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Discrepancies Confer Vulnerability to Depressive Symptoms: A Three-Wave Longitudinal Study

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Discrepancies (i.e., a subjective sense of falling short of one's own standards) are a key part of the perfectionism construct. Theory suggests discrepancies confer vulnerability to depressive symptoms. Since most research in this area is cross-sectional, longitudinal research is needed to disentangle directionality of relationships and to permit stronger causal inferences. Determining whether discrepancies are an antecedent of depressive symptoms, a consequence of depressive symptoms, or both is critical to understanding the discrepancies–depressive symptoms relationship. Knowledge about the temporal stability of discrepancies is also only starting to emerge, and it is unclear whether discrepancies predict incremental variance in depressive symptoms above and beyond neuroticism (i.e., a dispositional tendency to experience negative emotional states). The present study tested relationships among discrepancies, neuroticism, and depressive symptoms in 127 1st-year undergraduates using a 3-wave longitudinal design. Results suggest discrepancies may be understood as a trait-state where people are both highly consistent in their rank order on discrepancies and fluctuate somewhat in the level of discrepancies they experience at a particular point in time. As hypothesized, discrepancies predicted increases in depressive symptoms, even after controlling for neuroticism. Contrary to hypotheses, depressive symptoms did not predict changes in discrepancies. This study extends a long tradition of theory noting the depressing consequences of believing that one has fallen short of one's own standards. Harsh self-criticism and unobtainable self-expectations involving a strong sense of imperfection may be part of the premorbid personality of people vulnerable to depressive symptoms.

Keywords: discrepancies, perfectionism, depression, neuroticism, cross-lagged analysis

Depression is a common, expensive, and impairing problem involving symptoms such as apathy, sadness, irritability, anhedonia, hopelessness, and fatigue. We conceptualize depressive symptoms as a dimensional construct where symptoms lie along a continuum of severity from mild to severe. Conceptualizing depressive symptoms in this way is consistent both with recent evidence (Klein, 2008) and with counseling psychology's attention to the entire spectrum of functioning rather than just focusing on diagnosable disorders (Gelso & Fretz, 1992). Given the destructive nature of depressive symptoms, research is needed to understand factors such as perfectionism involved in the development and the maintenance of depressive symptoms.

Evidence suggests there are two key dimensions of perfectionism. The first dimension is self-critical perfectionism (i.e., harsh self-rebuke, excessive concerns over others' opinions, nagging doubts about performance abilities, and negative reactions to perceived failures; Blatt, 1995; Dunkley, Zuroff, & Blankstein, 2003). Self-critical perfectionism involves a family of traits (Clara, Cox, & Enns, 2007), including self-criticism (Blatt, D'Afflitti, & Quinlan, 1976), socially prescribed perfectionism (Hewitt & Flett, 1991), concern over mistakes (Frost, Marten, Lahart, & Rosenblate, 1990), and discrepancies (i.e., a subjective sense of falling short of one's own standards; Slaney, Rice, Mobley, Trippi, & Ashby, 2001). Traits in the self-critical perfectionism family are consistently tied to depressive symptoms (Graham et al., 2010).

The second dimension is perfectionistic strivings (i.e., rigidly and ceaselessly demanding perfection of oneself; Stoeber & Otto, 2006). Perfectionistic strivings involve a family of traits (McGrath et al., 2012), including self-oriented perfectionism (Hewitt & Flett, 1991), personal standards (Frost et al., 1990), and high standards (Slaney et al., 2001). Perfectionistic strivings are weakly or incon-

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sistently related to depressive symptoms and may sometimes be negatively related to depressive symptoms (Stoeber & Otto, 2006). As traits in the perfectionistic strivings family are largely unrelated to depressive symptoms (e.g., Dunkley et al., 2003; Graham et al., 2010; Stoeber & Otto), we do not discuss or measure these traits in our study. This also means our study measures only a relatively small part of the wider perfectionism construct.

Vulnerability Model of Perfectionism and Depression

Various models aim to explain the perfectionism–depressive symptom connection. The vulnerability model asserts traits in the self-critical perfectionism family place individuals at risk for increases in depressive symptoms over time (McGrath et al., 2012). A vulnerability factor (sometimes called a risk factor) is tested with a longitudinal design. To be a vulnerability factor, a variable (e.g., discrepancies) must precede an outcome (e.g., depressive symptoms) and have a statistical association with an outcome (Jacobi, Hayward, de Zwann, Kraemer, & Agras, 2004). To offer a clinical example, a freshman enters university with self-critical tendencies and lofty self-expectations (e.g., seeing herself a future physician). Amid the many challenges of the 1st year of university, she experiences a general, subjective sense of falling short of her own standards that contributes to depressive symptoms such as sadness, amotivation, and hopelessness. A central tenant of the vulnerability model is that high between-persons interindividual stability is present among traits in the self-critical perfectionism family. Interindividual stability represents the degree to which the rank order of individuals is maintained over time (Caspi, Roberts, & Shiner, 2005). Evidence indicates traits in the self-critical perfectionism family have high interindividual stability and predict future depressive symptoms (e.g., Cox, Clara, & Enns, 2009; Dunkley, Sanislow, Grilo, & McGlashan, 2009; Rice & Dellwo, 2001; Zuroff, Igreja, & Mongrain, 1990). For example, Rice and Aldea (2006) showed that discrepancies (measured by the Discrepancy subscale of Slaney et al.'s, 2001, Almost Perfect Scale–Revised; APS-R-D) predicted increases in depressive symptoms (measured by Radloff's, 1977, Center for Epidemiological Studies Depression Scale) over 8 weeks in a sample of 84 undergraduates. Consistent with the vulnerability model, traits in the self-critical perfectionism family seem to persist before, during, and after depressive symptoms (Graham et al., 2010).

Complication/Scar Model of Perfectionism and Depressive Symptoms

The complication/scar model posits depressive symptoms precede and produce changes in personality (e.g., traits in the self-critical perfectionism family). Depressive symptoms impact key domains of function (e.g., social, psychological, and biological), and such symptoms may change typical, personality-linked styles of behaving, thinking, relating, and perceiving (Cox & Enns, 2003; McGrath et al., 2012). The complication/scar model (Bagby, Quilty, & Ryder, 2008) argues depressive symptoms lead either to transient changes in personality traits that subside once depressive symptoms remit (a complication effect) or to permanent changes in personality traits (a scar effect). Our research design (a three-wave, 130-day longitudinal study) does not permit assessment of long-term alterations in personality traits. Thus, we see our study as testing complication effects (i.e., changes in personality traits seen over several months). To provide a clinical illustration, struggling with the various challenges of the 1st year of university, a freshman becomes depressed. She experiences symptoms of apathy, tearfulness, and fatigue along with impairments in academic performance that contribute to changes in her personality traits. These changes involve a subjective sense of being flawed and falling short of her own standards. Her distressing feeling of imperfection decreases upon symptom remission several months later.

The complication/scar model is seldom tested in research on perfectionism, and results from the few studies devoted to this topic are mixed. Some research shows depressive symptoms lead to increases in self-critical perfectionism over time (e.g., Cox & Enns, 2003; Zuroff, Blatt, Sanislow, Bondi, & Pilkonis, 1999); however, other research fails to provide support for such complication/scar effects (e.g., Hawley, Ho, Zuroff, & Blatt, 2006). For instance, research indicates depressive symptoms at baseline do not predict changes in discrepancies roughly 8 weeks later (Rice & Aldea, 2006). There is still much to learn about the potentially complicating influence of depressive symptoms on traits in the self-critical perfectionism family.

Reciprocal Relations Model of Perfectionism and Depressive Symptoms

Vulnerability and complication models are not necessarily mutually exclusive (Shahar, Blatt, Zuroff, Kuperminc, & Leadbeater, 2004): Personality traits and depression symptoms may be reciprocally related. For example, discrepancies may predict changes in depressive symptoms and depressive symptoms may predict changes in discrepancies as seen in Figure 1.

