

Honors Thesis

Title: Cognitive Reactivity and Affect

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Abstract

Cognitive Reactivity (CR), an established diathesis for depression, has been defined as the strength of association between displeasure and dysfunctional attitudes within person. However, displeasure could encompass a variety of distinct types of affect, among which only cognitive reactivity to sadness (CR-SAD) has been studied in prior research. In the current study, college student participants were recruited to validate a new daily diary measure of CR that includes dynamic assessments of CR-SAD, as well as Cognitive Reactivity to Negative Affect (CR-NA) and Cognitive Reactivity to Positive Affect (CR-PA). Three results emerged: (1) the daily diary measure of CR-SAD (CR-SAD-DD) converged with other measures of CR-SAD; (2) CR-SAD-DD was associated with depressive symptoms; (3) naturally occurring NA and PA were found to uniquely contribute to emergence of dysfunctional cognitions over and above sadness. The current study was the first to combine literature in CR and affective structures, and its findings may have profound implications in the diagnosis and treatment of depression and other psychopathologies.

Keywords: Cognitive reactivity, Affect, Depression, Mood disorder

Cognitive Reactivity and Affect

Introduction

Clinical observations and research have shown that depressed individuals are more likely to hold negatively biased cognitions toward their environment, themselves, or their future (e.g., Beck, 1967; Kovacs & Beck, 1978). For such individuals, those maladaptive cognitions are usually intensified by sad mood (Miranda & Persons, 1988), suggesting that depressed individuals have high cognitive reactivity (CR) to sad mood (Ingram, Miranda, & Segal, 1998; Segal & Ingram, 1994). Based on literature about the structure of affect (Watson & Tellegen, 1985), we suspect that negative affect (i.e., unpleasant affect with high arousal) and lack of positive affect (i.e., pleasant affect with high arousal) may also contribute to the development of maladaptive cognitions typically seen in depression. With the validation of a new ecological measure of CR, the current study examined CR to other naturally occurring negative moods in addition to sadness and their associated outcomes.

Studying psychological vulnerability to depression is important for understanding the onset, maintenance, and treatment of mood disorders. One of the most influential models of vulnerability to depression is Beck's cognitive model (Beck, 1967; Beck, Rush, Shaw, & Emery, 1979; Kovacs & Beck, 1978). The model suggests that early stressful life experiences contribute to the development of dysfunctional cognitive structures, which result in a tendency to interpret later life events in ways that resemble their earlier maladaptive cognitive styles. Over time, these maladaptive cognitive styles form dysfunctional cognitive schemata, which in turn become a cognitive diathesis of depression. Early work in cognitive psychology revealed that some cognitions are especially accessible in specific affective states, which serve as their retrieval cues (Bower, 1981; Johnson & Magaro, 1987). In a theory called the differential activation hypothesis

Teasdale (1988) proposed that the activation of dysfunctional cognitions may depend on affective state. According to this theory, for people prone to depression, dysfunctional cognitions become associated with negative affect following some stressful life events. Similar negative affect that appear later in their lives will activate their maladaptive cognitive schemata, thus prolonging the negative affective state to form a long, stable mood state of depression.

The differential activation hypothesis has led to the proposal of the construct called *cognitive reactivity* (CR), defined as the change in endorsement of dysfunctional beliefs in response to mild mood fluctuations (Williams, Van der Does, Barnhofer, Crane, & Segal, 2008; see also: Ingram, Miranda, & Segal, 1998; Segal & Ingram, 1994). More recently, Cole et al. (2014) noted that CR is a dynamic construct, reflecting the within-person strength of association between negative affective states and the triggering of maladaptive cognitions in a given individual. Most previous research has used a mood-induction methodology to study CR, in which subjects' cognitions are first measured when they are in a neutral affective state and measured again when they are mildly sad after some brief mood-induction procedure (e.g., Miranda & Persons, 1988; Ingram, Bernet, & McLaughlin, 1994; Segal, Kennedy, & Gemar, 2006). Studies using mood-induction have demonstrated that CR is abnormally high in people with a history of depression (Miranda & Persons, 1988) and is found to be a reliable predictor of depression relapse (Segal et al., 2006). Further, in Segal, Gemar, and Williams' (1999) study, cognitive therapy was found to reduce cognitive reactivity, whereas pharmacotherapy did not (Segal, Gemar, and Williams, 1999; Cladder-Micus, van Aalderen, Donders, Spijker, Vrijsen & Speckens, 2018). Though the evidence for the relation between cognitive reactivity and depression is not central to my proposed study and consequently not discussed in depth here,

such evidence is well summarized in two review articles (Scher, Ingram, & Segal, 2005; Segal & Ingram, 1994).

Although prior studies suggest that an elevated cognitive response to negative mood fluctuation is a diathesis for depression, there exists a discrepancy in how “mild mood fluctuation” was defined and operationalized. By definition, mood fluctuation could mean a small change in any sort of affect; affect such as “anxiety,” “anger,” “lethargy,” and “sadness” may all be the sources of such fluctuation. As a result, different researchers could adopt different variations of CR to serve their own research purposes, contributing to the jingle fallacy of the construct. Interestingly, despite the openness of the definition in “mild mood fluctuation”, classic studies in CR seemed to consensually focus on sadness as the mood that triggers subjects’ cognitive reactions, thus restricting the construct to capturing just the cognitive response to a relatively specific affect. In most studies that used mood induction to measure CR (e.g., Teasdale & Dent, 1987; Miranda & Persons, 1988; Ingram et al., 1994; Miranda et al., 1998; Segal, Kennedy, & Gemar, 2006), increase in sadness was assumed to be the goal of the mood induction procedure. Given the amount of studies of CR focusing on sadness, it is of no surprise that a widely-used questionnaire measure of CR, the Leiden Index of Depressive Sensitivity (LEIDS; Van der Does, 2002) also prompts subjects to imagine themselves being in a mildly sad mood. Considering the consensual assumption that sadness is the affect that triggers depression-related maladaptive cognitions, we argue that prior studies of CR focused on cognitive reactivity to *sadness* (CR-SAD) rather than cognitive reactivity to *general negative fluctuation in affect*. However, other types of affect may relate to depression. For example, it has been shown that tendency to express anger is a common symptom of depression, especially in children (Crowe, Ward, Dunnachie, & Roberts, 2006; Fava, Hwang, Rush, Sampson, Walters, & Kessler, 2010).

The lack of research on how fluctuations in affect other than sadness may influence dysfunctional thoughts motivated a more in-depth examination of CR and affect.

In order to clarify the discrepancy in definition and operationalization of mild mood fluctuation in CR, we need to understand which of the affective states are relevant to the construct of CR and its nomothetical span. To our knowledge, no existing research in CR has considered CR with respect to the complexity of human affect, a surprising omission given how significant a role that affect plays in depression. However, there are many relations between affective phenomena and CR that can be observed in the affiliated literature, and they should be noted here.

