

INHIBITORY PROCESSING OF SAD FACIAL EXPRESSIONS AND DEPRESSION  
VULNERABILITY

BY

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Submitted to the graduate degree program in Psychology and the Faculty of the  
Graduate School of the University of Kansas in partial fulfillment of the requirements  
for the degree of Doctor of Philosophy

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Date defended: December 3, 2010

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## Abstract

Depression vulnerability has been frequently linked to selective attention biases, but these biases may partly result from an inhibitory deficit for processing depressive information (Joormann, 2004). Reduced inhibition when encountering sad interpersonal information (e.g., faces) could lead to greater associative processing, deeper encoding among related depressive content in memory, increased rumination, and perhaps promote depressive episodes. Inhibition and selective attention can be examined through behavioral and psychophysiological indicators, including the N200, P300a, and P300b ERP components. The present study examined whether groups traditionally at risk of depression would show inhibitory deficits for depressive facial expressions as compared to a low-risk group. A 2 x 2 design yielded four groups with two levels of current dysphoria status (yes/no) and history of depression (yes/no), enabling comparisons of relative risk. Each participant completed two visual oddball tasks. In the experimental task, participants responded or inhibited a response to infrequently presented sad or happy target faces in the context of frequently presented neutral faces. In the non-affective control task, participants responded only to faces that fit into one of three broad age groupings. Behavioral (e.g., reaction times, response errors), psychophysiological (ERP components), and self-report (e.g., rumination) measures relevant to selective attention and inhibition were analyzed. Between- and within-groups contrasts were conducted to reveal whether at-risk groups exhibit attentional bias and inhibitory deficiency specific to depressive information. Also, the study examined whether different operationalizations of depression risk evince common or distinct mechanisms of vulnerability. Across the full sample, previous depression was associated with greater P3b amplitude for sad target faces than happy target faces, in contrast with the depression naïve group. However in males, only the combination of previous depression and current dysphoria

were linked to elevated P3s following sad targets. Evidence for a sad affect inhibition deficit was limited to dysphoric females' increased errors of commission following sad distracter faces. Results suggest that specific operationalizations of risk may be characterized by an attentional bias toward depressive facial affect in the social environment, which could promote additional depressogenic cognition and social behavior. Theoretical ramifications regarding gender and state versus trait vulnerability are also discussed.

Keywords: Depression, Vulnerability, Bias, Facial Emotion, ERP, Cognitive Neuroscience

## Acknowledgements

I would like to express sincere gratitude to the members of my dissertation committee. I am extremely grateful for Rick Ingram's mentorship and support throughout my training at the University of Kansas, particularly during the dissertation process. I am indebted to Ruthann Atchley for providing me the encouragement, guidance, and resources needed for my maiden voyage into the world of ERP research. I also want to thank Nancy Hamilton, Sarah Pressman, and William Skorupski for challenging me to consider important issues and improve the design of the current research. I would also like to acknowledge Aminda O'Hare for generously lending her time and technical expertise at several important points along the way. In addition, I sincerely thank each of the research assistants who put forth significant efforts to make this multi-year project run: Shandi Appier, George Bradshaw, Bryan Candea-Kromm, Andrew Garcia, Aaron Gates, Matt Judah, Whitney Kearney, Kayla Meyer, Elizabeth Powell, and Addie Timmons. Lastly, I cannot thank my wife, my family, and my friends enough. Your unconditional support and understanding throughout this process has been amazing.

## Table of Contents

### ABSTRACT

### INTRODUCTION

1. Studying Depression and Depression Vulnerability
2. Evidence of Cognitive Biases in Depression Vulnerable Populations
3. Selective Attention and Inhibition
4. Selective Attention to Affective Facial Expressions and Depression Vulnerability
5. Neuroelectric Activity Related to Selective Attention/Inhibition and Depression Vulnerability
6. The Current Research
7. Hypothesized Findings

### METHOD

8. Participants
9. Experimental stimuli
10. Experimental Procedure
11. Collecting and Cleaning Oddball Task Data
12. Data Analyses

### RESULTS

13. Self-Report Measures
14. Oddball Task Accuracy
15. Oddball Task Reaction Time
16. Oddball Task Event-Related Potentials