Research on the reciprocal relations model is scarce, and available results are inconsistent. McGrath et al. (2012) found reciprocal relations between self-critical perfectionism and depressive symptoms in a four-wave, 4-week longitudinal study of undergraduates. Shahar et al. (2004) also observed reciprocal relationships between self-criticism and depressive symptoms in a two-wave, 12-month longitudinal study of adolescents, but this effect was specific to girls. In contrast, Hawley et al. (2006) tested the reciprocal relations model using a five-wave, 16-week longitudinal study of depressed patients undergoing treatment and showed perfectionistic dysfunctional attitudes predicted depressive symptoms over time, without an opposite pattern occurring. These divergent findings may arise from several factors (e.g., different samples, age groups, measures, timeframes for measures, and measurement schedules). In summary, the reciprocal relations model has some promising initial support, but additional research is needed to thoroughly test this model and to resolve inconsistent findings in prior studies. In the present study, we focused on the reciprocal relations model because it synthesizes both the vulnerability model and the complication model into a single, coherent framework.

Advancing Research on Perfectionism and Depressive Symptoms

Despite notable advances (e.g., Rice & Aldea, 2006), improvements in tests of the reciprocal relations model are needed. It is important to know whether central parts of the perfectionism

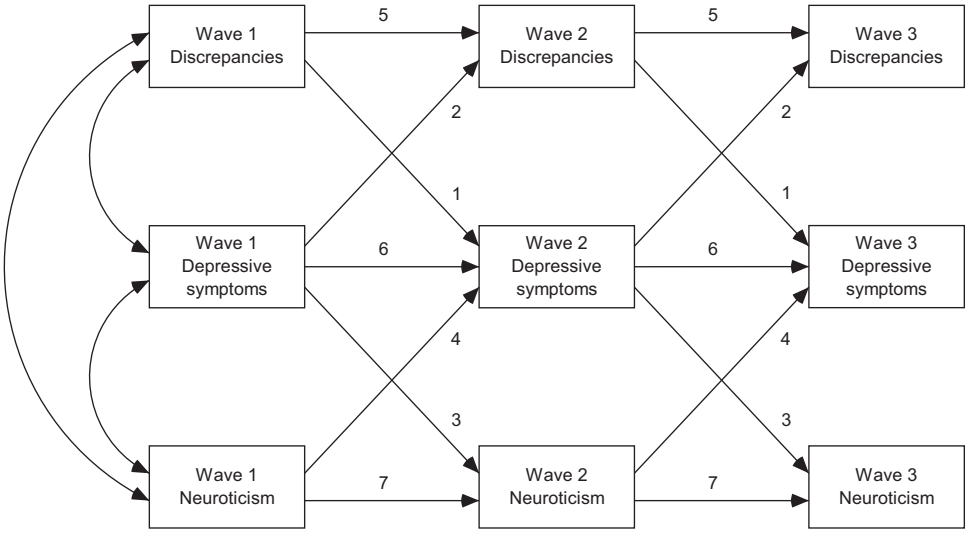

Figure 1. Cross-lagged path analysis. Rectangles represent measured variables. Double-headed black arrows represent correlations. Single-headed black arrows represent paths. Autoregressive paths are represented by horizontal arrows, and cross-lagged paths are represented by diagonal arrows. Covariances of paths sharing the same number were constrained to equality across waves. In the interest of clarity, error terms are not displayed.

construct (e.g., discrepancies) are an antecedent of depressive symptoms, a complication of depressive symptoms, or both. Such knowledge will guide theory, research, assessment, and intervention in counseling psychology and other areas.

Tests of the reciprocal relations model often involve two-wave longitudinal designs (for exceptions, see Hawley et al., 2006; McGrath et al., 2012). Such designs are problematic because they capture a narrow and potentially unrepresentative slice of change. Studies of the reciprocal relations model also often use long-term longitudinal designs with 12-month time lags (Shahar et al., 2004). Longer time lags may miss important short-term changes in depressive symptoms or perfectionism. Longitudinal studies of perfectionism and depressive symptoms also seldom use a research sample studied during a time period where change is likely (e.g., Graham et al., 2010). Finally, a multi-wave longitudinal design, combined with a cross-lagged analysis (see Figure 1), permits stronger causal inferences. Burkholder and Harlow (2003) argued cross-lagged analyses take into account temporal precedence (e.g., testing if Wave 1 discrepancies predict Wave 2 depressive symptoms) and competing third variable explanations (e.g., testing if the link between Wave 2 discrepancies and Wave 3 depressive symptoms is explained by Wave 2 depressive symptoms). Extant studies seldom use this rigorous approach to testing reciprocal relations.

Studies of the reciprocal relations model frequently measure perfectionism in a trait-like manner. For example, items are worded so as to suggest long-standing, stable attributes in the discrepancies measure (see Slaney et al., 2001) used by Rice and Aldea (2006). Other measures explicitly instruct participants to focus on "traits and characteristics" in responding to items (e.g., Hewitt & Flett, 1991). In testing the reciprocal relations model, this approach may lead to the overestimation of interindividual stability and the underestimation of complication effects.

Despite clear interest in the link between discrepancies and depressive symptoms (e.g., Wang et al., 2012), a reciprocal relations model involving discrepancies has yet to be tested. Rice and Aldea (2006) tested both the vulnerability and the complication models in their important research on discrepancies and depressive symptoms, but they did not directly test the reciprocal relations model. These authors also collected three waves of data, but they used only two waves of data (i.e., Wave 1 and 3) in their analyses of the vulnerability and the complication model. A more stringent data analytic strategy would arguably involve all three waves of data; variables measured at Wave 1 (unlike those measured in subsequent waves) do not take into account preexisting levels of discrepancies or depressive symptoms, making tests of vulnerability and complication effects involving only two waves of data less stringent (Burkholder & Harlow, 2003). Longitudinal research on discrepancies and depressive symptoms is also scarce. To our knowledge, only six studies use a longitudinal design (i.e., Grzegorek, Slaney, Franze, & Rice, 2004; Rice & Aldea, 2006; Rice, Leever, Christopher, & Porter, 2006; Rice, Richardson, & Clark, 2012; Wang et al., 2012; Wei, Heppner, Russell, & Young, 2006). To advance this literature, we used a longitudinal design and focused on discrepancies—a construct central to counseling psychology (e.g., Slaney et al., 2001) and to perfectionism research (e.g., Grzegorek et al., 2004). Several authors, using various orientations and terms, describe the depressing consequences of believing that one has fallen short of one's own standards (e.g., Horney, 1950; Slaney et al., 2001). Our research builds on this tradition.

We also questioned the following: Do predictions derived from the vulnerability, complication, and reciprocal relations models of perfectionism and depressive symptoms hold once neuroticism is taken in account? Neuroticism (i.e., a dispositional tendency to experience negative emotional states) is a higher order personality trait sharing substantial variance with discrepancies and depressive symptoms (Graham et al., 2010). Research has yet to test if depressive symptoms lead to changes in perfectionism after neu-

roticism is taken into account. Such knowledge is key to understanding the validity of the complication and the reciprocal relations models. The apparent depressive symptoms → perfectionism relationship may be an artifact arising from the variance that depressive symptoms and perfectionism share with the "third variable" of neuroticism.

To address the above shortcomings, we conducted a three-wave, 130-day longitudinal study that tested reciprocal relations between discrepancies and depressive symptoms while (a) adopting a 7-day timeframe for our discrepancies measure, (b) using a cross-lagged data analysis strategy, and (c) controlling for neuroticism. We also studied undergraduates attending university for the first time, a research sample and a time period where changes in personality traits and in depressive symptoms are likely (Lucas & Donnellan, 2011). Our study thus represents a rigorous test of the reciprocal relations model and a needed contribution to discrepancies research.