Structure of Affective Phenomena and Their Relation to Cognitive Reactivity

Factor analysis of self-reported descriptions of affect¹ usually reveals two independent dimensions, namely valence (or pleasure-displeasure) and arousal (or activation-deactivation). Valence is the subjective summary of how “well” one is doing. Arousal is the subjective sense of mobilization or energy. Various combinations of these two dimensions underlie linguistic descriptors of emotions (e.g., happy, angry, sad) that fall into the Cartesian space of affect. Many authors also use the name “circumplex” for it due to the arrangement of common descriptors in a roughly circular way (see *Figure 1* for a visual layout of the circumplex of core affect). For instance, high positive levels on both valence and arousal may result in “enthusiastic” or “excited” affective states, whereas high level of arousal combined with negative valence may

¹ Russel and Feldman Barrett (1999) noted that researchers in psychology often used the words “affect,” “mood,” and “emotion” interchangeably, and the same word used by different writers might refer to different constructs due to the lack of a clear definition for each. Because of this, they proposed to distinguish between “core affect,” defined as the most elementary consciously accessible affective feelings and their neurophysiological counterparts, from “prototypical emotional episode,” defined as a complex set of core affect and behaviors concerned with a specific object. Due to its complexity and dependency on circumstances, prototypical emotional episodes are yet to be described by a universal model. Therefore, in this paper, we will only focus on “core affect,” or sometimes simply “affect,” as the construct of interest, so that the cognitive component of it will be left out.

result in “nervous” or “hostile” states. Although different researchers (e.g., Feldman Barrett & Russel, 1998; Watson & Tellegen, 1985; Larson & Diener, 1992; Thayer, 1989) appear to take different approaches in describing the dimensions of affect, it is increasingly well acknowledged that their models use different labels for essentially the same underlying constructs (Mayer & Gaschke, 1988; Russel & Feldman Barrett, 1999; Ekkekakis, 2013, pp. 65-67).

As discussed in the previous section, classic studies in CR focused on sadness as the affective precursor for the activation of dysfunctional attitudes (e.g., Teasdale & Dent, 1987; Miranda & Persons, 1988; Ingram et al., 1994; Miranda et al., 1998; Segal et al., 2006). In the circumplex of core affect, sadness refers to the negative extreme of the valence dimension with slightly low level of arousal. Surrounding it are high-arousal displeasure (e.g., stressed, nervous) and low-arousal displeasure (e.g., lethargic, fatigue). Watson and Tellegen (1985) used the phrases positive affect (PA) and negative affect (NA)² to describe the high-arousal ends of pleasure and displeasure, because they found words describing affect with neutral level of arousal were rarely used in human languages.³ In 1999, Watson and Clark refined their model into a hierarchical structure based on results of factor analysis. In their refined model, PA and NA remain as general dimension scales, whereas sadness along with six other affect descriptors compose basic emotion scales. One important advantage of their PA-NA model is that PA and NA respectively correspond to Gray’s (1987) behavior activation and behavior inhibition system, which are neurophysiological systems frequently used to explain the cause of a variety of mental disorders. Moreover, the PA-NA division helps to distinguish depression and anxiety, where the lack of PA characterizes depression and high level of NA characterizes anxiety (Tellegen, 1985).

² Sometimes also called “positive activation” and “negative activation.”

³ Psychometrically, using PA and NA as two dimensions of core affect accounted for over 90% of variance accounted for by valence and arousal (Russel & Feldman Barrett, 1999), making it a valid varimax rotation of the empirically driven valence-arousal dimensional model of core affect.

Perhaps for this reason, their model and measure have been frequently used to study abnormal affective phenomena.

In the current study, we used Watson and Clark's (1999) hierarchical model of affect as our theoretical basis. Although evidence of CR-SAD as a diathesis for depression has been well documented, it remains unclear how PA and NA as two general dimensions of affect may trigger depression-related cognitive reactions as well. Because depressed individuals were less likely to experience PA and more likely to experience NA (Tellegen, 1985), we are interested in knowing how cognitive reactivity to low level of PA (CR-PA) and cognitive reactivity to high level of NA (CR-NA) may provide added value to the proven diathesis of depression in CR-SAD.

A New Ecological Measure of Cognitive Reactivity

Because of our expanded view of CR that include triggering of dysfunctional cognitions by negative affect other than sadness, traditional procedures to measure CR such as laboratory sad-mood provocation and self-report questionnaire are insufficient to induce the various types of affect people may experience. Additionally, some researchers in CR have suggested that naturally occurring affect might be more relevant to individuals in triggering negative cognitions than lab-induced affect (Wenze, Gunthert, & Forand, 2006, 2010; Scher, Ingram, & Segal, 2005). For example, studies that aimed to induce sadness by exposing subjects to sad music or films (Teasdale & Dent, 1987; Miranda et al., 1998; McCabe et al., 2000) may induce unnatural emotions in subject that did not typically listen to sad music or watch sad films. Besides, as Miranda and Persons (1988) pointed out in the limitations of their study, following an affect-induction, subjects' report of their affect might be influenced by demand characteristics. They might be afraid that showing no change in affect after watching a tragic video of someone else would make them look apathetic. To obtain a realistic picture of subjects' daily mood

fluctuations, we decided to develop a new measure of CR through a daily diary methodology. The details will be explained in the Method section.

The Current Study

Relating the structure of affect to our previous discussion, we may now gain a clearer look at CR. CR, in essence, should be operationalized as the within-person slope of a linear model predicting activation level of dysfunctional attitudes from a function of the degree of negative shift in affect. Prior studies (e.g., Teasdale & Dent, 1987; Miranda & Persons, 1988; Ingram et al., 1994; Miranda et al., 1998; Segal, Kennedy, & Gemar, 2006) assumed (usually implicitly) the negativity of affect to be unidimensional by only considering the effect of sadness in triggering maladaptive cognitions. This is insufficient in capturing some potentially significant distinctions among different forms of “negative affect.” Specifically, low-arousal negative affect such as feeling “depressed” or “lethargic” has been shown to differ from high-arousal affect such as feeling “stressed” or “frustrated” (Tellegen, 1985). Consequently, a unidimensional representation of displeasure or negative affect in the model of cognitive reactivity tells nothing about the role of arousal in the activation of dysfunctional attitudes. The current study aims to use a two-dimensional representation of affect (e.g., NA and PA) originally proposed by Watson and Tellegen (1985) to study the nature of CR by using our newly developed daily diary approach.

We had four goals for the current study. The first two goals, taken together, aim to validate our daily diary measure of CR-SAD. Our first goal was to show the daily diary measure of CR-SAD converges with other existing measures in literature. Our second goal was to test whether CR-SAD measured with daily diary was indeed associated with depressive symptoms (DS) as the theory suggests. The last two goals collectively explored CR to affect other than

sadness. Our third goal was to test whether NA and PA have unique roles that are different from the role of sadness in activating dysfunctional attitudes, a relation potentially omitted by previous studies of CR. If so, each of NA and PA will be associated with dysfunctional cognitions in our sample controlling for other types of affect. The fourth goal was to test whether those associations, namely CR-NA and CR-PA, would each explain an additional amount of variance in DS above and beyond CR-SAD.

Method

Design and Procedure

Participants began by answering demographic questions as well as completing the Leiden Index of Depressive Sensitivity-Re-revised (Van der Does, 2002) and Beck Depression Inventory II (BDI-II; Beck, Steer, & Garbin, 1996) online at their convenience. Within three days of finishing these tasks, they attended a laboratory session during which they completed the scenario-based measure of CR and underwent a mood induction procedure. Those will be described in detail in Measures section. After their lab visit, they completed a brief online survey every day for 10 days. On each daily survey, participants were asked to describe the most negative event that they experienced in the past 24 hours. They were asked to recall⁴ their mental state immediately following the event, and then answer the questions about their affect in reaction to those events and their cognitions in reaction to their immediate affect. An additional daily survey was given on the next day if a daily survey was skipped, so as to ensure maximum

⁴ We chose to give the survey retrospectively as opposed to momentarily because we expected momentary assessment to reveal low rates of negative events and negative mood in our non-clinical population, as evidenced in a previous study using pure momentary assessment to measure CR (Wenze, Gunthert, & Forand, 2006).

response. REDCap (Harris et al., 2009) was used as our survey distribution and data collection platform.

Participants

222 undergraduate students (68% female, 49% Caucasian) were recruited from a psychology research recruitment website at Vanderbilt University. Inclusion criteria included being between the ages of 18 and 25 and a current, full-time undergraduate student at Vanderbilt. All participants completed the initial survey bundle, 203 of them completed the in-lab mood induction section and 176 of them completed at least five daily dairies. A validity check question was administered in the middle of the initial survey group to ensure quality response. No participants answered the validity check question incorrectly. Participants were compensated with extra credits in their course based on the completeness of their participation.