### DISCUSSION

### REFERENCES

### APPENDICES

## Inhibitory Processing of Sad Facial Expressions and Depression Vulnerability

Two robust strains of research indicate that depressed individuals show patterns of negative cognition and deficits in social function. Further, theories have long proposed that cognitive and interpersonal factors are salient to the etiology, maintenance and treatment of major depression (Beck, 1967; Lewinsohn, 1974). Specifically, cognitive theories of depression have proposed that negative biases in attention and memory characterize depressed individuals and those who are at risk of developing depression. Interpersonal theories have suggested that social deficits impede reinforcement and often evoke punishing responses in social interactions. Integrating interpersonal themes with cognitive models, depression vulnerability might be associated with cognitive biases in processing social affective information. Human facial expressions in the social environment effectively transmit and evoke emotions (Ruys & Stapel, 2008), and a developing collection of studies has begun to examine whether depression and depression vulnerability are characterized by cognitive biases related to processing affective faces. Evidence suggests that depressed and depression susceptible individuals may attend to, interpret, and remember affective facial expressions in distinct ways that initiate or maintain depressive patterns of mood and cognition (Bistricky, Ingram, & Atchley, 2010). However, there is much to be learned about these apparent cognitive biases and how they may be instantiated in particular patterns of neural activity. Research is needed to elucidate possible mechanisms of risk.

The present research project employed behavioral and neuroelectric measurement tools to examine whether depression susceptible individuals exhibit depressotypic attentional biases and inhibitory deficits when they encounter affective facial expressions, which could contribute to them becoming and remaining depressed. However, before detailing the current research, the relevant context from which it emerges is reviewed. First, conceptual and methodological issues

that influence contemporary investigation of depression vulnerability are discussed. Next, evidence is reviewed regarding depressotypic cognitive biases found in groups representing different operationalizations of depression risk. This leads into more focused consideration of selective attention, inhibition, and a proposed theoretical model in which attentional bias promotes vulnerability to depression. Subsequently, the introduction provides a rationale for examining interpersonal facial expression processing biases and a brief summary of studies that have done so. Finally, I examine how cognitive neuroscience research (specifically event-related potentials; ERP) techniques are elucidating the neural underpinnings of attentional processing, and propose that these techniques can significantly augment the current understanding of depressotypic attentional biases toward affective interpersonal information.

#### *Studying Depression and Depression Vulnerability*

Although individuals can be directly classified as depressed by meeting diagnostic criteria, operationalizing depression vulnerability is necessarily indirect. Studies typically operationalize vulnerability to depression according to known risk factors, then compare high risk groups to low risk groups on variables hypothesized to mediate greater risk. Vulnerable groups are often composed of individuals who have experienced a past major depressive episode or who are currently dysphoric. Major depression is characterized by a high rate of recurrence, and the risk of recurrence increases with each new depressive episode (American Psychiatric Association, 2000). Similarly, individuals with subclinical depressive syndromes may be up to six-times more likely to develop major depression in the long-term than individuals who have never experienced a subclinical syndrome (Cuijpers & Smit, 2004; Eaton, Badawi, & Melton, 1995; Warner, Weissman, Fendrich, Wickramaratne, & Moreau, 1992). Thus, significantly increased risk for depression is indicated by the presence of stable dysphoria, a subclinical syndrome that can



include depressive and anxious symptoms (Ingram & Hamilton, 1999).

Beyond being markers of increased risk, depression history and current dysphoria may exemplify a diathesis-stress vulnerability model compatible with contemporary conceptualizations of depressive onset. Operationalizing depression history as a diathesis trait and dysphoria as a triggered stress state facilitates the exploration of complementary premises about vulnerability processes. First, those who have experienced major depression might be distinguished, from early development on, by factors (e.g., physiological, cognitive, interpersonal) that facilitate the development of depressive episodes. It had long been proposed that any such candidate factor found in formerly depressed persons could represent a permanent change, or *scar*, resulting from a past depressive episode (Lewinsohn, Steinmetz, Larson, & Franklin, 1981), but prospective tests have not supported the scar hypothesis (Beevers, Rohde, Stice, & Nolen-Hoeksema, 2007; Shea, et al., 1996). Thus, formerly depressed individuals may possess vulnerability factors long before and after their first onset. Second, dysphoria is thought to represent more of a state-dependent influence because, although it endures far longer than a transient mood induction, it is typically far less stable than major depression. Based on this profile, investigators assess research participants at two time points separated by weeks (i.e., double gating) to ensure the presence of stable dysphoria. A third premise, following from the first two, is that a person with a history of depression and current dysphoria would possess both state and operationalizations of increased risk. From a diathesis-stress or a cumulative risk perspective, this dual-risk individual might be imminently more vulnerable to depression than a person with only one of these risk factors. Given the paucity of research that includes all three of these operationalizations of risk, there is a clear need to understand and compare mechanisms of risk in these groups.