Although several influential, widely used models and measures of perfectionism exist (e.g., Frost et al., 1990; Hewitt & Flett, 1991), we focused on discrepancies because theory and evidence suggest the concept of discrepancies may represent one central, defining feature of the perfectionism construct (e.g., Rice & Slaney, 2002; Slaney et al., 2001). Research indicates a subjective sense of falling short of one's own standards—while comprising just one particular subpart of the perfectionism construct—is an important subpart of the perfectionism construct (perhaps especially the self-critical perfectionism construct). A confirmatory factor analysis by Blankstein, Dunkley, and Wilson (2008), for example, showed discrepancies loaded strongly ($\beta s > .87$, $p < .001$) onto a latent self-critical perfectionism construct involving socially prescribed perfectionism, concern over mistakes, doubt about actions, and discrepancies. Evidence also indicates discrepancies are centrally involved in depressive symptoms (Rice & Aldea, 2006; Rice et al., 2006). In fact, some evidence suggests that discrepancies add to variance explained in outcomes (e.g., depressive symptoms) above and beyond what is attributable to other traits in the self-critical perfectionism family (e.g., Dunkley, Zuroff, & Blankstein, 2006). Although some earlier efforts to measure discrepancies (e.g., Blatt et al., 1976) were criticized on psychometric grounds (e.g., Coyne & Whiffen, 1995), in the present study, we used the APS-R-D. Extensive research suggests that this scale represents a reliable and a valid means of operationalizing the discrepancies construct (e.g., Rice & Aldea, 2006; Slaney et al., 2001).

As measured by the APS-R-D, discrepancies are global in nature; that is, discrepancies involve a subjective sense of falling short of one's own standards in general and do not focus on one specific goal, task, or domain (e.g., physical appearance). Discrepancies also involve falling short of one's personally held standards as opposed to others' standards. Because measures of self-esteem (e.g., Rosenberg, 1965) often include items with self-evaluative content (e.g., "At times I think I am no good at all"), it is not surprising discrepancies are correlated strongly ($r = .59$, $p < .01$) with self-esteem (see Rice, Ashby, & Slaney, 2007, p. 393). However, discrepancies and self-esteem are not synonymous. Discrepancies focus more on the gap between perceived performance and lofty, unobtainable self-standards (e.g., "I was seldom able to meet my own high standards for performance"; Slaney et al., 2001). Whereas self-esteem focuses more on self-competence (i.e., seeing oneself as a causal agent who brings about desired outcomes; e.g., "I am a capable person") and self-liking (i.e., a sense of worth as a social being; e.g., "I'm secure in my sense of self-worth"; Tafarodi & Milne, 2002). Indeed, some authors regard self-esteem as an outcome of discrepancies instead of a defining feature of discrepancies (Rice & Slaney, 2002).

Hypotheses

Stability

Although not a focal point of our study, we tested mean-level consistency. Mean-level consistency is the degree to which the average trait level of a sample changes over time (Caspi et al., 2005). Building on theory and evidence (McCrae et al., 2000; Rice & Aldea, 2006), we hypothesized that mean levels of discrepancies and neuroticism would be highly stable (showing, at most, small changes over time) and that mean levels of depressive symptoms would be moderately stable (showing, at most, moderate changes over time). Drawing on current views of discrepancies, neuroticism, and depressive symptoms, and evidence of their interindividual stability (Graham et al., 2010; Prenoveau et al., 2011; Rice & Aldea, 2006), we hypothesized autoregressive paths for discrepancies (see Path 5 in Figure 1) and neuroticism (see Path 7 in Figure 1) would be highly stable and autoregressive paths for depressive symptoms (see Path 6 in Figure 1) would be moderately stable.

Given the overlap between discrepancies and self-esteem (e.g., Rice et al., 2007), when making our hypotheses about mean-level consistency and interindividual stability, we considered research on the stability of self-esteem in young adults. This research, which is generally congruent with our predictions about discrepancies, suggests self-esteem is strongly stable over time and more stable than depressive symptoms (Orth, Robins, & Roberts, 2008).

We also used generalizability theory (Shavelson, Webb, & Rowley, 1989) to advance our understanding of the temporal stability of discrepancies. We are only beginning to understand (see Rice & Aldea, 2006) whether discrepancies are best conceptualized as a static *trait* (reflecting highly stable between-persons differences over time), a dynamic *state* (reflecting substantial variability in between-persons differences over time), or a *trait-state* (reflecting a mostly stable trait with some variability in between-persons differences over time). As there was too little evidence to inform specific predictions, these analyses were considered exploratory.

Reciprocal Relations

Drawing upon earlier research (e.g., McGrath et al., 2012), we hypothesized discrepancies and depressive symptoms reciprocally influence one another as shown in Figure 1. Problems in establishing and in maintaining a stable, positive self-image are thought to predispose depressive symptoms (Blatt & Luyten, 2009), perhaps especially if these problems involve a subjective sense of falling short of one's own standards (e.g., Moretti & Higgins, 1999; Slaney et al., 2001). Discrepancies may be part of the premorbid personality of people vulnerable to depressive symptoms—central preoccupations (e.g., unobtainable self-expectations) and recurrent difficulties (e.g., harsh self-

criticism) that people who are at risk for depressive symptoms struggle with in their lives (e.g., Horney, 1950; McGrath et al., 2012). Consistent with this theory and research, we hypothesized discrepancies are both an antecedent of and a contributor to depressive symptoms (see Path 1 in Figure 1).

We also hypothesized discrepancies represent a complication of depressive symptoms (see Path 2 in Figure 1). Depressive symptoms involve a constellation of self-evaluation, behavior, cognition, affect, relatedness, and motivation that we propose activates and exacerbates discrepancies (see also McGrath et al., 2012). For instance, behavioral avoidance and social withdrawal frequently accompany depressive symptoms (A. T. Beck, Rush, Shaw, & Emery, 1979), and such avoidance (e.g., skipping class) and withdrawal (e.g., cancelling plans with friends) may contribute to a sense of being flawed, imperfect, or deficient. Indeed, avoidance and withdrawal may play a key role in actually generating events and experiences that contribute to a negative self-view where failures and disappointments are experienced as salient. Depressive symptoms also involve impaired functioning in occupational, academic, and social domains (Judd, Schettler, & Akiskal, 2002), and such impairments may result in a distressing discrepancy between the actual and the ideal self. Motivational problems that impede goal strivings are also typical of depressive symptoms (A. T. Beck et al., 1979), thereby generating opportunities for self-rebuke (e.g., "I could have done better!") and dissatisfaction. The self-evaluative and the cognitive processes characteristic of depressive symptoms (e.g., a negative, ruminative self-focus) may also encourage detection of shortcomings and a critical stance toward the self where good enough performance is seen as deficient (Mor & Winquist, 2002). In sum, building upon McGrath et al. (2012), we hypothesized discrepancies and depressive symptoms reciprocally influence each other (see Figure 1). As research on perfectionism and depressive symptoms typically shows the magnitude of vulnerability effects exceeds the magnitude of complication effects (Hawley et al., 2006; McGrath et al., 2012; Rice & Aldea, 2006; Shahar et al., 2004), we hypothesized that the effect of discrepancies on depressive symptoms would be relatively stronger than the effect of depressive symptoms on discrepancies.

Incremental Validity

We also explored reciprocal relations between neuroticism and depressive symptoms to see if neuroticism predicted changes in depressive symptoms (see Path 4 in Figure 1) and depressive symptoms predicted changes in neuroticism (see Path 3 in Figure 1). Including discrepancies and neuroticism in the same predictive model allowed us to test the unique and the relative contribution of each variable to depressive symptoms. Some researchers question if lower order personality traits such as discrepancies can predict incremental variance in depressive symptoms beyond higher order personality domains such as neuroticism (Rice et al., 2007). Neuroticism was thus treated as a potential confound and controlled for in our study; Rice and Aldea (2006) did not control for neuroticism. Drawing upon previous work (Graham et al., 2010; Mackinnon et al., 2011), all paths between discrepancies and depressive symptoms in Figure 1 were hypothesized to remain significant after controlling for neuroticism.

Method

Participants

Participants (N = 127; 28 men, 99 women) were all first-year undergraduates attending university for the first time. Participants ranged from 18 to 24 years of age with an average age of 18.31 years (SD = 0.80). Participants self-identified as Asian (5.5%), Arabic (3.9%), Caucasian (81.1%), Black (4.0%), or "other" (5.5%). Participants reported living in Canada for an average of 16.33 years (SD = 5.01). Most participants were undecided about their major in university (62.3%); 5.5% majored in kinesiology, 4.0% majored in nursing, and 28.2% majored in other categories (e.g., history). Participants averaged 13.27 years of formal education (SD = 1.04); 84.9% graduated from high school in the spring of 2010; 31.5% were employed at Wave 1. Our sample resembles other samples recruited from Dalhousie University (Graham et al., 2010).