Measures

The Daily Diary (DD). Each subject was asked to complete a total of 10 daily dairies over the course of two weeks, from which our new measure of CR was derived. The DD consisted of three parts. In part one, subjects briefly described the worst event that happened to them in the past 24 hours and were asked to imagine themselves in the situation. In part two, subjects rated their affect. In part three, subjects provided ratings for their endorsement of three negative cognitions. Because CR only concerns the relation between affect and cognitions, we only used part two and part three of the DD data for this study. In the following paragraphs, we will describe the affect and cognition questions of the DDs in detail, and then provide a statistical framework for analyzing our DD data.

Affect was measured in the DD with 15 five-point Likert-scale questions, each asking subjects to rate their feelings of specific affective states from 1 (not at all) to 5 (a whole lot).

Affect items were selected from the Positive Affect Negative Affect Schedule-Expanded form (PANAS-X; Watson & Clark, 1994), which has been successfully used in many studies of daily affective processes (e.g., Fichman, Koestner, Zuroff, & Gordon, 1999; Gunthert et al., 2002, Wenze, Gunthert, & Forand, 2005). Specifically, five items were drawn from the 10-item Negative Affect scale, five from the five-item “sadness” scale, and five from the 10-item Positive Affect scale of PANAS-X. Examples include “enthusiastic” (PA), “nervous” (NA), and “blue” (sadness). Choice of specific items in PA and NA was in line with Kercher’s (1992) PANAS-SF. A newer variant of PANAS-SF designed by Thompson (2007) was not used for two reasons. First, Thompson’s version of PANAS-SF was designed and worded to measure long-term trait affect as opposed to state affect. Second, Kercher’s version of PANAS-SF demonstrated better within-person factor loading onto the factors PA and NA (Merz & Roesch, 2011). We acknowledge that the arousal level of affect fall onto a spectrum, not just the two ends, but for the purpose of this study, it is our intention to select sample affect items that have high factor loadings onto Watson and Tellegen’s (1985) PA and NA. Scores from each five-item cluster were averaged to obtain an overall rating of each type of affect.

Dysfunctional cognitions were also collected during the participant’s DD entries. They were measured with three five-point Likert-scale questions (from 1, not at all to 5, a whole lot) drawn from the three subscales (self, world and future) of the Cognitive Triad Inventory (CTI; Beckham, Leber, Watkins, Boyer, & Cook, 1986). The CTI is an instrument developed to measure the cognitive triad (negative perceptions of self, world, and future), which Beck and colleagues (1979) conceptualized to be maladaptive cognitive schemata of depression. The scale and its subscales demonstrated high internal reliability in clinical samples as well as good convergent validity. It has been widely used to assess depression-related maladaptive cognitions.

Since we expect our sample to be healthier than the population in which the CTI was developed to assess, we modified the wording of CTI items to make them easier to endorse. Examples of cognitive items include “Sometimes, things don’t seem to work out for me” and “At times, it just seems that I can’t do anything right.” Scores from the three items were averaged to obtain an overall rating of dysfunctional cognition.

The DD data can be best analyzed with a Multi-Level Model (MLM). In our model, the daily rating of cognition (C_{pd}) can be predicted by two components of daily affect, represented in the MLM equations below as cA_p and cA_{pd} . To understand the first component (cA_p), recall that each subject may have different ratings of sad affect during the course of the study, and we denote the average daily sad affect for each of the N participants to be A_p , which represents the typical affective response of a person to a daily negative event or the “trait” affect of a person. Then, we centered A_p at its grand mean ($A_{..}$) to attain a specific person’s deviation score of trait affect against an “average” participant, denoted as cA_p . Mathematically, $cA_p = A_p - A_{..}$. This is a totally between-subject predictor, representing how one participant’s average sad affect during the course of the study compares to that of the “average” participant. For the second component (cA_{pd}), because each day is different for each subject, there is a rating difference between a person’s raw rating of sad affect at a specific day (A_{pd}) and that person’s average affect rating (A_p); we denote the difference as cA_{pd} , as it represents the person’s specific mood state on that day centered at A_p . Mathematically, $cA_{pd} = A_{pd} - A_p$. We also suspected that measures on consecutive diary entries may have stronger covariances than do distant ones. To account for that potential effect, we opted to include the day (D_{pd}) at which the diary was submitted as a level-1 predictor of daily cognition. For our level 1 residuals, we chose a first order auto-regressive structure to model the covariance between measurements at any two days. The first order

autoregressive structure (AR(1) in SPSS syntax), take the following form for each student: $\varepsilon_{pd} \sim$

$$MVN \begin{bmatrix} 0 \\ 0 \\ \vdots \\ 0 \end{bmatrix}, \begin{bmatrix} \hat{\sigma}^2 & \hat{\rho} & \dots & \hat{\rho}^{14} \\ \hat{\rho} & \hat{\sigma}^2 & \dots & \hat{\rho}^{13} \\ \vdots & \vdots & \ddots & \vdots \\ \hat{\rho}^{14} & \hat{\rho}^{13} & \dots & \hat{\sigma}^2 \end{bmatrix} \text{ where } \hat{\rho} \text{ is the correlation between measurements taken at}$$

successive days. The time between measurement occasions is the “lag”. We used first order autoregressive structure because the value of day d is predicted from the values at day $(d - 1)$.

No level-2 covariance structure was specified. Thus, we obtained the following model:

$$\text{Level 1: } C_{pd} = \beta_{0p} + \beta_{1p}cA_{pd} + \beta_{2p}D_{pd} + \varepsilon_{pd}$$

$$\text{Level 2: } \beta_{0p} = \gamma_{00} + \gamma_{01}cA_p + u_{0p}$$

$$\beta_{1p} = \gamma_{10} + u_{1p}$$

$$\beta_{2p} = \gamma_{20} + u_{2p}$$

$$\text{Reduced Form: } C_{pd} = \gamma_{00} + \gamma_{01}A_p + \gamma_{10}cA_{pd} + u_{1p}cA_{pd} + \gamma_{20}D_{pd} + u_{2p}D_{pd} + u_{0p} + \varepsilon_{pd}$$

$$\varepsilon_{pd} \sim MVN \begin{bmatrix} 0 \\ 0 \\ \vdots \\ 0 \end{bmatrix}, \begin{bmatrix} \hat{\sigma}^2 & \hat{\rho} & \dots & \hat{\rho}^{14} \\ \hat{\rho} & \hat{\sigma}^2 & \dots & \hat{\rho}^{13} \\ \vdots & \vdots & \ddots & \vdots \\ \hat{\rho}^{14} & \hat{\rho}^{13} & \dots & \hat{\sigma}^2 \end{bmatrix} \text{ and } \begin{bmatrix} u_{0p} \\ u_{1p} \\ u_{2p} \end{bmatrix} \sim MVN \left(\begin{bmatrix} 0 \\ 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \tau_{00}^p & & \\ \tau_{10}^p & \tau_{11}^p & \\ \tau_{20}^p & \tau_{21}^p & \tau_{22}^p \end{bmatrix} \right)$$

In this formulation, CR-SAD-DD is operationalized as the subject’s random slope (β_{1p}), reflecting the strength of relation between a person’s deviation or shift from their average affect and their concurrent depressive cognitions. Note that our operationalization of CR-SAD-DD leaves out the association of a person’s general tendency to experience sadness (i.e., trait sadness) with daily depressive cognitions (represented by the fixed slope γ_{01}), as it is a fixed parameter that describes the *sample*, not the *individual*. The final model converged under Restricted Maximum Likelihood (REML) estimation and the estimation of the fixed slopes and intercepts are shown in Table 1. Observe that γ_{10} is significant ($p < .001$), indicating that on

average, a person's daily dysfunctional cognition is dependent on their sad affect at that day after controlling for their general tendency to experience sad affect.

