations among three latent variables reflecting general personality functioning: the meta-trait of Stability, GPP, and generalized, cross-domain variability (g-VAR). MSEM accommodates the multi-level structure of the AA data (i.e., observations nested within people) and enables estimation of latent variable path models (i.e., associations among general factors of personality functioning).

In MSEM, observed variables are partitioned into within- and between-person variance using latent decomposition. Variables at the between-person level represent trait-like individual differences. Included at this level were the IPIP-NEO-120/PID-5 traits as well as the between-person part of the AA measures which estimates of each person’s average (i.e., trait-like) endorsement of those variables over the study period. This is similar to calculating person means and then person-mean centering the variables as is often done in standard multi-level modeling, except in MSEM the partitioning is estimated by the model rather than being done manually (Asparouhov & Muthén, 2019). Our analyses focused on associations at the between-person level, so although we did not interpret within-person parameters, we modeled this variance to provide unbiased estimates of the averages for AA constructs. A small number of participants ($n = 16$ to 21) were missing data on personality traits. Missing data were handled using full information maximum likelihood estimation.

To test our principal hypotheses, we fit models estimating the correlations between Stability, GPP, and g-VAR in both the clinical and community samples. The Stability factor was estimated from the shared variance of Neuroticism, Conscientiousness, and Agreeableness, and the GPP factor from the shared variance of maladaptive traits. g-VAR was estimated from the shared variance of AA-measured variability of negative and positive affect,

dominant and warm behavior, and perceived dominance and warmth in others. We also conducted sensitivity analyses with models including Plasticity, which was estimated from the shared variance of Extraversion and Openness.

To index variability, we used the square of successive differences (SSD) between adjacent observations of the same measure (e.g., negative affect) within-person. Unlike other measures of variability like the standard deviation, the SSD accounts for the temporal ordering of observations and thus is less influenced by trends (e.g., linear) in the data. As such, the SSD may be the most appropriate index of the volatile reactions elicited from situation-to-situation thought to indicate dysregulation.[1] We used the latent, between-person variance in AA measures to estimate each person's mean SSD (MSSD) because this provides a more reliable estimate than observed person means (Lüdtke et al., 2008).[2]

Model fit was evaluated using standard benchmarks for alternative fit indices (i.e., root mean square error of approximation [RMSEA] ≤ .05, comparative fit index [CFI] ≥ .95, standardized root mean residual [SRMR] ≤ .08; Hu & Bentler, 1999) along with interpretation of the size and statistical significance (two-tailed p-value < .05) of the factor loadings and strength and significance of associations between personality functioning variables. The χ^2 is traditionally used to assess global model fit, but it can be highly sensitive to ignorable sources of ill fit in large samples such as those used in the current study, so we prioritized alternative fit indices. In cases that the model fit indices suggested misspecification, we determined sources of model misfit by identifying strong bivariate correlations between indicator variables in combination with theoretical considerations. Details for our modeling decisions about the parameters included in the final models are in the supplement.

In prior work using these samples (Ringwald et al., 2020; 2021), we estimated GPP and examined its associations with affective and interpersonal variability. However, in those studies we only investigated variability in single functioning domains, and did not examine the role of normative personality traits. Here, we estimated a distinct construct of variability across affective and behavioral domains. Furthermore, in prior work, we operationalized variability using latent residual variances instead of MSSDs. We attempted to estimate a general factor of variability using latent residual variances in the current study, but these models would not converge due to their complexity (i.e., dozens of variances and covariances among random effects). Thus, the current study builds on prior investigations, but the analyses and results are unique.

All analyses were done using Mplus Version 8.6 (Muthén & Muthén, 2020) and Mplus Automation package for R (Hallquist & Wiley, 2018).

[1]Some authors studying the same constructs using the same metrics we do here have used the term "instability" to refer to this behavior pattern. We use the term variability to distinguish the observed behavior pattern from the inferred construct of psychological stability.

[2]We ran all models with observed MSSDs which attenuated the effect sizes (i.e., factor loadings, regression/correlation coefficients) but the overall pattern of associations was the same

Results

Bivariate correlations among study variables are presented in Table 2. Of note, the correlations between indicators of pathology (e.g., variability, maladaptive traits, low stability traits) were generally much stronger in the clinical sample than in the community sample. Indeed, as reported below, nearly all associations among pathology measures in the structural equation models (e.g., factor loadings, regression coefficients) were stronger in the clinical sample. The likely reason for this is that the clinical sample includes a wider range of personality pathology due to selecting participants with very high pathology (people diagnosed with borderline personality) and very low pathology (healthy controls). The community sample included participants with a narrower range of pathology (i.e., less representation of very high pathology), and this range restriction may have attenuated the observed correlations relative to the more representative range in the clinical sample.