Procedure

The present study was approved by the Research Ethics Board at Dalhousie University. Participants were recruited via class announcements, online ads, and paper flyers. Our study was conducted in a laboratory. Participants completed three waves of questionnaires during a 130-day period. All questionnaires for Wave 1 were completed within the first 50 days of Semester 1 (i.e., September 9 to October 29). Wave 2 was scheduled during the second half of Semester 1 (45 days after Wave 1). Wave 3 was scheduled at the start of Semester 2 (130 days after Wave 1). Questionnaires were identical across all waves (except demographics were collected at only Wave 1). Participants received phone or e-mail reminders to attend scheduled appointments, and incentives were offered to complete our study (i.e., $25 and 3.0 bonus credits or $55). Each participant was debriefed. Participant attrition was low: 127 participants (100%) completed Wave 1; 125 participants (98.4%) completed Wave 2; and 115 participants (90.6%) completed Wave 3. Participant compliance was high; Wave 2 occurred an average of 44.96 (SD = 4.97) days after Wave 1, and Wave 3 occurred 133.18 (SD = 8.08) days after Wave 1.

Materials

Discrepancies. Discrepancies were assessed with a modified version of Slaney et al.'s (2001) APS-R-D. The modified APS-R-D is a 12-item self-report measure that uses a 7-point scale (1 = *strongly disagree*, 7 = *strongly agree*) such that scores can range from 12 to 84. For all scales in our study, higher scores signify higher levels. Departing from the original APS-R-D instructions, which do not specify a timeframe, our participants were asked to respond to items based on how they felt or behaved "during the last seven days." APS-R-D items were modified to complement a 7-day time frame; for instance, "Doing my best never seems to be enough" was modified to "Doing my best never seemed to be enough." Other sample items in the modified APS-R-D include "I rarely lived up to my high standards," and "I was never satisfied with my accomplishments." Sherry (2011) found high alpha reliability for the

modified APS-R-D (α = .96).[1] In the present study, alpha reliabilities for the modified APS-R-D were high across all waves (αs = .95–.96). Between-persons reliabilities (see p. 925 in Cranford et al., 2006), calculated based on our three waves of data, were also high (i.e., > .99) for the modified APS-R-D. This suggests the modified APS-R-D reliably assessed between-persons differences in discrepancies. Within-person reliabilities (see p. 925 in Cranford et al., 2006), calculated based on all three waves of data, were high (i.e., > .99) for the modified APS-R-D. This indicates the modified APS-R-D reliably assessed within-person change in discrepancies. Test–retest correlations were also high for the modified APS-R-D in our study (*r*s = .78–.81). These test–retest correlations were not different at the $p < .05$ level when we used a Fisher *r*-to-*z* transformation to compare our results to 1-month test–retest correlations in Rice and Aldea (2006; i.e., *r*s = .76–.87). Supporting the validity of the modified APS-R-D, Sherry (2011) found that the modified APS-R-D was correlated moderately to strongly with the original APS-R-D (r = .88), the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977; r = .62), Benet-Martínez and John's (1998) Neuroticism subscale of the Big Five Inventory (BFI-N; r = .39), and the Concern Over Mistakes subscale of Frost et al.'s (1990) Multidimensional Perfectionism Scale (r = .47).

Depressive symptoms. Depressive symptoms were assessed with Radloff's (1977) CES-D. The CES-D is a 20-item self-report measure assessing current levels of depressive symptoms (e.g., "I felt sad"). CES-D items are rated on a 4-point scale (0 = *rarely or none of the time*, 3 = *most or all of the time*). Scores on the CES-D can range from 0 to 60. Participants responded to CES-D items based on how they felt or behaved "during the last seven days." Wei et al. (2007) found high alpha reliability for the CES-D (α = .89). Alpha reliabilities for the CES-D were high across all waves in the present study (αs = .90–.92). Between-persons reliabilities were also high (i.e., >.99) for the CES-D, suggesting the CES-D reliably assessed between-persons differences in depressive symptoms. Additionally, within-person reliabilities were high (i.e., > .99) for the CES-D, indicating the CES-D reliably assessed within-person change in depressive symptoms. Test–retest correlations were also high for the CES-D in the present study (*r*s = .52–.68). Evidence supports the validity of the CES-D. Wu and Wei (2008) found that the CES-D correlated strongly with the Depression subscale of Lovibond and Lovibond's (1995) Depression, Anxiety, and Stress Scales (r = .80).

Neuroticism. Neuroticism was assessed with Benet-Martínez and John's (1998) BFI-N. The BFI-N (e.g., "I see myself as someone who gets nervous easily") is an eight-item self-report measure that uses a 5-point scale (1 = *strongly disagree*, 5 = *strongly agree*), which means scores can range from 8 to 40. Participants responded to BFI-N items based on how they felt or behaved "over the past several years." Selection of this time frame was informed by evidence suggesting neuroticism is highly stable (e.g., Graham et al., 2010). Sherry and Hall (2009) found adequate alpha reliability for the BFI-N (α = .79). In the present study, alpha reliabilities for the BFI-N were adequate across all three waves (αs = .80–.81). Moreover, between-persons reliabilities for the BFI-N were high (i.e., > .99), which suggests the BFI-N reliably assessed between-persons differences in neuroticism. Within-person reliabilities for the BFI-N were also high (i.e., > .99), which indicates the BFI-N reliably assessed within-person change in neuroticism. Test–retest correlations in the present study were high for the BFI-N (*r*s = .75–.80). Research supports the validity of the BFI-N. Benet-Martínez and John found that the BFI-N is correlated strongly with the Neuroticism subscale of Costa and McCrae's (1992) NEO Five-Factor Inventory (r = .76).

Data Analytic Strategy

Mean-level consistency was tested using Tukey's honestly significant difference (HSD) test with Cohen's d as a measure of effect size. Generalizability theory (Cranford et al., 2006) was used to decompose the variance of, and to compute the between-persons reliabilities and the within-person reliabilities for, our study variables. Cross-lagged path analysis was used to test reciprocal relations between (a) discrepancies and depressive symptoms and (b) neuroticism and depressive symptoms. Standard errors and the chi-square for determining model fit were calculated using the MLR estimator in Mplus 6.0 (i.e., maximum likelihood parameter estimates). The MLR estimator provides accurate estimates even if multivariate normality is violated (Muthén & Muthén, 2010). Model fit was assessed with several indices. A well fitting model is suggested by a comparative fit index (CFI) and a Tucker–Lewis index (TLI) around .95, a standardized root-mean-square residual (SRMR) around .08, and a root-mean-square error of approximation (RMSEA) around .06 (Hu & Bentler, 1999).

We compared three models: An unconstrained model (with all paths allowed to freely vary across waves), a constrained model (with the covariances of all paths constrained to equality across waves), and a trimmed model (removing all nonsignificant paths from the constrained model). We used a log likelihood test (i.e., a chi-squared difference test) adjusted for the scaling correction factors of MLR estimation and ΔCFI to compare nested models. If the chi-square difference test is nonsignificant, and the $\Delta\text{CFI} < |.01|$, the models are not significantly different (Cheung & Rensvold, 2002). If the models are not significantly different, the most parsimonious model (i.e., the model with the fewest parameters) should be preferred. Figure 1 illustrates what covariances were constrained to equality in the constrained model. Finally, for the cross-lagged panel analysis, two types of correlated error were specified a priori (see Cole & Maxwell, 2003). The first type is cross-wave correlated error (e.g., error for Wave 2 neuroticism correlated with error for Wave 3 neuroticism). The second type is within-wave correlated error (e.g., error for Wave 2 discrepancies correlated with error for Wave 2 depressive symptoms).

[1] A cross-sectional, supplementary psychometric study was conducted to test the relation between the original, unmodified APS-R and the slightly modified version of the APS-R used in the main study. All participants were recruited using an approach similar to the main study. This psychometric study involved 91 undergraduates. These participants averaged 21.10 years of age (SD = 5.05) and 15.98 years of formal education (SD = 2.72); 85.6% of participants were Caucasian. This supplementary psychometric study is referenced as Sherry (2011) in the text of the main study.