The College Student Behind Your Back Survey (BYB; Cole et al., 2019). The BYB survey was administered in lab before the mood induction procedure to measure CR. The instrument was developed and validated as a part of a larger study by our lab to measure cognitive and emotional reactivity in college students. It asks participants to read a number of scenarios that might occur with peers "behind their backs." Scenarios run the full spectrum of peer interactions that take place between college students from very mild to very harsh. After reading each scenario, participants responded to a number of cognitive and affect items regarding what they would think or how they would feel about themselves if the situation were to have occurred in their lives. In the current study, participants were told that the BYB survey was an "imagining activity" as part of their laboratory session.

Because the scenario-based BYB also evaluates CR from multiple data points of the same person, MLM was used to analyze it. The only difference in this model and the one used to analyze the DD is that the "day" predictor (in this case, hypothetical scenario) was not included because we have no reason to assume consecutive BYB scenarios elicit more similar responses than do distant scenarios. In the formulation, CR-SAD-BYB reflects the strength of relation between a person's deviation or shift from their average affective response to a negative social scenario and their induced depressive cognitions. The model converged under REML estimation and the estimation of the fixed slopes and intercepts are shown in Table 3. The significance of γ_{10} implies that on average, a person's dysfunctional cognition initiated after a BYB scenario depends on their sad affect after that scenario, controlling for their general tendency to respond those scenarios with sad affect.

Leiden Index of Depressive Sensitivity-Re-Revised (LEIDS-RR; Solis, Antypa, Conijin, Kelderman, & Van der Does, 2017). To test the concurrent validity of our new measure of CR, we administered a conventional self-report questionnaire measure of CR, the LEIDS-RR. The 30-item LEIDS-RR is an improved and abbreviated version of LEIDS-R. It aims to assess general effects of sad mood on cognition as well as more specific effects. Example items include “When I feel down, I more often feel hopeless about everything,” and “When I am sad, I am more ready to help others.”. Principal components analysis demonstrated that the LEIDS-RR contains 5 subscales (Hopelessness, Perfectionism/Control, Avoidant Coping, Aggression, and Acceptance/Coping). Subscales exhibit sufficient intercorrelations to allow combination. The internal consistency of the LEIDS-RR was satisfactory in the current study (coefficient alpha = 0.89).

The Sad Mood Induction (MI). The sad mood induction was conducted in lab after the completion of BYB instrument to measure CR. First, participants completed a “writing activity,” in which they were asked to reflect on a situation in which they felt particularly sad or upset and write about it on a piece of paper. Participants were asked specifically to reflect on and write about their role in the situation. Sad music (Prokofiev’s “Russia Under the Mongolian Yoke” played at half speed) was played while participants were engaged in the task. Previous studies have used similar mood induction techniques with no adverse effects (e.g., Clark and Teasdale, 1985). Before and after the mood induction, the positive affect scale and negative affect scale in PANAS-X (Watson & Clark, 1994) and one form of the Dysfunctional Attitudes Scale (DAS; Weissman, 1979) were administered to measure change in affect and dysfunctional cognition during mood induction. To conceal our purpose of inducing sad mood, the baseline affect measures and DAS Form A were administered directly after the BYB instrument, as though they

were a package separated from the later writing activity. DAS Form B and the second affect measures were given directly after the writing activity. The DAS is a commonly used well-validated measure designed to assess the severity of some of the dysfunctional attitudes commonly seen in depression. Two forms are available so that researchers or clinicians can measure dysfunctional attitudes across time while reducing the risk of participant effects. It asks respondents to rate their experiences of 40 attitudes (80 total between the two forms) on a 1 to 7 scale. Reliability for both forms of the DAS were good in the current study (coefficient alpha = .90 for both forms). The change in DAS scores (i.e., DAS_B – DAS_A) were used as an alternative measure of CR. At the end of MI, participants were debriefed and shown a humorous video.

Beck Depression Inventory II (BDI-II; Beck, Steer, & Garbin, 1996). The BDI-II was administered at the beginning of subjects' participation to measure DS. The BDI-II is a commonly used well-validated measure designed to assess severity of depressive symptoms in a variety of populations. It asks respondents to rate their experiences of 21 different depressive symptoms over the past two weeks on a 0 to 3 scales. In our study, the suicidality item of BDI-II was removed due to confidentiality concerns. The internal consistency of BDI-II in our sample was high (coefficient alpha=.90).

Results

Descriptive Statistics

All subjects ($N = 222$) were included in the analysis, and missing data were handled with FIML. The means, standard deviations and correlations among major variables involved in the

analysis are shown in Table 2. Means and standard deviations for BDI-II and LEIDS-RR are similar to other studies with college student samples.

Nearly all of the variables were positively correlated except for DAS_Change. A *post hoc* t-test revealed that there *was* a small but significant increase in sad mood (average change on a five-point Likert scale = .112) before and after the mood induction procedure, $t(202) = 5.469, p < .001$, and a small and significant decrease in dysfunction attitudes (average change on DAS = -2.18, $t(202) = -2.290, p = .023$). Gender differences in means, SDs, and pairwise correlations among study variables were small and nonsignificant.

Overview of Analysis

There were four major goals in the current study. For goal 1, we aimed to demonstrate the convergent validity of our daily diary measure of CR-SAD by conducting a confirmatory factor analysis on the four measures of CR-SAD that we collected. For goal 2, we aimed to demonstrate the construct validity of the daily diary measure of CR-SAD by using MLM on the daily diary measure of CR-SAD with concurrent DS as a person-level predictor. For goal 3, we aimed to examine the unique associations of naturally occurring dysfunctional cognitions with NA and PA by including NA and PA as affect predictors of daily cognitions in an MLM. For goal 4, we tested the construct validity of CR-NA and CR-PA by examining their associations with DS. We will present the analyses and results from each goal in the following sections.

Goal 1: Relation of Daily Diary Measure of CR-SAD to Three Other Measures of CR-SAD

Due to difficulties in model convergence, we opted not to use multilevel structural equation modeling to test the convergent validity of our four measures of CR-SAD, two of which (BYB and daily diary) involved a multilevel element. Instead, we performed the test in three separate steps, on each of which model convergence was achieved. First, we used multilevel

modeling to compute CR-SAD-DD. Then, we used a similar approach to compute CR-SAD-BYB. Finally, we entered the four CR-SAD measures as manifest variables into a confirmatory factor analytic model to examine model fit and factor loadings. Details in the first two steps were described in the Method section under descriptions of each measure. In step 3, we extracted the random slope β_{1p} in each of the first two steps as indices of CR-SAD for each subject and compared the four CR-SAD measures that we administered in a confirmatory factor analytic (CFA) model so as to test the convergent validity of our daily diary measure of CR-SAD. The CFA model fit well ($\chi^2 = .955, p = .621; RMSEA = .019; CFI = 1.00; TLI = 1.213$). Factor scores of the results are shown in Table 4. All factor loadings were significant except DAS_Change, the mood induction measure of CR-SAD.