Generalized variability factor

To test our first hypothesis, we evaluated whether variability in disparate domains of everyday functioning reflect shared, higher-order processes (i.e., a general factor). As hypothesized, we found that the MSSD in all domains of functioning were strong indicators of a single, latent variability factor which we will refer to as generalized variability or *g-VAR*. The mean factor loading for MSSDs in each domain was .73 in the clinical sample and .68 in the community sample (complete factor loadings are in Table 3). Although the MSSDs in each domain were strongly, positively correlated, there were no consistent correlations among average levels of affect, behavior, and perceptions (mean |r| = .23, |range| = .01 - .96). These results support the idea that domain-general reactivity *per se* is captured by g-VAR.

Associations among Stability, general personality pathology, and generalized variability

Just as variability in distinct areas of functioning reflects a common factor, we confirmed previous work showing that Neuroticism, Agreeableness, and Conscientiousness were strong indicators of a latent Stability factor and maladaptive personality traits were all strong indicators of a GPP factor (all factor loadings ($|\lambda|$) ≥ .59 in the clinical sample and $|\lambda|$ ≥ .47 in the community sample; see Table 3 for complete factor loadings). To test our second hypothesis, we examined associations between all three of these general factors thought to reflect overall personality functioning: Stability, GPP, and g-VAR.

The models including intercorrelations between Stability, GPP, and g-VAR fit the data well according to all global fit indices in the clinical sample (RMSEA= .01, CFI = .96, SRMR = .08) and the community sample (RMSEA = .01, CFI = .97, SRMR = .08) (models are shown in Figure 1). Stability and GPP were strongly, negatively correlated consistent with prior work and with the idea that they represent opposite ends of a functioning continuum. Unlike prior work comparing general factors, these models allowed us to go beyond speculation about how individual differences in general functioning manifest in everyday life. As hypothesized, we found that Stability was correlated with lower g-VAR indicative of generally less reactive (i.e., well-regulated) responses and GPP was correlated with higher g-VAR indicative of more reactive (i.e., dysregulated) responses.

We modeled Stability and GPP as separate variables because they come from distinct theoretical traditions and empirical literatures. However, we also tested our hypothesis by modeling the correlation between g-VAR and a single personality functioning variable estimated from all maladaptive traits and Stability traits. Full results from these exploratory analyses are in the supplementary material. In sum, these models fit the data comparably to those used for our main analyses, every trait loaded strongly onto a single general factor (all $|\lambda| > .38|$, and the correlation between g-VAR and the general personality functioning factor was comparable to our main analyses ($r_{community} = .36$; $r_{clinical} = .62$). While both modeling approaches support our claim that Stability and GPP are part of a unified spectrum of functioning, we prioritize results with separate general factors to provide continuity with previous work.

Sensitivity analyses of the role for Plasticity in personality functioning

Given the theoretical interrelationship of Stability and the meta-trait of Plasticity for overall functioning, we conducted sensitivity analyses examining the specificity of Stability (versus Plasticity) to variability and general personality functioning. Full results from these analyses are in the supplementary material. In one model, we examined correlations between Stability, Plasticity, GPP, and g-VAR. In line with the broader literature that often fails to recover Plasticity, we were only able to model it in the community sample because Extraversion and Openness were uncorrelated in the clinical sample. Supporting the interpretation that variability reflects dysregulation more than flexibility, we found that Plasticity was uncorrelated with g-VAR in the community sample ($r = -.11$, $p = .248$). Furthermore, the association between GPP and Stability was nearly three times the size of the association with Plasticity ($r_{Stability} = -.76$, $r_{Plasticity} = -.26$). Taken together, these results suggest g-VAR is a manifestation of Stability, not Plasticity, and that Stability is more closely related to general personality dysfunction. Because Stability and Plasticity were positively correlated with one another ($r = .37$), and previous work has raised concerns about mono-method measures of personality (i.e., same rater and same instrument; DeYoung, 2006; Hopwood et al., 2011), we estimated a second model at the suggestion of reviewers adjusting for this shared variance. In this model, Stability and Plasticity were entered as simultaneous predictors of g-VAR. We did not include GPP in this model because of the collinearity of Stability and GPP. This model showed that Stability was uniquely associated with lower g-VAR ($\beta = -.63$) and Plasticity with higher g-VAR ($\beta = .40$), indicating a suppression effect.