Results

Missing Data

Overall, 4.8% of data were missing, and covariance coverage ranged from 0.85 to 1.00. A nonsignificant Little's Missing Completely at Random test, $\chi^2(61, N = 127) = 64.14$, $p = .37$, suggested our missing data were missing completely at random (R. J. A. Little, 1988). Participants who dropped out of our study did not differ significantly ($p < .05$) from participants who completed our study in terms of measured variables (e.g., discrepancies). Listwise deletion was used to handle missing data for descriptive statistics and bivariate correlations, resulting in a sample of 103 participants. Listwise deletion was not used to handle missing data for path analysis. Instead, a full information maximum likelihood (FIML) estimation approach was used to handle missing data in path analysis, resulting in a sample of 127 participants. FIML was used since its estimates are less biased than estimates generated by listwise deletion or mean imputation (Acock, 2005).

Descriptives

Means, standard deviations, alpha reliabilities, ranges, and bivariate correlations are in Table 1. We compared our means to means observed in past research using Tukey's HSD test with Cohen's *d* as a measure of effect size. Compared to past research (Rice & Aldea, 2006; Rice & Ashby, 2007; Rice & Van Arsdale, 2010), means for discrepancies were slightly elevated at Waves 1 and 2 (5 of 10 post hoc comparisons significant at $p < .05$; *d*s from 0.29 to 0.50) but were not significantly different from prior research at Wave 3 (0 of 5 post hoc comparisons significant at $p < .05$; *d*s from 0.07 to 0.20). Our means for depressive symptoms were similar to earlier work in freshman university student samples (R. Beck, Taylor, & Robins, 2003; Rice & Aldea, 2006; Sargent, Crocker, & Luhtanen, 2006), with only 1 of 15 post hoc comparisons significant at $p < .05$ and *d*s from −0.01 to 0.40. Means for neuroticism were similar to established norms (Benet-Martínez & John, 1998) at all three waves, *d*s from 0.00 to 0.10, *p*s > .05. In sum, means from our study are generally congruent with means from comparable samples of undergraduates.

Mean-level consistency. Post hoc mean comparisons across waves using Tukey's HSD test (see Table 1) and estimates of effect size using Cohen's *d* showed small changes over time. The mean for Wave 3 discrepancies was significantly lower than the means for Wave 1 ($d = 0.23$) and Wave 2 ($d = 0.25$) discrepancies; (b) the mean for Wave 3 depressive symptoms was significantly lower than the mean for Wave 2 ($d = 0.28$) depressive symptoms; and (c) the mean for Wave 3 neuroticism was significantly lower than the mean for Wave 1 neuroticism ($d = 0.31$). These comparisons were generally consistent with our hypotheses that discrepancies and neuroticism would show, at most, small changes in mean levels over time and that mean levels of depressive symptoms would show, at most, moderate changes in mean levels over time.

Bivariate Correlations

Discrepancies, depressive symptoms, and neuroticism were positively and significantly correlated with each other across all waves. As Table 1 illustrates, these correlations involved moderate to large effect sizes (*r*s from .32 to .63; Cohen, 1992). These correlations also resemble previous investigations (Graham et al., 2010; Wei et al., 2007) and suggest merit in testing the hypothesized reciprocal relations model with our data. Correlations also indicate neuroticism is a suitable covariate. Discrepancies, depressive symptoms, and neuroticism were not significantly correlated ($p > .05$) with demographics (i.e., gender, age, major, years of formal education, years living in Canada, and ethnicity). Thus, demographics are not used as covariates in analyses.

Table 1
Means, Standard Deviations, Alpha Reliabilities, Ranges, and Bivariate Correlations

	Wave 1			Wave 2			Wave 3							
Variable	1	2	3	4	5	6	7	8	9	*M*	*SD*	α	Potential range	Actual range
Wave 1														
1. Discrepancies	—									46.19[c]	17.79	.95	12–84	15.0–84.0
2. Depressive symptoms	.63	—								16.10	10.40	.90	0–60	1.0–45.0
3. Neuroticism	.38	.61	—							24.68[c]	5.69	.81	8–40	12.0–38.0
Wave 2														
4. Discrepancies	**.81**	.57	.39	—						46.68[c]	19.40	.96	12–84	12.0–84.0
5. Depressive symptoms	.49	**.62**	.43	.60	—					17.10[c]	11.27	.92	0–60	1.0–48.0
6. Neuroticism	.36	.51	**.79**	.44	.57	—				23.94	6.15	.80	8–40	8.0–38.0
Wave 3														
7. Discrepancies	**.79**	.55	.35	**.78**	.53	.39	—			42.91[a,b]	19.48	.96	12–84	12.0–84.0
8. Depressive symptoms	.54	**.52**	.32	.56	**.68**	.41	.63	—		14.68[b]	10.38	.91	0–60	0.0–45.0
9. Neuroticism	.45	.54	**.75**	.45	.52	**.80**	.51	.58	—	23.41[a]	6.09	.81	8–40	9.0–39.0

Note. Test–retest correlations are in bold. All correlations in Table 1 are statistically significant at $p < .001$.
Superscript letters indicate significant ($p < .05$) post hoc mean comparisons across waves using Tukey's honestly significant difference test: [a] Significantly different from Wave 1. [b] Significantly different from Wave 2. [c] Significantly different from Wave 3.

Variance Partitioning Using Generalizability Theory

Drawing on work by Cranford et al. (2006) and by Shavelson et al. (1989), we used generalizability theory to decompose the variance of our study variables. Using an analysis of variance framework and the VARCOMP procedure in SPSS, we decomposed the variance of each variable into person, wave, and item variability as well as variability explained by interactions among person, wave, and item (see Table 2). We treated person, wave, and item variability as random factors in our generalizability theory analyses.

As Table 2 displays, two components explained most of the variance: person variability and person-by-wave variability. The large proportion of variance in discrepancies (71.9%), depressive symptoms (61.8%), and neuroticism (75.4%) explained by person variability suggests that there are trait-like, between-persons differences across all waves and across all items in our study. In addition, the large proportion of variance in discrepancies (23.0%), depressive symptoms (36.9%), and neuroticism (20.9%) explained by person by wave variability indicates that there are between-persons differences at different waves across all items in our study. Taken together, generalizability theory analyses suggested that, as measured in the present study, discrepancies, depressive symptoms, and neuroticism represent trait-states. Put differently, these three variables were characterized by stable, trait-like individual differences that fluctuated somewhat in their degree of expression over the three waves of our study.

Cross-Lagged Path Analysis

Unconstrained model. As Figure 1 displays, path analysis was used to test reciprocal relations between (a) discrepancies and depressive symptoms and (b) neuroticism and depressive symptoms. The first model we tested was an unconstrained model, where all paths were permitted to freely vary across waves. The unconstrained model fit the data well: $\chi^2(10, N = 127) = 12.59$, $p = .25$; CFI = 1.00; TLI = .99; SRMR = .04; RMSEA = .05 (90% CI [.00, .11]).

Constrained model. We tested a second model constraining the covariance of paths to equality across waves, as specified in Figure 1. If the constrained model does not differ from the unconstrained model based on a chi-square difference test and ΔCFI, the constrained model is preferred as a more parsimonious model. The constrained model fit the data well: $\chi^2(17, N = 127) = 20.26$, $p = .26$; CFI = 1.00; TLI = .99; SRMR = .05; RMSEA = .04 (90% CI [.00, .09]). Model fit did not deteriorate after constraints were applied, ΔCFI = –.001; $\chi^2(7) = 7.67$, $p > .05$, suggesting the constrained model is preferable to the unconstrained model.

In the constrained model, autoregressive paths were significant for discrepancies (βs of .77 for Wave 1 and .90 for Wave 2, $ps < .05$), depressive symptoms (βs of .52 for Wave 1 and .61 for Wave 2, $p < .05$), and neuroticism (βs of .81 for Wave 1 and .89 for Wave 2, $ps < .05$). Discrepancies predicted significant increases in depressive symptoms (βs of .12 for Wave 1 and .15 for Wave 2, $ps < .05$), but depressive symptoms did not predict significant increases in discrepancies (βs of .04 for Waves 1 and 2, $ps > .05$). These findings are congruent with our hypothesis that the effect of discrepancies on depressive symptoms would be relatively stronger than the effect of depressive symptoms on discrepancies. Neuroticism did not predict significant increases in depressive symptoms (βs of .02 for Waves 1 and 2, $ps > .05$); depressive symptoms did not predict significant increases in neuroticism (βs of .02 for Waves 1 and 2, $ps > .05$).