Goal 2: Relation of Daily Diary Measure of CR-SAD to Depressive Symptoms

To establish the construct validity of the daily diary measure of CR-SAD, we examined its relation to depressive symptoms, measured by BDI-II. We did this by adding scores of BDI-II as an additional between-subject (i.e., level-2) predictor of both the random slopes and the random intercept into the multilevel model we run in the first step of Goal 1-1. In addition, we allow BDI to interact with the between-subject component of affect. The model was as follows:

$$\text{Level 1: } C_{pd} = \beta_{0p} + \beta_{1p}cA_{pd} + \beta_{2p}D_{pd} + \varepsilon_{pd}$$

$$\text{Level 2: } \beta_{0p} = \gamma_{00} + \gamma_{01}cA_p + \gamma_{02}BDI + \gamma_{03}cA_p.BDI + u_{0p}$$

$$\beta_{1p} = \gamma_{10} + \gamma_{11}BDI + u_{1p}$$

$$\beta_{2p} = \gamma_{20} + \gamma_{21}BDI + u_{2p}$$

Results presented in Table 5 show that the effect of BDI on the within person aspect of CR is significant was significant ($p < .001$), indicating that CR-SAD-DD is associated with

depressive symptom after controlling for the association of depressive symptoms with daily depressive cognitions.

Goal 3: Adding NA and PA into the Picture

Our second goal was to explore the role of PA and NA in triggering depressive cognition and how their associated CRs are related to depressive symptoms. To test the structural validity of having three affective predictors for daily depressive cognitions, we modified our previous multilevel model in Goal 1 to include centered daily negative and positive affect ratings (cNA_{pd} and cPA_{pd}) as level-1 predictors and the person's centered average ratings (cNA_p and cPA_p) as level-2 predictors. The model was formulated as follows:

$$\text{Level 1: } C_{pd} = \beta_{0p} + \beta_{1p}cSAD_{pd} + \beta_{2p}cNA_{pd} + \beta_{3p}cPA_{pd} + \beta_{4p}D_{pd} + \varepsilon_{pd}$$

$$\text{Level 2: } \beta_{0p} = \gamma_{00} + \gamma_{01}cSAD_p + \gamma_{02}cNA_p + \gamma_{03}cPA_p + u_{0p}$$

$$\beta_{1p} = \gamma_{10} + u_{1p}$$

$$\beta_{2p} = \gamma_{20} + u_{2p}$$

$$\beta_{3p} = \gamma_{30} + u_{3p}$$

$$\beta_{4p} = \gamma_{40} + u_{4p}$$

Reduced Form:

$$\begin{aligned} C_{pd} = & \gamma_{00} + \gamma_{01}cSAD_p + \gamma_{02}cNA_p + \gamma_{03}cPA_p + \gamma_{10}cSAD_{pd} + \gamma_{20}cNA_{pd} \\ & + \gamma_{30}cPA_{pd} + \gamma_{40}D_{pd} + u_{0p} + u_{1p}cSAD_{pd} + u_{2p}cNA_{pd} + u_{3p}cPA_{pd} + u_{4p}D_{pd} \\ & + \varepsilon_{pd} \end{aligned}$$

$$\varepsilon_{pd} \sim MVN \left(\begin{bmatrix} 0 \\ 0 \\ \vdots \\ 0 \end{bmatrix}, \begin{bmatrix} \hat{\sigma}^2 & \hat{\rho} & \dots & \hat{\rho}^{14} \\ \hat{\rho} & \hat{\sigma}^2 & \dots & \hat{\rho}^{13} \\ \vdots & \vdots & \ddots & \vdots \\ \hat{\rho}^{14} & \hat{\rho}^{13} & \dots & \hat{\sigma}^2 \end{bmatrix} \right) \text{ and } \begin{bmatrix} u_{0p} \\ u_{1p} \\ u_{2p} \\ u_{3p} \\ u_{4p} \end{bmatrix} \sim MVN \left(\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \tau_{00} & & & & \\ \tau_{10} & \tau_{11} & & & \\ \tau_{20} & \tau_{21} & \tau_{22} & & \\ \tau_{30} & \tau_{31} & \tau_{32} & \tau_{33} & \\ \tau_{40} & \tau_{41} & \tau_{42} & \tau_{43} & \tau_{44} \end{bmatrix} \right)$$

Results for this model is shown in Table 6. Both γ_{10} and γ_{20} were positive and significant (for both, $p < .001$), suggesting that in our sample, each of daily sadness and daily negative affect was positively associated with depressive cognitions after controlling for the person's trait sadness and trait NA, as well as the effects from other types of affect. γ_{30} was significant ($p < .001$) but negative, suggesting that in our sample, increase in naturally occurring positive affect was associated with decrease in the concurrent dysfunctional cognition.

Goal 4: Examine the Clinical Significance of CR-NA and CR-PA in Relation to Depressive Symptoms

To explore the relative clinical significance in having three affective predictors for daily depressive cognitions, we modified our previous multilevel model in Goal 3 to include scores of BDI-II and its potential interactions with other level-2 predictors as additional between-subject (i.e., level-2) predictors of both the random slopes and the random intercept into the multilevel model. The final model was formulated as follows:

$$\text{Level 1: } C_{pd} = \beta_{0p} + \beta_{1p}cSAD_{pd} + \beta_{2p}cNA_{pd} + \beta_{3p}cPA_{pd} + \beta_{4p}D_{pd} + \varepsilon_{pd}$$

$$\text{Level 2: } \beta_{0p} = \gamma_{00} + \gamma_{01}cSAD_p + \gamma_{02}cNA_p + \gamma_{03}cPA_p + \gamma_{04}BDI + \gamma_{05}cSAD_p.BDI + \gamma_{06}cNA_p.BDI + \gamma_{07}cPA_p.BDI + u_{0p}$$

$$\beta_{1p} = \gamma_{10} + \gamma_{11}BDI + u_{1p}$$

$$\beta_{2p} = \gamma_{20} + \gamma_{21}BDI + u_{2p}$$

$$\beta_{3p} = \gamma_{30} + \gamma_{31}BDI + u_{3p}$$

$$\beta_{4p} = \gamma_{40} + \gamma_{41}BDI + u_{4p}$$

$$\varepsilon_{pd} \sim MVN \left(\begin{bmatrix} 0 \\ 0 \\ \vdots \\ 0 \end{bmatrix}, \begin{bmatrix} \hat{\sigma}^2 & \hat{\rho} & \dots & \hat{\rho}^{14} \\ \hat{\rho} & \hat{\sigma}^2 & \dots & \hat{\rho}^{13} \\ \vdots & \vdots & \ddots & \vdots \\ \hat{\rho}^{14} & \hat{\rho}^{13} & \dots & \hat{\sigma}^2 \end{bmatrix} \right) \text{ and } \begin{bmatrix} u_{0p} \\ u_{1p} \\ u_{2p} \\ u_{3p} \\ u_{4p} \end{bmatrix} \sim MVN \left(\begin{bmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \begin{bmatrix} \tau_{00} & & & & \\ \tau_{10} & \tau_{11} & & & \\ \tau_{20} & \tau_{21} & \tau_{22} & & \\ \tau_{30} & \tau_{31} & \tau_{32} & \tau_{33} & \\ \tau_{40} & \tau_{41} & \tau_{42} & \tau_{43} & \tau_{44} \end{bmatrix} \right)$$

The model converged under REML estimation. Results from this analysis are displayed in Table 7. Despite that γ_{10} and γ_{20} remained significant compared to results from previous model of Goal 3 (Table 6), γ_{30} became nonsignificant noticeably. Also note that none of the three types of CR evinced a significant relation to depressive symptoms, which was also inconsistent with the results from Goal 2 in which CS-SAD was related to scores of BDI-II.

Discussion

Major Findings and Implications

Three major findings emerged from the current study. First, the daily diary measure of CR-SAD demonstrated convergence with both the conventional self-report questionnaire measure (LEIDS-RR) and the scenario-based measure (BYB). Second, the daily diary measure of CR-SAD was found to relate to depressive symptoms. Third, naturally occurring NA and PA were both related to daily levels of dysfunctional cognitions in unique ways. We will elaborate on each of the findings below.