Associations between lower-order traits and generalized variability

Finally, to assess the extent to which g-VAR is accounted for by general personality functioning versus lower-order traits, we conducted a series of regression analyses disentangling these sources of variance. Because systems theories of personality posit generalized, higher-order processes that regulate stability across domains, we expected that Stability and GPP, not the lower order traits, would predict g-VAR. As a basis of comparison, we first estimated the bivariate associations between g-VAR and personality traits. With few exceptions, all personality traits were significantly associated with g-VAR in bivariate regression models. Next, we regressed g-VAR simultaneously on Neuroticism, Conscientiousness, and Agreeableness in one multivariable model, and on the five

maladaptive traits in another model. These models are akin to partialling out Stability and GPP, respectively, to estimate associations between traits and g-VAR net general personality functioning, though they also partial out the portion of variance shared between pairs of traits that is not shared among all traits (i.e., variance captured by the general factor).[3] Results comparing bivariate and multivariable results are shown in Table 4. After adjusting for their shared variance (i.e., general personality functioning), Neuroticism and Negative Affectivity in both samples, and Antagonism in the community sample, were the only lower-order traits that remained significantly associated with g-VAR. This pattern of results indicates that Neuroticism/Negative Affectivity, Antagonism, and general personality functioning capture shared *and* distinct processes. All other associations between the lower-order traits and g-VAR reduced in magnitude and were non-significant. As expected, this shows that the link between personality and reactivity is largely accounted for by the higher-order dimension of general functioning.

The finding that associations between Neuroticism/Negative Affectivity were virtually unaffected by partialling out the substantial shared variance with Stability/GPP led to our re-evaluating these models. Based on modification indices and considering the content overlap between Neuroticism/Negative Affectivity and negative affect variability (i.e., the trait scales contain items related to emotional lability), we added a path allowing their residuals to freely correlate in exploratory analyses. Inclusion of this path resulted in overall weaker bivariate and specific associations between Neuroticism/Negative Affectivity and g-VAR. This could indicate that in models without this path, the association between Neuroticism/Negative Affectivity and g-VAR was inflated by the traits' specific correlation with negative affect variability. However, because it is debatable whether the influence of overlapping content between Neuroticism/Negative Affectivity and negative affect variability should be considered artifactual or substantive, we only report results adjusting for their residual correlations in the supplementary materials.

Discussion

According to systems theories, an organism regulates its responses to the environment in a way that maintains internal equilibrium. Across two samples, we found support for foundational but untested hypotheses following from this claim in systems theories of personality: (1) shared processes account for reactivity across major domains of functioning, (2) individual differences in this generalized pattern of perceiving and responding to situations is strongly associated with general personality function (Stability) and dysfunction (GPP). These findings provide insight into how psychological equilibrium is maintained or lost in everyday life, and suggest generalized regulatory processes underlie the multitude of ways people adapt (or not) to their environment.

[3]We also examined correlations between g-VAR and the lower-order trait residuals of Stability/GPP and g-VAR to estimate associations between traits net general personality functioning. For these models to be identified, we estimated one trait residual correlation per model. These models produced highly unstable results (reported in full in the supplementary materials), in part due to the requirements for model specification and the number of parameters relative to the sample size. We also attempted several other MSEM models that removed only Stability/GPP but these produced similarly unstable results. Given these considerations, we think that the multivariable regression approach offers the most accurate estimates of trait-specific associations.

We identified a clear behavioral profile of general personality functioning that is indicative of reactivity. The strong general factor of variability shows that people who tend to experience more intense shifts in affect also report more extreme changes in behavior across interactions and tend to perceive others in more extreme terms. Likewise, people who report more stable affect also report more stable interpersonal behavior and perceptions of others. The lack of associations between generalized variability and specific traits reinforces the idea that reactivity is a defining feature of general personality functioning, and our finding that Plasticity only relates to variability after adjusting for Stability clarified that this variability is not simply adaptive flexibility.