### *Evidence of Cognitive Biases in Depression Vulnerable Populations*

It has long been proposed that biases in selective attention, interpretation, and memory exist in depression vulnerable individuals (Beck, 1967). From a cognitive diathesis-stress perspective, vulnerable individuals are thought to possess a depressive cognitive “schema” that distinguishes them from nonvulnerable individuals. When activated, this schema generates depressogenic information processing patterns that ultimately lead to the development of depression (Beck, 1967; Ingram, 1984; Ingram, Miranda, & Segal, 1998). When a depressive schema is not activated, it is characterized as “latent but reactive” to stress, such as event-triggered dysphoria (Segal & Shaw, 1986). Studies employing mood-priming have largely supported this idea of cognitive reactivity (see Scher, Ingram, & Segal, 2005; Segal & Ingram, 1994 for reviews). That is, formerly depressed individuals in a nondysphoric mood usually cannot be distinguished from never depressed nondysphoric individuals in terms of self-report or performance-based affective cognition. However, typically when each of these groups complete an affective challenge (e.g., sad mood induction), depressotypic patterns of cognition emerge only in the formerly depressed group (e.g., McCabe, Gotlib, & Martin, 2000). Although cognitive models originally conceived depressive biases to be strictly toward negative information, subsequent theories and data support the existence of biases away from positive information as well (Clark, Beck, & Stewart, 1990). Dual-valence biases appear consistent with major depressive episode presentations, which are often characterized by both increased negative affect and decreased positive affect (Clark & Watson, 1991), but these biases also may contribute to the development of depressive onsets in subsyndromal individuals.

It is worth noting that a few recent *psychophysiological* studies have reported evidence of depressotypic cognitive processing in the absence of sad mood in formerly depressed individuals

(Atchley, Ilardi, & Enloe, 2003; Atchley, Stringer, Mathias, Ilardi, & Minatrea, 2007; also see Steidtmann, Ingram, & Siegle, 2010 pre-mood induction finding). Important theoretical and methodological issues emanate from these findings. First, *active* cognitive vulnerability factors may be present in nondysphoric depression-susceptible individuals. Secondly, modern cognitive neuroscience techniques may complement traditional behavioral performance measures of cognition to increase researchers' ability to detect depressogenic patterns of information processing, such as biases in selective attention. For example, if differences in performance are not found in a study but differences in neural activity *are* observed, there could be several possible explanations. Measures of neural activity might provide better resolution for detecting differences; neural activity differences might precede the appearance of performance differences; or alternate areas of the brain might compensate to maintain normal task performance (Drummond, Gillin, & Brown, 2001; Sumich, Kumari, Heasman, Gordon, & Brammer, 2006). This idea is central to the proposed research.

Also pertinent, biases in selective attention related to processing emotional information have been found to distinguish dysphoric individuals from nondysphoric individuals (Bradley, Mogg, & Lee, 1997; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005; Siegle, Ingram, & Matt, 2002). Prior to reviewing research that has examined depressotypic attention biases toward affective facial expression stimuli, a brief primer on selective attention and inhibition is included to provide background and theoretical rationale that implicates these interrelated processes as a plausible mechanism of vulnerability and maintenance.

### *Selective Attention and Inhibition*

Selective attention requires discriminating incoming information that is relevant to a current objective from information that is irrelevant, subsequently activating the relevant and

inhibiting the irrelevant (Houghton & Tipper, 1994; Neill, Valdes, & Terry, 1995). Inhibition is also thought to be instrumental in efficient memory encoding and retrieval. In the case of deficient inhibitory processing, irrelevant information can become associated with goal-relevant material during encoding. Consequently, subsequent retrieval of goal-relevant information may also activate the associated irrelevant information.