First, naturally occurring fluctuations in sadness were positively associated with the fluctuations in depressive cognitions, and results from CFA found that this operationalization of CR aligned with other measures of CR-SAD (e.g., the LEIDS-RR and the BYB). Specifically, the positive association between daily-recorded sad affect and dysfunctional cognitions indicates that higher levels of sadness tend to co-occur with higher levels of dysfunctional cognitions that are typically seen in people with depression. The convergence of our daily diary measure with LEID-RR indicates that CR-SAD assessed by the dynamic daily diary method captures the same underlying construct as CR-SAD assessed by a conventional self-report measure. The convergence with BYB, another dynamic measure of CR-SAD, shows the associations between

affect and cognitive reactions to be consistent within a given individual, suggesting that reactions to hypothetical and actual events might be controlled by common neurophysiological processes.

Second, we found that the daily diary measure of CR-SAD was related to depressive symptoms. This means people whose levels of dysfunctional cognitions are more strongly associated with their sad affect tend to have more depressive symptoms, a result consistent with numerous studies in CR (see reviews by Scher, Ingram, & Segal, 2005; Segal & Ingram, 1994). Clinically, it justifies the use of the daily diary approach to estimate dynamically a person's tendency to experience depressive symptoms. Further research is needed to see if this approach can also predict onset, duration, and relapse of depression.

Third, when sadness, NA, and PA were examined simultaneously, each of the three types of affect was found to contribute to levels of dysfunctional cognitions. Increase in sadness or NA was generally associated with increase in levels of dysfunctional cognitions, suggesting that the emergence of those cognitions may be precipitated by NA as well as sadness. To the contrary, increase in PA was generally associated with decrease in levels of dysfunctional cognitions, suggesting that dysfunctional cognitions may be inhibited by PA. These results were consistent with our expectations based on Watson and Tellegen's (1985) study of PA and NA. Note, though, that we did not find evidence to support the unique clinical significance of CR-PA and CR-NA in terms of their associations with depressive symptoms. Psychometrically, this might be because our sample size could not support so a complex model with multiple affective predictors and their interactions with DS estimated simultaneously. It could also be due to the multicollinearity among PA, NA and sadness. Future analysis can estimate the relations of CR-NA, CR-PA and CR-SAD to DS in separate models to resolve those concerns. Nomothetically, it is possible that (1) CR-NA and CR-PA are related to other diathesis which may explain the

associations of depression-related dysfunctional cognitions with PA and NA, or (2) they may relate other closely related psychopathologies such as anxiety disorders.

Taken together, these findings provide both psychometric and nomothetic support for the assessment of CR using naturally occurring affect and cognition extracted from daily dairies. Compared to conventional measures of CR, the daily diary method has the following advantages: (1) the stimuli in the daily diary are what have actually occurred to the person in their life, ensuring the ecological validity and personal relevance; (2) instead of relying on just two data points to draw inference on a dynamic construct such as CR, the daily dairies provide multiple data points and are estimated with MLM, thus drastically reducing estimation error and avoiding capitalizing on chance; (3) due to the longitudinal nature of daily dairies, clinicians are able to dynamically monitor potential changes in CR during different phases of treatment, adding another tool for them to guide the treatment process; (4) the daily diary approach is flexible enough to measure other dynamic constructs simultaneously without sacrificing much if any validity. For example, the current study proposed and measured CR-NA and CR-PA within the daily diary framework, but emotional reactivity, defined as the association between the appraisal of an event and the strength of affective reaction, can also be derived from the daily diary approach.

Limitations and Future Directions

There are several limitations to the current study. First, our mood induction procedure in assessing CR-SAD did not produce its expected effects. On average, the mood induction only caused very small changes in affect and dysfunctional cognitions. Besides, it did not correlate significantly with DS nor have a significant factor loading in the CFA model as did other measures of CR-SAD. We suspect that the ineffectiveness of mood induction might be due to

factors that are innovative in our experimental settings. For example, allowing participants to write down their experience of sad feelings might produce an effect of catharsis, and consequently relieving their sad mood. It would be interesting to study the effect of writing on relieving mood. Another possible explanation for the small change in affect is that the affect measure administered after the mood induction only came after subjects have taken time to answer the 40 questions in DAS Form B, during which their mood might be restored. For the small change in DAS scores, it is possible that when filling out DAS Form B, participants might expect that they were supposed to provide consistent answers with DAS Form A, which in our study were administered along with the BYB instrument so as to conceal the real propose of the writing activity. The ineffectiveness of our lab-based mood induction procedure might indicate that mood induction is not a reliable way of measuring of CR in nonclinical population, but further investigation may refine the mood induction procedure to increase its efficacy.

Second, our daily diary method relies on subjects' retrospective recall and self-report of negative events and their subsequent reactions, which may hinder the fidelity of the data. Due to the time lag between actual events and the report, subjects' memory of their mood and cognition may not be completely accurate. For example, retrospective reports about negative events are affected by current mood when filling out the survey (Monroe & Simons, 1991). Despite this limitation, we decided to use daily diary methodology as opposed to a pure ecological momentary assessment for its advantage in capturing subject's negative mood fluctuation and to avoid potential floor effects when ecological momentary assessments fail to coincide with negative events. Future research could replace daily diary with daily clinical interviews, which could increase subjects' commitment to the study and consequently their accuracy of report, though the cost of such study could be prohibitive.

Third, because most of the measures were administered within a short period of time, causal inferences could not be drawn. For example, our results could not prove that CR-SAD was a personological diathesis that contributes to DS, for high reactivity to sad affect could just be a situational phenomenon that tend to coexist with DS. Causal inferences could be achieved by assessing change in DS over a certain time span. Future work could also aim to examine the effect of factors that may cause both CR and DS, such as perceived stress and ruminative thinking.

Fourth, because we study non-clinical population (i.e., college students), our sample had a skewed distribution on DS, for BDI was developed and validated in a clinical sample. This may have affected our regression-based statistical analysis by not providing sufficient data points in the high ends of DS. A direct remedy for this problem is to increase both the sample size and the sample diversity to obtain a better representation of adults. We also encourage future research to replicate this study in a clinical sample to better make use of the results for clinical research and practice.

Besides improving on the limitations of the current study, there are several additional directions that we recommend for future research. First, one of the diatheses for depression that is closely related to CR is emotional reactivity (ER), defined as the activation, duration, and intensity of one's emotional response to a typical stimulus (Nock, Wedig, Holmberg, & Hooley, 2008). It would be interesting to know the relation among ER and multiple types of CR, and whether they combine to form a larger diathesis for depression and other internalizing disorders.

Second, because different people may exhibit different patterns in CR, the relative scores in CR-SAD, CR-PA, and CR-NA will provide a unique CR profile for each individual. We suspect that the CR profile may have profound clinical implications beyond depression. For

example, due to the association between NA and anxiety disorders (Tellegen, 1985), a worthwhile direction for subsequent research is to substitute anxiety-related cognitions into our model of CR-NA. If CR-NA with anxiety-related cognitions is also found to be a diathesis for anxiety disorders, then elevated CR-NA may constitute one of the common risk factors for internalizing disorders. One may also suspect that patients with high CR-PA are more likely to experience anhedonia. Examining the potential link between CR profile and other psychopathologies represents an important path for future work.

Third, although CR may be a diathesis for depression, it remains unclear what exactly contributes to individual differences and potential development of different types of CR. We recommend two directions for future investigations. Psychologically, many types of personality traits and developmental experience may have strong associations with both CR as a whole and different types of CR. Knowing both the personological and the situational precursors to different types of elevated CR might be crucial in the enhancing children and adults' mental health conditions. Neurologically, we suspect that particular brain regions or circuits may be responsible for relating dysfunctional cognitions to various types of affect. A recent meta-analysis on the brain basis of PA and NA revealed that affective valence is flexibly implemented across instances by a set of valence-general limbic and paralimbic brain regions (Lindquist, Satpute, Wager, Weber, & Feldmann Barrett, 2016), so it still remains mysterious as to what distinguishes PA and NA in terms of neurochemical processes. Advance in neuroscience may eventually provide a biological explanation of CR and its structure.