Although general personality functioning accounted for most associations between lower-order traits and generalized variability, the unique effects of Neuroticism and Negative Affectivity were of comparable or greater magnitude to those of Stability and GPP. One interpretation of this finding could be that Neuroticism/Negative Affectivity is the main driver of dysregulation rather than a higher-order, general functioning trait, in line with arguments that negative emotionality is the core of psychopathology (Lahey et al., 2017; Tackett et al., 2013). However, these associations we found between variability and Stability/GPP and Neuroticism/Negative Affectivity are independent effects; that is, the strong, unique effects of Neuroticism/Negative Affectivity reflect only features of this trait that do not overlap with Agreeableness and Conscientiousness or all other maladaptive traits, whereas Stability/GPP only capture features that overlap with other lower-order traits. Our view is that there may be separate regulatory processes that give rise to variability linked to Neuroticism/Negative Affectivity versus Stability/GPP, and that both sets of processes are important for understanding different aspects of functioning. For instance, it could be that variance in Neuroticism/Negative Affectivity that is not shared with other traits relates to primarily emotional problems (e.g., depression, anxiety) in which interpersonal impairments (e.g., manifested in variable social behavior/perceptions) are secondary, whereas Stability/GPP relates to more pervasive problems in which interpersonal impairments are primary (e.g., personality disorders). Our supplementary analyses hint at the specificity of Neuroticism/Negative Affectivity to negative affect variability as allowing their residuals to correlate notably attenuated its association with general variability. Research investigating the course, correlates, and outcomes of these independent sources of variability will help clarify the nature of the underlying processes.

Differences in sensitivity of the clinical and community samples to detect certain results highlight how general personality dysfunction overpowers trait-specific patterns of behavior. In the clinical sample, the associations between Neuroticism/Negative Affectivity and variability were comparable or somewhat weaker than the associations with general personality functioning, but in the community sample, the associations between variability and Neuroticism/Negative Affectivity were stronger than those with general personality functioning. Because the general factor was relatively weak in the community sample due to limited representation of very high psychopathology, participant's everyday patterns of behavior were less influenced by general dysfunction which allowed for more trait-specific effects to be detected. The trait-specific results observed in the community sample suggest that even at relatively low levels of pathology, more neurotic or antagonistic people tend to be more reactive. In contrast, the higher level of impairment and wider range of pathology

in the clinical sample (i.e., healthy controls and people diagnosed with BPD) allowed us to obtain a stronger signal of how general personality functioning manifests in everyday life. Results from both samples support the conclusion that personality dysfunction manifests in generalized reactivity, and that there may be shared *and* specific processes underlying this tendency.

Plasticity is important for adaptive functioning, but our results provide evidence it may be less fundamental than Stability. In line with the broader literature suggesting Plasticity is not a robust component of basic personality structure, we could not estimate it in the clinical sample. Additionally, in the community sample, although Plasticity did predict higher variability, it was only after accounting for shared variance with Stability, and Stability was much more strongly associated with generalized variability than was Plasticity. Taken together, our findings suggest that variability does indicate flexibility to an extent such that holding level of Stability constant, a person with higher levels of Plasticity will be more variable in their emotions, behaviors, and perceptions of others–but variability primarily reflects poorly regulated reactions. These results also underscore a complicating factor for making inferences from variability—namely, that the adaptiveness of affective or behavioral responses is inherently contextual. High variability could indicate flexibility if a wider response repertoire was needed to navigate shifts in the environment. Likewise, low variability could indicate rigidity if a person was failing to adapt their behavior to new circumstances. Put another way, the magnitude of a person's responses at any given moment (or AA assessment) is only dysfunctional if it does not meet the demands of the situation. We focused on between-person differences in typical response patterns by aggregating across contexts, but examining the contextualized, within-person processes can help differentiate between flexibility versus instability and stability versus rigidity. Measuring and modeling the within-person relationships between a response and the situation is a challenge, which some analytic approaches have begun to address (e.g., Geukes et al., 2017; Morse et al. 2015; Sadikaj et al., 2013).

Although variability is not a direct measure of regulatory processes, our results support its validity as a regulatory index and establish necessary groundwork for answering the next question of what *mechanisms* explain the observed pattern of variability. Several regulatory processes have been proposed by systems theories based on evidence from various scientific disciplines (e.g., neurobiology, clinical, and cognitive psychology) that may explain individual differences in reactivity. For example, higher reactivity may be caused by stronger sensitivity to situational cues (Depue & Spoont, 1986) and overreliance on reflexive, impulsive modes of processing (Carver & Johnson, 2018; Huys & Renz, 2017). These broad processes are consistent with our results showing that personality dysfunction correlated with more extreme shifts in emotional and behavioral reactions from one situation to the next. At the neurobiological level, serotonergic functioning may be a broad-based mechanism that governs variation in these complex, behavioral processes that maintain stability. Along with indirect evidence from human and non-human animal studies, the meta-trait of Stability has recently been directly linked to low central serotonergic functioning using experimental psychopharmacology (Wright et al., 2019). Importantly, serotonergic functioning is not a unitary system, so another piece of the puzzle will be establishing which serotonergic pathways and receptor subtypes are implicated in affective and behavioral

variability. A comprehensive model of human functioning must account for multiple levels of analysis including personality traits, neurobiology, and naturalistic behavior patterns and our study puts us one step closer towards this end.