Trimmed model. We tested a third model where nonsignificant paths in the constrained model were omitted, and the model was re-estimated. If the trimmed model does not differ from the unconstrained or constrained model based on a chi-square difference test and ΔCFI, the trimmed model is preferred as a more parsimonious model. The trimmed model fit the data well: $\chi^2(20, N = 127) = 21.00$, $p = .40$; CFI = 1.00; TLI = 1.00; SRMR = .06; RMSEA = .02 (90% CI [.00, .08]). Model fit did not deteriorate compared to the unconstrained model, ΔCFI = .002; $\chi^2(10) = 8.45$, $p > .05$, or the constrained model, ΔCFI = .003; $\chi^2(3) = 0.94$, $p > .05$. Thus, the trimmed model represents the most parsimonious model of the three models tested. Standardized

Table 2
Variance Components for Discrepancies, Depressive Symptoms, and Neuroticism

Source of variance	Discrepancies (12 items)	% of overall variance	Depressive symptoms (20 items)	% of overall variance	Neuroticism (8 items)	% of overall variance
Person	208.98	71.90	71.72	61.81	25.95	75.37
Wave	2.05	0.71	0.95	0.82	0.38	1.10
Item	7.84	2.70	0.43	0.37	0.46	1.34
Person by wave	66.86	23.00	42.79	36.88	7.20	20.91
Person by item	4.35	1.50	0.11	0.09	0.40	1.16
Wave by item	0.10	0.03	0.00	0.00	0.00	0.00
Error	0.49	0.16	0.03	0.03	0.04	0.12
Total	290.67	100.00	116.03	100.00	34.43	100.00

Note. Person = variance due to between-persons differences across all waves and across all items; Wave = variance due to differences between waves across all persons and across all items; Item = variance due to responses to scale items across all persons and across all waves; Person by wave = variance due to between-persons differences at different waves across all items; Person by item = variance due to between-persons differences in responses to scale items across all waves; Wave by item = variance due to differences between waves in responses to scale items across all persons; Error = systematic error (i.e., the Person × Wave × Item interaction) plus random error (i.e., unknown sources of variance; Cranford et al., 2006). When estimating variance components, we found one small (i.e., near zero) negative variance. Brennan (2001) noted that such variances are frequently observed and are usually attributed to sampling error. Negative variances are not possible on a theoretical level. Thus, we followed Brennan's recommendations and set the negative variance to zero.

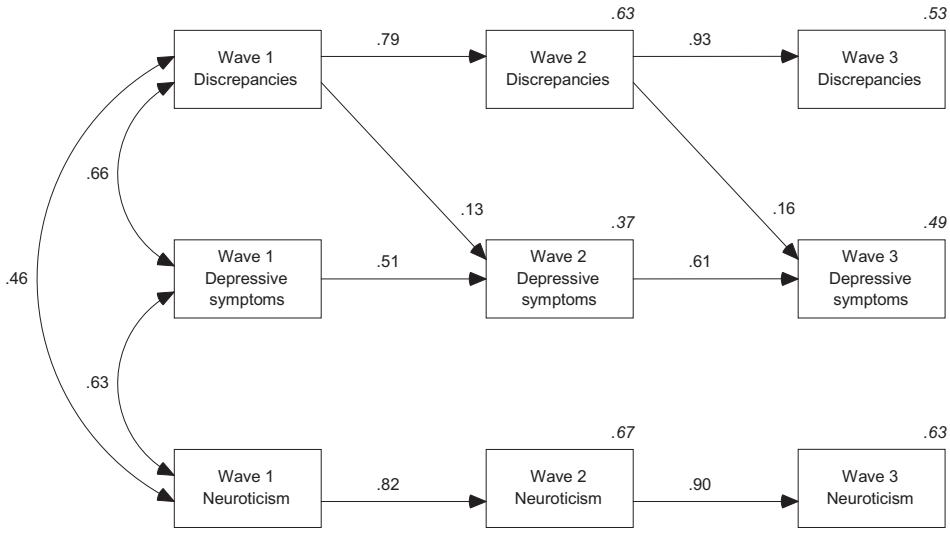

Figure 2. Cross-lagged path analysis with covariances of paths constrained to equality across waves, and non-significant paths omitted (trimmed model). Rectangles represent measured variables. Double-headed black arrows represent significant correlations ($p < .05$). Single-headed black arrows represent significant paths ($p < .05$). Path coefficients are standardized. This means, for example, a one standard deviation increase in Wave 1 discrepancies predicted a 0.13 standard deviation increase in Wave 2 depressive symptoms. Horizontal arrows represent autoregressive paths, and diagonal arrows represent cross-lagged paths. Italicized numbers in the upper right hand corner of each rectangle represent the proportion of variance accounted for by associated exogenous variables (i.e., R^2 values). Though unstandardized path coefficients were constrained to equality, standardized path coefficients may still vary slightly (see Burkholder & Harlow, 2003). In the interest of clarity, error terms are not displayed.

path coefficients and R^2 values for the trimmed model are presented in Figure 2.[2]

Interindividual stability. Autoregressive paths (e.g., Wave 1 discrepancies to Wave 2 discrepancies) in the trimmed model tested interindividual stability (see Figure 2). As hypothesized, autoregressive paths for discrepancies and neuroticism were significant and highly stable, and autoregressive paths for depressive symptoms were significant and moderately stable.

Reciprocal relations. Cross-lagged paths tested whether a change in one variable (e.g., Wave 2 discrepancies) was related to a change in another variable (e.g., Wave 3 depressive symptoms). As hypothesized, discrepancies predicted increases in depressive symptoms in the trimmed model (see Figure 2). Counter to our hypotheses, depressive symptoms did not predict increases in discrepancies and these null paths were trimmed (see Figure 2). The hypothesized pattern of reciprocal relations was therefore not found in our study, though discrepancies did confer vulnerability to depressive symptoms over time. Neuroticism did not predict significant increases in depressive symptoms, and depressive symptoms did not predict significant increases in neuroticism; these null paths were trimmed (see Figure 2). In the present study, neuroticism was neither an antecedent nor a consequence of depressive symptoms.

Bootstrap analyses were also utilized to test the statistical significance of the indirect effect of Wave 1 discrepancies on Wave 3 depressive symptoms (see Figure 2). The statistical significance of the indirect effect was calculated using bias-corrected bootstrapping with 20,000 resamples. If the 95% bootstrapped confidence interval (95% CI) for this indirect effect does not contain zero, this indirect effect is significant at $p < .05$ (T. D. Little, Preacher, Selig, & Card, 2007). Results indicated Wave 1 discrepancies had a significant indirect effect on Wave 3 depressive symptoms through Wave 2 discrepancies and Wave 2 depressive symptoms, 95% CI [.04, .37].