Depressotypic selective attention has long been hypothesized to be a vulnerability mechanism for depression, but recently it has been proposed that this phenomenon could be partly due to a specific inhibitory deficit for processing depressive information. Although prior research has implicated general executive deficits in response inhibition (e.g., Kaiser, Unger, Kiefer, Markela, Mundt et al., 2003), Joormann (2004) found that on an emotion-focused negative priming information processing task, dysphoric individuals exhibited an inhibition deficiency specifically for negative words compared to positive words. Specifically, following negative distracter word primes, dysphoric individuals showed deficient inhibitory carryover from prime to test presentations, resulting in faster reaction times for negative targets as compared to positive-positive trials. In an another experiment, Joormann (2004) found that presently euthymic formerly depressed individuals exhibited significantly less inhibition to negative self-referent words than never depressed individuals. This might suggest a possible trait-like vulnerability factor specific to self-concept related processing.

Extending this putative mechanism, when an individual is focusing on emotional information, a global selective attentional bias or a specific inhibitory deficit for negative information might lead to increased elaborative processing of negative content. With time and repetition, this pattern could strengthen connections among depressive cognitive structures (Ingram, 1984). Also, when an individual attempts to focus attention on *nonaffective* task-

relevant information but previously activated negative cognitive content has been degraded (i.e., another inhibitory function) insufficiently, the negative content could linger and contaminate working memory. The remaining activated task-irrelevant negative content, or cognitive residue, could produce several maladaptive consequences. As suggested earlier, associations between task-relevant nonaffective information and irrelevant depressive information could be paired in encoding and later retrieval. Once this occurs, depressive cognitive structures could be activated and strengthened by activating depressive *or* associated nonaffective information (Linville, 1996). Also, depressive cognitive residue could *interfere* with working memory, leading to impaired performance in the kinds of complex problem solving required in daily life, ultimately increasing depressive thoughts and feelings. For this individual, the omnipresent salience of a depressive schema could override attempts to focus on nonaffective goal-relevant information processing, resulting in the subjective experience of intrusive streams of depressive thoughts. Therefore, selective attention biases for depressive information could lead to patterns of rumination, which would perpetuate depressive moods (Nolen-Hoeksema, Morrow, & Fredrickson, 1993) and deeply encode depressive memories, the combination of which might initiate or maintain depressive episodes.

#### *Selective Attention to Affective Facial Expressions and Depression Vulnerability*

Although the majority of studies investigating possible emotional information processing biases in depressive populations have used lexical stimuli, there are good reasons to employ facial expression stimuli. First, facial affect can simultaneously reveal the tenor of a social milieu and influence one's feelings (Ruys & Stapel, 2008; Wild, Erb, & Bartels, 2001). As such, an attentional bias toward sad expressions could result in disproportionately depressive moods and mental representations of the social environment. Second, direct angle emotional faces likely

trigger automatic self-referent processing (i.e., “she is glancing at me”) as well as self-relative-to-other processing (e.g., “is her reaction to me unfavorable and dominant?”). In this way, facial expressions may evoke the looking glass self, which is continually shaped by others’ reflections and responses. An attentional bias toward others’ sad, discouraged affect could thus lead to frequent negative attributions and a devalued sense of self-worth (e.g., “I make people unhappy”). Third, inasmuch as emotions are preparatory states for behavior, perceiving facial affect would ordinarily prime rapid, appropriate social reactions. Conversely, language is typically more abstract and detached from the immediate setting. Consistent with this conceptual difference, empirical evidence indicates that facial affect may be a more evocative emotional medium than words (De Houwer & Hermans, 1994; Glaser & Glaser, 1989; Vanderploeg, Brown, & Marsh, 1987). Thus, tasks that utilize affective facial stimuli might more reliably evoke pathology-specific emotional information processing biases (Gotlib, Krasnoperova, Yue, & Joormann, 2004). Fourth, and in the same vein, facial affect embodies particularly relevant information to depression-susceptible individuals negotiating complex interpersonal environments. Impairment in interpersonal functioning is a common feature in depressive syndromes and may prolong periods of dysphoria (Coyne, 1976; Segrin & Abramson, 1994; Swann, Wenzlaff, Krull, & Pelham, 1992). Negatively biased processing of interpersonal affective information might promote social isolation and rejection-eliciting behaviors.