Our finding that g-VAR is a behavioral commonality among various manifestations of psychopathology (e.g., Antagonism and Detachment) informs the interpretation of general factors from cross-sectional trait measures. It has been argued that features with opposing surface characteristics (e.g., quarrelsomeness in Antagonism and withdrawal in Detachment) could not logically share a substantive commonality; rather, according to this view, the general factor of personality pathology must represent overall impairment resulting from distinct, yet unrelated, mechanisms (Oltmanns et al., 2018; Smith et al., 2020).[4] Our findings contradict this perspective, as variability across domains shared sizable variance indicating substantive processes—namely, reactivity—subsume the particular eliciting cues, emotions, or behaviors associated with a given person's problems. This suggests that what is shared among quarrelsomeness and withdrawal, for instance, is that these features arise from a tendency to have intense, short-sighted reactions, albeit to different types of situations (e.g., perceived dominance in others versus perceived coldness) and in the form of different emotions (e.g., anger versus shame) and behaviors (e.g., picking fights versus avoiding social contact).

The possibility of shared mechanisms of general functioning aligns with evidence for common mechanisms of change in psychotherapy and the effectiveness of serotonergic drugs for wide-ranging psychopathologies. Much like pathologies with different surface characteristics may reflect core processes, there are thought to be core processes driving outcomes across superficially different psychotherapy approaches (Cuijpers et al., 2019; Mansell, 2011). Likewise, the fact that serotonin-targeting medications are first-line treatment for many mood disorders suggests core neurobiological processes. It is reasonable to consider whether generalized mechanisms of change in treatment work because they target generalized mechanisms of dysfunction. Studying the interface of these functional processes has important, practical implications for how psychopathology is conceptualized and treated. To illustrate, a proposed, generalized mechanism of change in psychotherapy is emotional awareness (Høglend & Hagtvet, 2019) which may be effective because it promotes more reflective modes of responding, which reduces reactivity and enables psychological equilibrium. Similarly, serotonin-targeting medications may have moderate effectiveness for different problems by affecting various neural systems implicated in emotional and behavioral control (note that the behavioral pharmacology of serotonin is complex and beyond the scope of this discussion; see e.g., Cools et al., 2008). Empirical evidence for mechanisms of change in psychotherapy has remained elusive and exactly how serotonin-altering medication influences behavior is not fully understood. Future investigations could leverage behavioral indicators of general dysfunction (i.e., g-VAR) as treatment outcomes in randomized control trials or experimental psychopharmacology studies to test whether treatments target mechanisms underlying reactivity. Isolating effects on general mechanisms could also clarify symptom-specific mechanisms, and matching

[4]The cited authors' impairment interpretation extends to a general factor of personality (i.e., shared variance of the five major traits), which our results do not provide evidence for or against. Our tentative, substantive interpretation applies only to GPP and Stability.

general and specific processes of problem maintenance and change could lead to greater precision in treatment. Matching general and specific processes of problem maintenance and change could lead to greater precision and efficiency in treatment. More generally, joining together research and theory on general mechanisms of dysfunction and interventions that change functioning could deepen our understanding of each side of the equation.

There are a number of additional ways future research could expand on our findings. To further evaluate the extent to which variability is a truly general pattern, it would be informative to test whether variability in other domains of functioning (e.g., self-esteem, daily activity rhythms, sleep) also form a general factor. For example, (in)stability in self-esteem and sense of identity are important components of personality, and it could be fruitful to evaluate the extent to which they are tied to affective and interpersonal processes or whether they entail separable processes. If people tend to be unstable in both self and interpersonal domains, this would be consistent with personality theories that propose self and interpersonal functioning are inherently intertwined (e.g., Bender et al., 2011; Luyten & Blatt, 2013; Pincus, 2005). If, however, instability in these domains are uncorrelated individual differences, this would suggest relatively independent pathogenesis for self and interpersonal problems. It will also be important to establish the generality of these processes across cultures and regions. It is possible that the macro-level social context plays a role in whether variability relates to dysfunction. The samples used in our study did not have adequate variation or data on these factors to test such questions; thus, future research in more geographically diverse samples is needed to determine the cultural-specificity of our results.