Incremental validity. As hypothesized, the discrepancies → depressive symptoms paths in Figure 2 were significant even after controlling for neuroticism. To expand, when paths from neuroticism to depressive symptoms and paths from depressive symptoms to neuroticism were added to Figure 2, these four paths were nonsignificant—and the discrepancies → depressive symptoms paths remained significant as shown in Figure 2. This suggests discrepancies are not redundant with neuroticism when predicting depressive symptoms in the present study. We also questioned if the depressive symptoms → discrepancies paths in Figure 2 were nonsignificant because neuroticism was taken into account. To test

[2] Wang (2010, p. 188) argued that several APS-R-D items have affective content (e.g., "frustrated"), which may confound discrepancies and emotional reactions to discrepancies. In our study, two doctoral-level psychologists reviewed APS-R-D items and agreed that Items 1, 5, 6, 8, 10, 11, and 12 contained affective content. In supplementary analyses, we calculated a new total score for discrepancies omitting these seven items and retested the trimmed model. This new model fit the data well: $\chi^2(20, N = 127) = 26.30$, $p = .40$; CFI = .99; TLI = .98; SRMR = .06; RMSEA = .05 (90% CI [.00, .10]), and all paths in Figure 2 remained significant ($p < .05$). Thus, our results were virtually unchanged when these affectively-laden items were dropped.

this possibility, we removed the neuroticism variable from Figure 2 and found all paths from depressive symptoms to discrepancies remained nonsignificant, (βs = .03, $p > .05$). This indicates the nonsignificant paths from depressive symptoms to discrepancies in Figure 2 were not due to the influence of neuroticism.[3]

Discussion

The present study represents one of the most extensive tests of the connection between discrepancies and depressive symptoms conducted to date. We found strong to moderate mean-level consistency for discrepancies, depressive symptoms, and neuroticism, thereby generally supporting our predictions. As hypothesized, discrepancies and neuroticism showed strong interindividual stability whereas depressive symptoms showed moderate interindividual stability. Generalizability theory analyses also indicated that discrepancies and neuroticism are more temporally stable than depressive symptoms. Seen through the lens of generalizability theory analyses, discrepancies may be conceptualized as a trait-state where people are highly consistent in their rank order on discrepancies, but still fluctuate somewhat in the level of discrepancies they experience at a particular point in time. Congruent with our hypotheses, discrepancies predicted changes in depressive symptoms, even after controlling for neuroticism. Contrary to our hypotheses, depressive symptoms did not predict changes in discrepancies.

Reciprocal Relations Model of Discrepancies and Depressive Symptoms

Stability. We found only small changes in the mean-level consistency of discrepancies over time. This result is consistent with Rice and Aldea (2006), who used the original APS-R-D. In particular, small declines in the average level of discrepancies, neuroticism, and depressive symptoms were found at Wave 3 of our study. Wave 3 of the present study occurred following a (potentially restorative) break of several weeks duration, which suggests that mean levels of discrepancies, neuroticism, and depressive symptoms may, in small ways, fluctuate along with the challenges and the breaks of the academic year (see also Rice & Aldea, 2006, pp. 210–211).

As in past work (e.g., Rice, Richardson, & Clark, 2012), and consistent with hypotheses, both test–retest correlations and autoregressive paths suggested discrepancies have high interindividual stability. These findings are generally consistent with Strauman (1996), who used an interview version of Higgins, Bond, Klein, and Strauman's (1986) Selves Questionnaire, which measured perceived discrepancies between the actual and the ideal self. Strauman (1996) reported that this measure of discrepancies (which appears to differ substantively from the item content of the APS-R-D) was temporally stable, with a 3-year test–retest reliability of $r = .42$ ($p < .01$). Our study also complements a wider literature suggesting traits in the self-critical perfectionism family have high interindividual stability (Rice & Dellwo, 2001).

As hypothesized, neuroticism showed high interindividual stability and depressive symptoms showed moderate interindividual stability in a manner congruent with past research (e.g., Graham et al., 2010). Accounting for the interindividual stability of variables in our cross-lagged analyses helped rule out pre-existing levels of variables as a competing third variable explanation for our findings (e.g., the link between Wave 2 discrepancies and Wave 3 depressive symptoms is not completely explained by pre-existing levels of Wave 1 depressive symptoms).

The degree to which a personality trait (e.g., discrepancies) appears temporally stable depends, to a certain extent, on how temporal stability is defined, measured, and analyzed (Sanislow et al., 2009). Our generalizability theory analyses offered both a novel and a nuanced approach to understanding the temporal stability of discrepancies that was not used in Rice and Aldea (2006). These analyses suggested discrepancies may represent a trait-state (i.e., a mostly stable trait with some variability in between-persons differences over time). Discrepancies did not appear to be set like plaster. Our results suggest people high in discrepancies have a stable, trait-like tendency to fall short of their own standards that also fluctuates somewhat in its degree of expression from one time period to the next (see also Sherry & Hall, 2009).

Variability in discrepancies seems likely during the students' 1st year of university, perhaps especially if people enter university with unrealistic expectations and self-critical tendencies. These expectations and tendencies may not be especially problematic—so long as everything is perfect. However, life is seldom perfect when attending university. Attending university for the first time can involve serious academic and social challenges such as demanding classes with negative feedback and fitting into new social networks (American College Health Association, 2009). People high in discrepancies are also more likely to receive lower grades (Grzegorek et al., 2004) and to encounter social problems (Slaney, Pincus, Uliaszek, & Wang, 2006) during university. Such challenges and imperfections may lead to variability in discrepancies over time.

Depressive symptoms showed moderate stability along with substantial variability when analyzed using generalizability theory analyses. This finding is consistent with past research and conceptualizations of depressive symptoms as a chronic, but episodic, problem with a relapsing and remitting course (Prenoveau et al., 2011). Using generalizability theory analyses, we also found neuroticism showed stability coupled with some variability. This finding is congruent with research suggesting variation in neuroticism occurs over time (Graham et al., 2010).

Reciprocal relations. We used a reciprocal relations model that simultaneously tested predictions from both vulnerability and complication models. Our study extends past work (e.g., Rice & Aldea, 2006) by conducting a very stringent test of discrepancies as a vulnerability factor for depressive symptoms (e.g., cross-lagged path analysis and controlling for neuroticism). Traits in the self-critical perfectionism family are viewed as vulnerability factors for depressive symptoms by several researchers (McGrath et al., 2012). Consistent with this view, we found that discrepan-

[3] Though not included in the main text to conserve space, similar results were found when using the extraversion subscale of the Big Five Inventory (Benet-Martínez & John, 1998) in place of neuroticism as a covariate. In this model, extraversion was negatively correlated with discrepancies ($r = .28$, $p = .001$) and depressive affect ($r = -.42$, $p < .001$) at Wave 1, and cross-lagged paths to and from extraversion were nonsignificant (βs from –.05 to .01, ps $> .05$). These analyses are available upon request from the first author.

cies—one important part of the self-critical perfectionism family of traits (Blankstein et al., 2008)—predicted changes in depressive symptoms in the hypothesized manner (see Figure 2). This finding, considered with past research (Rice & Aldea, 2006), suggests the vulnerability model is a plausible way of conceptualizing the discrepancies-depressive symptoms connection. As in past research (e.g., McGrath et al., 2012; Rice & Aldea, 2006), and congruent with our hypotheses, we also found the effect of discrepancies on depressive symptoms was relatively stronger than the effect of depressive symptoms on discrepancies.

The present study complements a long tradition of theory and research noting the depressing consequences of believing that one has fallen short of one's own standards (Horney, 1950; Slaney et al., 2001). People high in discrepancies appear to have personality traits that increase their vulnerability to depressive symptoms: Highly valued, but unattained, unrealistic self-expectations, incongruence between the actual and the ideal self, and a strong sense of being flawed, imperfect, or deficient. Establishing and maintaining a stable, positive self-image may be especially challenging during the 1st year of university, a period of transition often involving both academic (e.g., lower grades) and interpersonal stressors (e.g., romantic break-ups; Le, Dove, Agnew, Korn, & Mutso, 2010). First-year undergraduates in our study, who emerged from high school with academic records strong enough to gain admission to a top university, may have found such stressors unfamiliar and depressing. The magnitude of our observed vulnerability effects (e.g., Wave 2 discrepancies to Wave 3 depressive symptoms) was small, and longitudinal research is needed to test whether the discrepancies–depressive symptoms link is stronger in the context of stressors (e.g., academic or acculturative stress). Cross-sectional data suggesting discrepancies interact with stressors to predict depressive symptoms (Rice, Choi, Zhang, Morero, & Anderson, 2012; Wei et al., 2007) provide a foundation for such research.

Bootstrap analyses also indicated Wave 1 discrepancies had a significant indirect effect on Wave 3 depressive symptoms via Wave 2 discrepancies and Wave 2 depressive symptoms. These results suggest an unfolding temporal relationship between discrepancies and depressive symptoms in our study. It seems the perceived flaws, imperfections, and deficiencies of first semester can have a lasting, negative influence on 1st-year undergraduates, indirectly affecting their depressive symptoms during second semester.