Based on studies using behavioral measures (reaction times, accuracy/error rates), depressed individuals generally show unbiased recognition of unambiguous facial expressions of sadness and happiness (Segrin, 2001), but they can exhibit impaired recognition of subtle mildly-happy or ambiguous-neutral facial expressions (e.g., Gollan, Pane, McCloskey, & Coccato, 2008; Gur et al., 1992; Leppanen, Milders, Bell, Terriere, & Hietanen, 2004; Raes, Hermans, &

Williams, 2006; Surguladze et al., 2004). Specifically, depressed individuals tend to take longer to classify neutral faces and are more apt to mistakenly assign sad affective meaning to them than nondepressed individuals, who can show positivistic biases to neutral faces (Gollan, et al., 2008; Gur, et al., 1992; Leppanen, et al., 2004; Surguladze, et al., 2004). Therefore, at a practical level, studies that enroll depressed or depression susceptible groups to examine attention and memory for affective facial stimuli need to rule out interpretive biases as a potential confounding variable. In addition, preliminary evidence suggests that depression and perhaps depression vulnerability are associated with biased recall of sad facial expressions (Gilboa-Schechtman, Erhard-Weiss, & Jeczemien, 2002; Jermann, van der Linden, & D'Argembeau, 2008; Ridout, Astell, Reid, Glen, & O'Carroll, 2003). Given evidence of memory and interpretive biases, cognitive models would predict that mood episodes might influence attentional processing of affective facial expressions. Available evidence has supported this prediction.

A collection of studies have reported differences when comparing currently depressed, formerly depressed, or dysphoric groups to healthy control groups on various aspects of attention following presentations of affective facial stimuli. Most reported evidence has indicated that depressed individuals engage attention more efficiently and sustain attention for longer with sad faces compared to nondepressed individuals (Gotlib, Kasch, et al., 2004; Gotlib, Krasnoperova, et al., 2004; Joormann & Gotlib, 2007; Karparova, Kersting, & Suslow, 2005). However, research has also suggested that depression is associated with impaired attentional orientation with or bias away from positive faces presented at brief latencies (Gotlib, Kasch, et al., 2004; Suslow, et al., 2004; Suslow, Junghanns, & Arolt, 2001). The difference in findings appears to be a product of which stage of attentional processing is examined by the given research paradigm (see Bradley, Mogg, & Millar, 2000 for discussion). Such a dissociation could plausibly emerge

given the contemporary understanding that negative faces are detected through automatic parallel processing and happy faces are detected via serial, effortful processing (White, 1995). Therefore, because depression is characterized by intact automatic processing and impaired effortful processing (Hartlage, Alloy, Vazquez, & Dykman, 1993), attention to happy (but not sad) faces would be deficient. Also, of particular relevance to the present research, behavioral evidence has linked depression to deficient inhibition of sad facial affect (Goeleven, De Raedt, Baert, & Koster, 2006).

Results from groups at risk for depression are relatively consistent with those found with currently depressed samples. For example, Joormann and Gotlib (2007) found that at relatively long stimulus durations (1000ms), formerly depressed individuals selectively attended to sad facial expressions in the absence of any mood manipulation. This study provided evidence of a cognitive marker that persists beyond symptomatic recovery from depression, a notable exception from the collection of findings consistent with the latent-but-reactive concept (see Scher, et al., 2005 for review). Hsieh and Ko (2004) found similar biases in high trait-depressed individuals (i.e., more akin to at-risk groups than to groups with major depression) and suggested that these individuals might be exhibiting diminished inhibition specific to sad faces. In contrast, dysphoric individuals have evinced a tendency to shift initial attention away from happy faces relative to neutral faces (Bradley, Mogg, Falla, & Hamilton, 1998; Bradley, et al., 2000) when happy-neutral stimuli pairs are presented. Again, this phenomenon could represent a depressotypic deficit in orienting attention toward positive interpersonal information. Thus, similar to depressed groups, at-risk groups have shown deficient *orienting* to happy facial expressions and greater attentional *maintenance* with sad facial expressions compared to healthy control groups. Also, preliminary findings suggest that distressed depression-susceptible



individuals may lack sufficient inhibition of incoming sad facial affect in the social environment. Lastly, findings from depression/depression vulnerability studies that have examined attentional biases with emotional facial expression stimuli largely parallel studies that have used emotional word stimuli. However, while specific inhibition biases have been found for negative distracting words in depressed and at-risk groups, research has yet to discern whether a similar result emerges with sad facial expression stimuli. In summary, studies using indirect behavioral measures of cognition have linked depression vulnerability with biased attentional processing of facial affect.