Our study only used self-report measures, albeit from both cross-sectional and intensive longitudinal assessments, which provides a limited view of a multi-system construct like personality functioning. Self-report is the optimal way to capture subjective socioemotional experiences, which play a critical role in regulatory processes. However, a more complete picture could be gained by investigating the relationship between personality and variability assessed by other reporters and direct behavioral measures like smart phone sensor data. Partitioning sources of variance through multi-method approaches could be used to compare the relative influence of subjective, evocative (i.e., evoking reactions from the environment), and behavioral processes on functioning outcomes (Caspi et al., 2005). For instance, it could be fruitful to compare the effects of *perceiving* instability in one's everyday life (self-report), being objectively variable (e.g., behavioral consistency measured by smartphone GPS sensors), or being *perceived* as unstable by others (informant report). Disentangling these sources of variance in ongoing research can clarify the role of these interacting components of personality, the environment, and their interrelationship.

Our study provides evidence for shared regulatory processes that account for reactivity across major domains of functioning consistent with what has been termed constraint and impulsive responsivity in systems theories of personality. These results suggest that psychological equilibrium at the core of human functioning is maintained by generalized processes of responding to situations. Our findings give insight into essential questions of how we adapt (or not) to our environment; by identifying a concrete, behavioral profile for general functioning, we set a foundation for developing more empirically-based and

practical conceptual models and for uncovering mechanisms of change needed to improve treatment outcomes.

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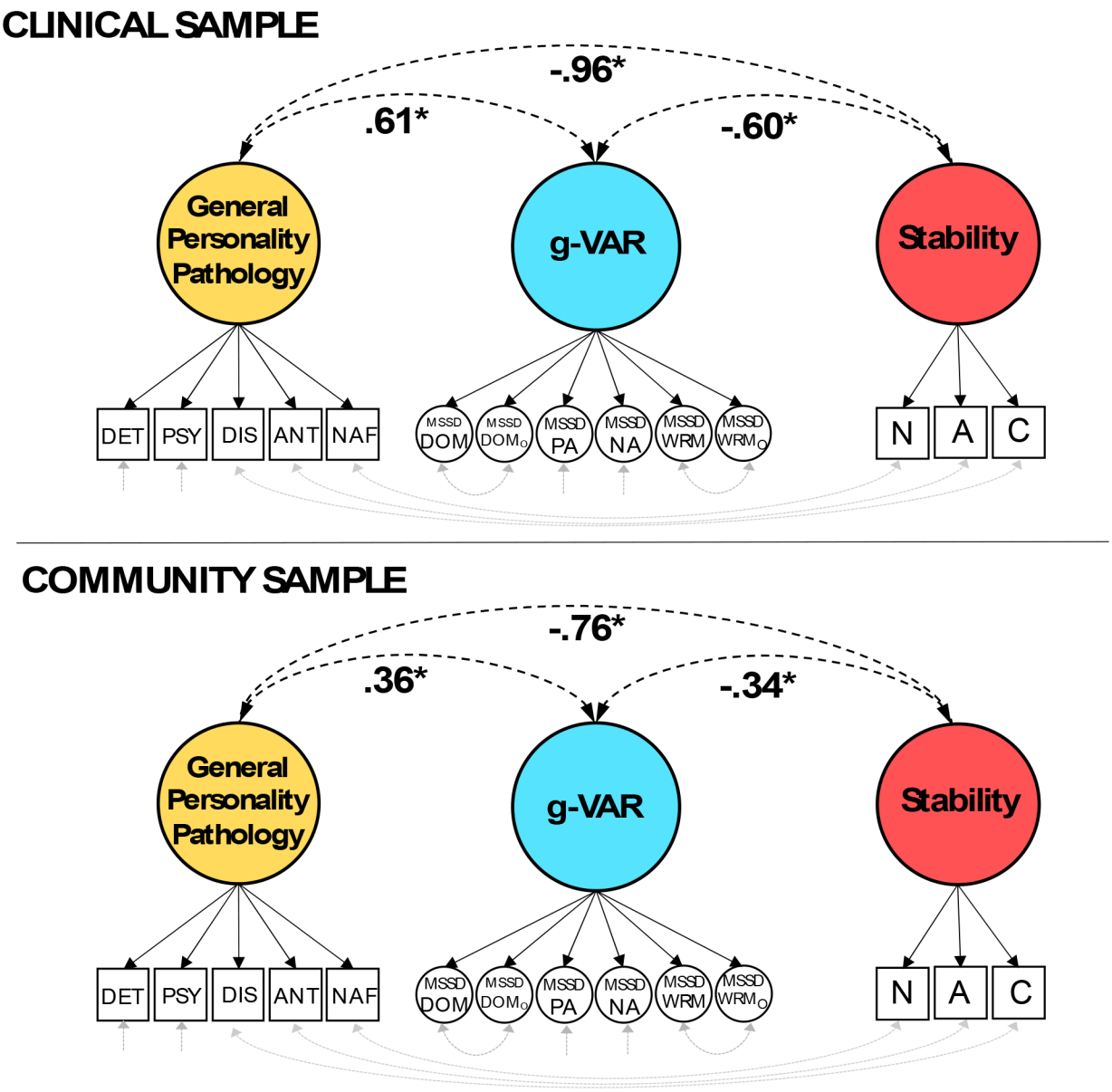