Counter to our hypotheses, complication effects were not found in our study. Depressive symptoms did not precede and produce changes in discrepancies, a finding consistent with Rice and Aldea (2006). Future research should test whether depressive symptoms indirectly affect discrepancies through negative events (e.g., job loss). When considering all traits belonging to the self-critical perfectionism family, support for complication effects is mixed, with studies both supporting (Cox & Enns, 2003; McGrath et al., 2012; Shahar et al., 2004; Zuroff et al., 1999) and failing to support (Hawley et al., 2006) complication effects. Divergent findings in this literature may stem from various factors, including different samples (e.g., psychiatric vs. undergraduate), age groups (e.g., adolescent vs. adult), measures (APS-R-D vs. socially prescribed perfectionism subscale), timeframes for measures (7 days vs. over the past several years), and measurement schedules (1-week measurement vs. 1-year measurement intervals). In addition, the course, severity, expression, impairment, comorbidity, and phenomenology of depressive symptoms are heterogeneous (Klein, 2008). Some forms of depressive symptoms may influence personality traits more than other forms (e.g., introjective forms of depressive symptoms may predict changes in discrepancies; Blatt et al., 1976). More research is needed (including direct replications of past studies) before we can endorse, refine, or dismiss the complication model.

Neuroticism and depressive symptoms. To our knowledge, no other study has tested reciprocal relations between neuroticism and depressive symptoms. Our results suggest that neuroticism is a concomitant of depressive symptoms (see Table 1), but not an antecedent or a consequence of depressive symptoms (see Figure 2). Ormel, Rosmalen, and Farmer (2004, p. 906) argued "neuroticism is not an explanatory concept in the aetiology of psychopathology, since it measures a person's characteristic level of distress over a protracted period of time." Our results are generally consistent with Ormel et al.'s assertion: Once baseline, pre-existing levels of depressive symptoms were accounted for, neuroticism did not confer vulnerability to depressive symptoms. The null neuroticism → depressive symptoms paths are also understandable from another perspective. Evidence suggests neuroticism interacts with life stress to predict depressive symptoms (Brown & Rosellini, 2011)—a moderator not included in our study. A diathesis-stress model may be needed to explain the neuroticism–depressive symptoms connection. We also did not find complication effects involving neuroticism, which is consistent with a recent review by Klein, Kotov, and Bufferd (2011), who concluded there is little evidence showing that depressive symptoms come before and contribute to neuroticism (for an exception to this conclusion, see De Fruyt, Van Leeuwen, Bagby, Rolland, & Rouillon, 2006).

Incremental Validity

The discrepancies → depressive symptoms paths in Figure 2 were significant even after controlling for neuroticism, supporting the incremental validity of the discrepancies construct. Our study thus extends Rice and Aldea (2006), who found a similar vulnerability effect but who did not control for neuroticism. It is notable that the discrepancies → depressive symptoms path held after controlling for neuroticism, as neuroticism is a higher order personality trait that strongly overlaps with depressive symptoms (Graham et al., 2010). Our results suggest discrepancies may be a unique, lower order vulnerability factor for depressive symptoms that is not fully captured by neuroticism. In contrast, Rice et al.'s (2007) cross-sectional study found that, after controlling for neuroticism, conscientiousness, and other perfectionism variables, discrepancies were not related to depressive symptoms. Differences between our study and Rice et al.'s study may result from using different research designs (i.e., longitudinal vs. cross-sectional), measures of neuroticism (i.e., BFI vs. NEO Five-Factor Inventory), or data analytic strategies (i.e., Rice et al., 2007, included discrepancies, neuroticism, and nine covariates when predicting depressive symptoms). More generally, our study joins and extends a wider research literature suggesting traits in the self-critical perfectionism family predict depressive symptoms once neuroticism is taken into account (for a review, see Zuroff, Mongrain, & Santor, 2004).

Limitations and Future Directions

Our predominantly female, young, Caucasian, Westernized, educated sample raises questions about the representativeness and the generalizability of our data. Tests of vulnerability and complication effects may be harder to detect in such samples where levels of discrepancies and depressive symptoms are relatively homogenous. Future studies involving clinical samples are needed. Our study also involved a small sample size. Future researchers should recruit a larger sample and conduct growth curve analyses (e.g., estimating individual trajectories of depressive symptoms for people high and low in discrepancies across measurement occasions). Our three-wave, 130-day longitudinal design represents just one possible measurement schedule. Time lags between waves (e.g., 45 days vs. 1 year) may impact results. The null depressive symptoms → discrepancies finding may have occurred because too much (or too little) time passed between waves. More short- and long-term longitudinal designs with three or more waves of data are needed to test reciprocal relations between discrepancies and depressive symptoms. In addition, dynamic, momentary transactions between discrepancies and depressive affect might be captured using an experience sampling design (Sherry & Hall, 2009). Future studies might also adopt a person-centered approach and test the temporal stability of clusters of perfectionists. Finally, as discrepancies represent only one specific subpart of the perfectionism construct, it will be important for future research to test whether discrepancies predict changes in depressive symptoms above and beyond other perfectionism dimensions (e.g., concern over mistakes).

Evidence clearly supports the reliability and the validity of the APS-R-D (e.g., Slaney et al., 2001). There are, however, other ways to operationalize the discrepancies construct. Some authors (e.g., Boldero, Moretti, Bell, & Francis, 2005) argue that more idiographic measures (e.g., open-ended questions or narrative interviews) capture unique aspects of the discrepancies construct that are not measurable via self-report, Likert-type questionnaires. Future research might test whether the discrepancies–depressive symptoms link is observable using different methods.

We used a slightly modified version of the APS-R-D. This modified scale evidenced strong psychometric properties (e.g., alpha reliability, between-persons reliability, within-person reliability, test–retest reliability, convergent validity, and predictive validity) in both our main study and our supplementary psychometric study. Ultimately, however, less is known about the psychometric properties of our modified version of the APS-R-D. Using a 7-day timeframe for the APS-R-R might have contributed to more instability in discrepancies scores. In addition, our instructions directed participants to focus on how they felt or behaved "over the past several years" when responding to BFI-N items. Adopting a long-term timeframe might have resulted in overestimation of stability effects and underestimation of complication effects for neuroticism.

Implications for Clinical Practice

Our study indicates depressive symptoms do not complicate assessment of discrepancies. For clinicians conducting assessments, this is notable as it suggests APS-R-D scores are not unduly altered by past depressive symptoms (at least when assessing undergraduates with mild to moderate depressive symptoms). Nevertheless, clinicians still need to consider if scores on other perfectionism measures are altered by severe depressive symptoms (see Cox & Enns, 2003).

If the complication model was supported in our research, we might have suggested that clinicians pursue eliminative treatment goals (e.g., reducing depressive symptoms via cognitive behavioral therapy) to alleviate discrepancies. When using the complication model to inform treatment, depressive symptoms (e.g., impaired functioning) are conceptualized as contributing to a distressing discrepancy between the actual and the ideal self, and this discrepancy might be ameliorated by reducing depressive symptoms.

In contrast, our results suggest discrepancies are a unique, persistent vulnerability factor for depressive symptoms that warrant consideration as a treatment target when attempting to prevent the occurrence or the relapse/recurrence of depressive symptoms. Using the vulnerability model to inform treatment, our results indicate a reduction in discrepancies during treatment may lead to a corresponding reduction in depressive symptoms. For example, teaching people high in discrepancies to sooth themselves using nurturing, compassionate self-talk and imagery might lower their discrepancies (Kelly, Zuroff, & Shapira, 2009), thereby reducing problems with or vulnerability to depressive symptoms. That said, we concede our remarks about treatment go beyond our data and should be viewed with caution and as impetus for more research.

Concluding Remarks

Knowing whether discrepancies are an antecedent of depressive symptoms, a complication of depressive symptoms, or both is critically important to understanding the discrepancies–depressive symptoms connection and to guiding theory, research, assessment, and intervention in this area. Yet, longitudinal research on discrepancies and depressive symptoms is scarce. Our three-wave longitudinal study indicated that discrepancies are a uniquely important vulnerability factor for depressive symptoms in undergraduates attending university for the first time. Harsh self-criticism and unobtainable self-expectations involving a distressing sense of imperfection may be part of the premorbid personality of people vulnerable to depressive symptoms.

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