Conclusion

The current study contributed to the literature on cognitive diathesis of depression by (1) developing and validating an ecological measure of CR, and (2) probing into the cognitive

processes triggered by multiple types of affect. Our finding of significant associations between each of PA and NA and daily fluctuation of dysfunctional attitudes confirmed the idea that the depression-related dysfunctional attitudes do not just emerge from sadness; responding to a naturally occurring negative event with negative affect and very low levels of positive affect are also precursors to those maladaptive thoughts. Our study was a solid step toward studying the connections between the dynamics of human emotions and psychopathologies. We hope that our findings can open up a gate for future research in this area.

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Table 1

Basic Model Results for Cognitive Reactivity to Sadness in Daily Diary Measure

Parameter descriptions	Estimate	SE	<i>p</i> <
γ_{00} : fixed intercept of cognition (β_{0p})	0.805	0.045	0.000
γ_{01} : fixed effect of trait sadness on cognition	0.822	0.055	0.000
γ_{10} : fixed effect of CR-SAD	0.478	0.032	0.000
γ_{20} : fixed effect of day on cognition	0.005	0.005	0.282
$\hat{\sigma}^2$: variance of a subject at a particular day	0.310	0.014	0.000
$\hat{\rho}$: correlation between successive measurement	0.133	0.037	0.000
τ_{00} : variance of β_{0p} at person level	0.177	0.040	0.000
τ_{11} : variance of β_{1p} at person level	0.081	0.018	0.000
τ_{22} : variance of β_{2p} at person level	0.001	0.000	0.277
τ_{10} : covariance of β_{0p} and β_{1p} at person level	0.061	0.018	0.001
τ_{20} : covariance of β_{0p} and β_{2p} at person level	-0.003	0.004	0.346
τ_{21} : covariance of β_{1p} and β_{2p} at person level	0.004	0.002	0.055

Table 2

Correlations, Means, and Standard Deviations for Study Variables

Measure	M (SD)	1	2	3	4	5
1. BDI-II	10.59 (8.54)	-				
2. CR-SAD-DD	.48 (.23)	.28**	-			
3. CR-SAD-BYB	.46 (.20)	.23**	.13	-		
4. LEIDS-RR	33.46 (16.07)	.61**	.15*	.22**	-	
5. DAS_Change	-2.18 (13.58)	-.04	.02	-.03	-.09	-

Note. BDI-II = Beck Depression Inventory II; CR-SAD-DD = Daily Diary Measure of Cognitive Reactivity to Sadness; CR-SAD-BYB = Behind Your Back Measure of Cognitive Reactivity to Sadness; LEIDS-RR = Leiden Index of Depressive Sensitivity - Re-revised; DAS_Change = Difference in Dysfunctional Attitude Scale scores before and after mood induction.

* $p < .05$. ** $p < .01$.

Table 3

Basic Model Results for Cognitive Reactivity to Sadness in BYB Measure

Parameter descriptions	Estimate	SE	$p <$
γ_{00} : fixed intercept of cognition (β_{0p})	1.134	0.058	0.000
γ_{01} : fixed effect of person's typical sadness on cognitions	0.842	0.051	0.000
γ_{10} : fixed effect of CR-SAD	0.458	0.019	0.000
σ_{ε}^2 : variance of ε_{ps} at scenario level	0.541	0.012	0.000
τ_{00} : variance of β_{0p} at person level	0.471	0.060	0.000
τ_{11} : variance of β_{1p} at person level	0.052	0.006	0.000
τ_{10} : covariance of β_{0p} and β_{1p} at person level	-0.122	0.017	0.000

Table 4

Confirmatory Factor Analysis Results for Four Measures of Cognitive Reactivity to Sadness

Manifest Variable	β	SE	t
CR-SAD-DD	0.281**	0.111	2.526
CR-SAD-BYB	0.419**	0.144	2.907
LEIDS-RR	0.544**	0.178	3.058
DAS_Change	-0.105	0.104	-1.005

** $p < .01$.

Table 5

Multilevel Model of Daily Diary Measure of CR with BDI as Level 2 Predictor

Parameter descriptions	Estimate	SE	$p <$
γ_{00} : fixed intercept of cognition (β_{0p})	0.587	0.070	0.000
γ_{01} : fixed effect of trait sadness on cognition	0.641	0.088	0.000
γ_{02} : fixed effect of BDI on cognition	0.021	0.006	0.000
γ_{03} : BDI x cA_p . (BDI \rightarrow Effect of trait sadness on cognition)	0.009	0.005	0.102

γ_{10} : fixed effect of CR	0.340	0.051	0.000
γ_{11} : BDI x cA_{pd} (BDI \rightarrow CR)	0.012	0.004	0.001
γ_{20} : fixed effect of Day on cognition	0.012	0.008	0.140
γ_{21} : BDI x D_{pd} (BDI \rightarrow Effect of Day on cognition)	-0.001	-0.001	0.287
$\hat{\sigma}^2$: variance of a subject at a particular day	0.309	0.014	0.000
$\hat{\rho}$: correlation between successive measurement	0.131	0.037	0.000
τ_{00} : variance of β_{0p} at person level	0.145	0.037	0.000
τ_{11} : variance of β_{1p} at person level	0.073	0.017	0.006
τ_{22} : variance of β_{2p} at person level	0.001	0.000	0.263
τ_{10} : covariance of β_{0p} and β_{1p} at person level	0.047	0.017	0.000
τ_{20} : covariance of β_{0p} and β_{2p} at person level	-0.002	0.004	0.521
τ_{21} : covariance of β_{1p} and β_{2p} at person level	0.004	0.002	0.026

Table 6

Basic Model Results for Cognitive Reactivity to Three Affects in Daily Diary Measure

Parameter descriptions	Estimate	SE	$p <$
γ_{00} : fixed intercept of cognition (β_{0p})	0.813	0.045	0.000
γ_{01} : fixed effect of trait sadness on cognition	0.627	0.080	0.000
γ_{02} : fixed effect of trait NA on cognition	0.227	0.074	0.002
γ_{03} : fixed effect of trait PA on cognition	-0.062	0.061	0.309
γ_{10} : fixed effect of CR-SAD	0.341	0.035	0.000
γ_{20} : fixed effect of CR-NA	0.268	0.030	0.000
γ_{30} : fixed effect of CR-PA	-0.097	0.033	0.004
γ_{40} : fixed effect of Day on cognition	0.004	0.005	0.420
$\hat{\sigma}^2$: variance of a subject at a particular day	0.245	0.012	0.000
$\hat{\rho}$: correlation between successive measurement	0.105	0.041	0.010
τ_{00} : variance of β_{0p} at person level	0.211	0.040	0.000
τ_{11} : variance of β_{1p} at person level	0.090	0.024	0.000
τ_{22} : variance of β_{2p} at person level	0.059	0.016	0.000

τ_{33} : variance of β_{3p} at person level	0.032	0.016	0.041
τ_{44} : variance of β_{4p} at person level	0.001	0.000	0.031
τ_{10} : covariance of β_{0p} and β_{1p} at person level	0.021	0.021	0.318
τ_{20} : covariance of β_{0p} and β_{2p} at person level	0.044	0.018	0.017
τ_{30} : covariance of β_{0p} and β_{3p} at person level	-0.048	0.021	0.021
τ_{40} : covariance of β_{0p} and β_{4p} at person level	-0.008	0.004	0.034
τ_{21} : covariance of β_{1p} and β_{2p} at person level	-0.033	0.016	0.045
τ_{31} : covariance of β_{1p} and β_{3p} at person level	-0.014	0.014	0.327
τ_{41} : covariance of β_{1p} and β_{4p} at person level	0.006	0.002	0.012
τ_{32} : covariance of β_{2p} and β_{3p} at person level	-0.010	0.013	0.327
τ_{42} : covariance of β_{2p} and β_{4p} at person level	-0.002	0.002	0.213
τ_{43} : covariance of β_{3p} and β_{4p} at person level	0.001	0.002	0.801