Figure 1.

Structural equation models of associations between personality functioning and general instability

Note. Double-headed arrows are correlation paths; * indicates the correlation coefficient is statistically significant ($p < .001$); circles are latent variables, squares are observed variables; g-VAR = generalized variability, DET = Detachment, PSY = Psychoticism, DIS = Disinhibition, ANT = Antagonism, NAF = Negative Affectivity, N = Neuroticism, A = Agreeableness, C = Conscientiousness, MSSD = mean squared successive differences, DOM = self dominant behavior, DOM_o = perception of other's dominant behavior, WRM = self warm behavior, WRM_o = perception of other's warm behavior, PA= positive affect, NA = negative affect.

Table 1.

Participant demographics

	Clinical Sample	Community Sample
Age (mean[SD])		
	32.4(9.6)	27(4.9)
Gender (n)		
Female	163	177
Male	35	162
Transgender/non-binary/other	7	3
Race (n) [5]		
African-American	28	16
American Indian	4	1
Asian	14	28
Pacific Islander	2	2
White	152	299
Other	--	6
Income (n) [6]		
Less than $14,999	50	21
$15,000–29,999	33	51
$30,000–59,999	44	109
$60,000 and above	40	161

[5]Race data was unavailable for 5 participants in the clinical sample. Community participants could identify as more than one race so the race N > 342.

[6]Income data was unavailable for 38 participants in the clinical sample.

Table 2.

Bivariate correlations among study variables

	PA MSSD	NA MSSD	Dom MSSD	Warm MSSD	Dom_o MSSD	$Warm_o$ MSSD	N	A	C	E	O	NAF	ANT	DIS	DET	PSY
PA MSSD		.40	.40	.46	.30	.33	.14	−.16	−.15	.20	.22	.20	.21	.21	−.08	.25
NA MSSD	.67		.43	.59	.45	.61	.63	−.41	−.48	−.31	.25	.60	.42	.58	.50	.50
Dom MSSD	.38	.36		.71	.88	.61	.26	−.21	−.33	.00	.19	.35	.18	.35	.07	.22
Warm MSSD	.44	.54	.53		.73	.87	.46	−.25	−.36	−.16	.29	.50	.25	.40	.19	.30
Dom_oMSSD	.33	.34	.91	.49		.72	.36	−.29	−.41	−.08	.15	.45	.31	.46	.18	.34
$Warm_o$MSSD	.44	.53	.52	.79	.58		.48	−.26	−.41	−.21	.23	.48	.30	.48	.25	.37
N	.22	.40	.18	.25	.12	.16		−.39	−.74	−.59	.37	.87	.44	.75	.65	.55
A	−.01	−.13	−.11	−.26	−.14	−.17	−.30		.47	.15	−.05	−.44	−.73	−.55	−.42	−.48
C	−.04	−.07	−.06	−.03	−.05	−.10	−.27	.25		.47	−.33	−.71	−.53	−.86	−.53	−.55
E	.15	.10	.03	.10	.04	.09	−.25	.07	.28		−.02	−.42	.00	−.37	−.69	−.24
O	.06	−.01	.04	−.03	.05	.04	−.06	.12	.07	.25		.41	.26	.33	.07	.46
NAF	.24	.45	.19	.26	.20	.18	.77	−.21	−.25	−.16	−.05		.57	.79	.55	.62
ANT	.06	.22	.26	.27	.22	.24	.16	−.47	−.28	.15	−.04	.31		.65	.27	.61
DIS	.08	.19	.13	.09	.17	.19	.34	−.27	−.69	−.16	−.01	.50	.46		.58	.68
DET	−.06	.11	.03	.03	.04	.04	.43	−.34	−.36	−.57	−.17	.40	.31	.41		.50
PSY	.11	.20	.03	.06	.10	.09	.29	−.28	−.22	−.12	.07	.50	.40	.45	.39	