Increasingly, cognitive neuroscience research techniques have been used to augment contemporary knowledge of cognitive processing biases and how they are instantiated in the brain. Cognitive neuroscience can uniquely examine mind-brain relationships, drawing connections between mental events and neural events. Such “vertical” integration across levels of analysis represents a higher-order objective in advancing scientific knowledge (Cosmides, Tooby, & Barkow, 1992; Wilson, 1999), in this case moving toward a holistic understanding of depressotypic information processing. To this end, behaviors (e.g., reaction times and errors) *and* neural activity (e.g., ERP: event-related potentials components) associated with selective attention and inhibition can be concurrently examined in depression-prone groups, with results illuminating potential psychological-physiological linkages.

#### *Neuroelectric Activity Related to Selective Attention/Inhibition and Depression Vulnerability*

Specific neural activity has been related to selective attention, and differences in this activity have been found between depressed and healthy individuals via cognitive neuroscience techniques. Because orienting, engagement, disengagement, and shifting of attention are events that take place on a brief time scale, ERP research, with its precise temporal resolution, is

uniquely positioned to help elucidate how attentional processes are instantiated in time within particular neurocognitive systems. In terms of the electrophysiological signals associated with specific event-related mental processing (i.e., ERP), *amplitude* of positive or negative deflections in the electroencephalogram (EEG) waveform is often examined. Comparing susceptible and nonsusceptible individuals with respect to attentional ERP waveform components could shed light on depression vulnerability at a neurocognitive level of analysis.

### *Selective Attention ERP Components*

ERP waveform components related to aspects of selective attention include the N200 and the P300, (including P300a and P300b variants). Based on current understanding, these components can be examined to assess attentional activation and inhibition in vulnerable and nonvulnerable individuals. In ERP research, these components are reliably evoked by Go/No-go and oddball paradigms (Bertoli & Probst, 2005; Debener, Kranczioch, Herrmann, & Engel, 2002), the latter of which was used in the present investigation.

The P300 (or P3), the third pronounced positive deflection in the waveform beginning roughly 300ms after stimulus onset, has long been an important component with respect to studying attentional processes (see Polich, 2007 for thorough review). More recently, the P3 “complex” has been sub-divided into the P3a and P3b, elements representing functionally distinct cognitive operations arising from topographically different brain regions. The P3a is a variant of the “novelty P300” potential evoked by novelty oddball tasks, which augment the traditional two-category oddball paradigm with a third task-irrelevant distracter category. The P3a is elicited by these rarely presented distracters (Debener, et al., 2002; Simons, Graham, Miles, & Chen, 2001). The P3a typically occurs between 230-360ms as frontocentrally-generated activity that may reflect automatic bottom-up processing aspects of attention, including the

orienting response (Debener et al., 2002), and/or top-down processing, as in identifying mismatches with a standard stimulus type stored in memory (Polich, 2007). Evidence suggests that the P3a is not meaningfully distinguished from the “No-Go P300” (Polich, 2007), a frontally located component thought to reflect inhibition of a planned response or conflict between competing responses (Bekker, Kenemans, & Verbaten, 2004; Bokura, Yamaguchi, & Kobayashi, 2001; Bruin, Wijers, & van Staveren, 2001; Fox, Michie, Wynne, & Maybery, 2000; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Smith, Johnstone, & Barry, 2007). Boys with ADHD and patients with Parkinson’s disease, populations with significant behavioral inhibition deficits, have both been found to exhibit diminished No-Go P300s (Bokura, Yamaguchi, & Kobayashi, 2005; Fallgatter, et al., 2004).