Table 7

*Relation of Depressive Symptoms to Cognitive Reactivity to Three Types of Affect
Derived from a Daily Diary Measure*

Parameter descriptions	Estimate	SE	$p <$
γ_{00} : fixed intercept of cognition (β_{0p})	0.627	0.072	0.000
γ_{01} : fixed effect of trait sadness on cognition	0.242	0.123	0.050
γ_{02} : fixed effect of trait NA on cognition	0.486	0.123	0.000
γ_{03} : fixed effect of trait PA on cognition	-0.021	0.086	0.810
γ_{04} : fixed effect of BDI on cognition	0.018	0.006	0.003
γ_{05} : fixed effect of trait sadness*BDI on cognition	0.025	0.008	0.001
γ_{06} : fixed effect of trait NA*BDI on cognition	-0.021	0.009	0.028
γ_{07} : fixed effect of trait PA*BDI on cognition	-0.002	0.009	0.822
γ_{10} : fixed effect of CR-SAD	0.251	0.056	0.000
γ_{20} : fixed effect of CR-NA	0.238	0.048	0.000
γ_{30} : fixed effect of CR-PA	-0.021	0.052	0.689
γ_{40} : fixed effect of Day on cognition	0.008	0.008	0.293

γ_{11} : fixed effect of BDI on CR-SAD	0.008	0.004	0.055
γ_{21} : fixed effect of BDI on CR-NA	0.003	0.004	0.441
γ_{31} : fixed effect of BDI on CR-PA	-0.008	0.004	0.071
γ_{41} : fixed effect of BDI*Day on cognition	-0.001	0.001	0.422
$\hat{\sigma}^2$: variance of a subject at a particular day	0.245	0.012	0.000
$\hat{\rho}$: correlation between successive measurement	0.108	0.041	0.008
τ_{00} : variance of β_{0p} at person level	0.190	0.038	0.000
τ_{11} : variance of β_{1p} at person level	0.086	0.024	0.000
τ_{22} : variance of β_{2p} at person level	0.058	0.016	0.000
τ_{33} : variance of β_{3p} at person level	0.029	0.015	0.055
τ_{44} : variance of β_{4p} at person level	0.001	0.001	0.019
τ_{10} : covariance of β_{0p} and β_{1p} at person level	0.014	0.021	0.486
τ_{20} : covariance of β_{0p} and β_{2p} at person level	0.043	0.018	0.017
τ_{30} : covariance of β_{0p} and β_{3p} at person level	-0.042	0.020	0.033
τ_{40} : covariance of β_{0p} and β_{4p} at person level	-0.008	0.004	0.028
τ_{21} : covariance of β_{1p} and β_{2p} at person level	-0.034	0.016	0.037
τ_{31} : covariance of β_{1p} and β_{3p} at person level	-0.009	0.014	0.506
τ_{41} : covariance of β_{1p} and β_{4p} at person level	0.006	0.002	0.013
τ_{32} : covariance of β_{2p} and β_{3p} at person level	-0.008	0.013	0.517
τ_{42} : covariance of β_{2p} and β_{4p} at person level	-0.003	0.002	0.201
τ_{43} : covariance of β_{3p} and β_{4p} at person level	0.001	0.002	0.760

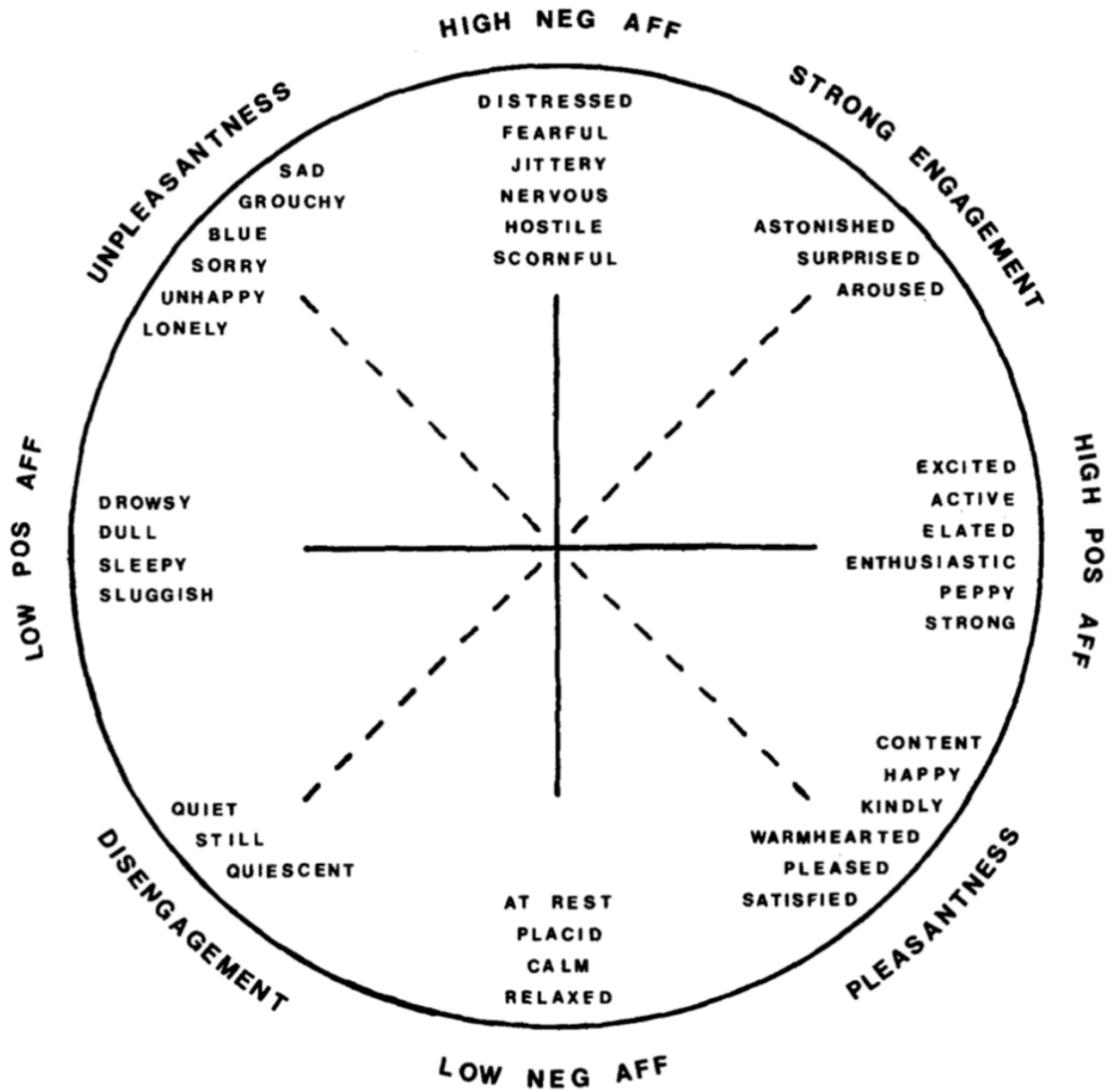


Figure 1. The structure of affect in a circumplex. NEG AFF = Negative Affect, POS AFF = Positive Affect. Adapted from "Toward a Consensual Structure of Mood" by Watson and Tellegen (1985).