Note. Results from the community sample are below the diagonal, results from the clinical sample are above the diagonal. Affect and interpersonal behavior and perception were measured in the ambulatory assessment protocol, traits were measured by cross-sectional questionnaires. AA = ambulatory assessment; DET = Detachment, PSY = Psychoticism, DIS = Disinhibition, ANT = Antagonism, NAF = Negative Affectivity, N = Neuroticism, A = Agreeableness, C = Conscientiousness, MSSD = mean squared successive differences, Dom = self dominant behavior, Dom_O = perception of other's dominant behavior, Warm = self warm behavior, $Warm_O$ = perception of other's warm behavior, PA= positive affect, NA = negative affect. Cells are colored grey for ease of interpretation.

Table 3.

Factor loadings for the general factors of personality functioning

	Clinical Sample	Community Sample
Stability		
Neuroticism	−.85	−.66
Conscientiousness	.85	.46
Agreeableness	.59	.55
General Personality Pathology		
Antagonism	.68	.57
Detachment	.65	.54
Disinhibition	.93	.70
Negative Affect	.87	.72
Psychoticism	.75	.69
g-VAR		
Dominance MSSD	.72	.54
Other dominance MSSD	.80	.53
Warmth MSSD	.90	.68
Other warmth MSSD	.89	.69
Negative affect MSSD	.70	.84
Positive affect MSSD	.43	.74

Note. All factor loadings are statistically significant ($p < .001$); MSSD = mean squared successive differences

Table 4.

Bivariate and multivariable associations between lower-order traits and generalized variability (g-VAR)

	Bivariate β [95% CI]	Multivariable β [95% CI]
Clinical sample		
IPIP-NEO-120		
Neuroticism	**.54 [.41, .65]**	**.57 [.35, .78]**
Agreeableness	**-.34 [−.51, −.19]**	−.11 [−.29, .04]
Conscientiousness	**-.48 [−.64, −.32]**	−.17 [−.47, .08]
Extraversion	−.16 [−.36, .03]	—
Openness	**.29 [.13, .42]**	—
PID-5		
Negative Affectivity	**.61 [.48, .73]**	**.60 [.15, .97]**
Detachment	**.26 [.07, .45]**	−.16 [−.37, .04]
Antagonism	**.36 [.19, .53]**	−.10 [−.31, .11]
Disinhibition	**.58 [.43, .72]**	.30 [−.01, .62]
Psychoticism	**.43 [.26, .60]**	.06 [−.20, .32]
Community sample		
BFI-2		
Neuroticism	**.39 [.27, .49]**	**.39 [.25, .52]**
Agreeableness	−.16 [−.32, .00]	.03 [−.10, .16]
Conscientiousness	−.07 [−.20, .06]	−.16 [−.33, .01]
Extraversion	**.13 [.02, .25]**	—
Openness	.03 [−.09, .15]	—
PID-5		
Negative Affectivity	**.44 [.29, .58]**	**.44 [.29, .56]**
Detachment	.05 [−.09, .18]	−.14 [−.29, .01]
Antagonism	**.27 [.09, .44]**	**.30 [.10, .51]**
Disinhibition	**.19 [.05, .31]**	−.01 [−.19, .17]
Psychoticism	**.17 [.04, .29]**	−.10 [−.28, .08]

Note. Bolded values indicate the coefficient was statistically significant ($p < .05$). g-VAR = latent factor estimated from the shared variance of the mean squared of successive differences (MSSD) for positive/negative affect, dominant/warm behavior, and perceptions of others' dominance/warmth; PID-5= Personality Inventory for the DSM-5, IPIP-NEO-120 – International Personality Item Pool; BFI-2 = Big Five Inventory. Bivariate models included each trait as an independent predictor of g-VAR and reflect the total, unadjusted associations. Multivariable models included all traits from the same instrument as simultaneous predictors of g-VAR and reflect specific associations over and above shared trait variance (i.e., general personality functioning).