On the other hand, the P3b, referred to as the “target P300” in oddball paradigms, is typically elicited by the identification of a salient, rarely presented target stimulus. The P3b is a temporoparietal-generated activation that occurs between 400-580ms, and it may reflect voluntary, top-down related aspects of attention (Debener et al., 2002) and/or bottom-up aspects, as in matching a stimulus with a categorical template (Polich, 2007). The amplitude of the P3b is also thought to reflect the amount of attentional processing resources allocated to stimulus evaluation (Debener, et al., 2002; Rich, et al., 2005). Evidence suggests that P3b is a variant of the same potential that manifests as the “Go P300” (Polich, 2007), a potential evoked by the selection of a go response in Go/No-Go paradigms (Jonkman, 2006). Pertinent to experimental design, P3b amplitude tends to increase with time between target stimuli. Relevant to theoretical considerations, Polich (2007) recently proposed that P3a and P3b may represent activity of a neural circuit that, upon identifying a rare, salient stimulus, inhibits irrelevant cognition to facilitate attentional focus and memory storage (consonant with Donchin’s context-updating

model; Donchin & Coles, 1988).

In addition, an N200, or N2, component (second negative deflection peaking around 250ms) has been identified which may be related to inhibition (Jodo & Kayama, 1992; Johnstone, et al., 2007; Lavric, Pizzagalli, & Forstmeier, 2004), but of late has been linked more to conflict monitoring (Bekker et al., 2004; Donkers & van Boxtel, 2004; Jonkman, 2006). It is possible this component could be related to both inhibition and conflict monitoring, as No-Go N200 amplitudes are smaller than normal in inhibition-deficient Parkinson's patients (Bokura, et al., 2005) and larger than normal in individuals with obsessive compulsive disorder (Ruchow, Reuter, Hermle, Ebert, Kiefer et al., 2007). The latter may reflect greater response conflict or, phrased differently, greater cognitive interference. N200 amplitude has been found to be positively correlated with the probability of a target stimulus presentation, compared to stimuli to be ignored (Bekker, Kenemans, & Verbaten, 2005; Donkers & van Boxtel, 2004).

Neuroanatomically, the N2 appears to be generated by a region that connects medial frontal and anterior cingulate areas (Bekker et al., 2005; Bokura, et al., 2001). Therefore, the P3a, P3b, and N2 attributes can help illuminate neural processes that may underlie behaviorally observed interference and response inhibition phenomena in psychopathological populations, and possibly in individuals who are vulnerable to depression.

#### *ERP Components and Depression Susceptibility*

Major depression is typically associated with decreased P3 amplitude in response to neutral, nonaffective stimuli (e.g., Ancy, Gangadhar, & Janakiramaiah, 1996; Kemp, et al., 2009), which could be thought to reflect generalized, symptomatic deficits in attention and concentration. However, mounting evidence indicates that depressed individuals show normal or even elevated P3s in response to negative information on certain cognitive tasks. For example,

Ohira (1996) reported general evidence of P3 attenuation among depressed individuals, but also a specific *lack* of P3 attenuation when these individuals were responding to words with a negative emotional tone. In a later study, Ilardi, Atchley, Enloe, Kwasny and Garratt (2007) found that negative affective stimuli evoked higher amplitude P3s in currently depressed individuals than in never depressed controls and formerly depressed individuals.

The few studies that have examined attentional ERP components in depression-susceptible populations have not focused on depressotypic processing biases; however abnormal ERP patterns have been reported. For example, subclinically depressed groups have exhibited augmented parietal P3s in response to pain-related words in a lexical decision task and different asymmetrical activation profiles of the P300 (and N200) on an auditory oddball task when compared to healthy control groups (Nikendei, Dengler, Wiedemann, & Pauli, 2005; Sumich, et al., 2006). Also, children of parents that experienced childhood depression have shown larger amplitude P3s than a control group during a Posner task in which negative affect was induced via performance pressure (Perez-Edgar, Fox, Cohn, & Kovacs, 2006). Importantly, this set of findings hints at possible psychophysiological parallels between depression and depression-vulnerability. For example, vulnerable individuals experiencing distress may recruit abnormally increased neural resources while selectively attending to mood-relevant information or attempting to inhibit “affective interference” during neutral task performance (Siegle et al., 2002). Simultaneously, these individuals may allocate fewer attentional resources toward affectively neutral mental tasks that are nonetheless vital to one’s livelihood and daily functioning. As intriguing as these findings are, far more research is needed to understand the psychophysiology of various types of information processing in depression-prone groups.

Research has yet to examine whether or not elevated P3s can be detected in depressed

individuals in response to depressive *facial expression* stimuli, or in negative-schema-activated depression-susceptible individuals who do not meet current major depression criteria, in response to facial stimuli. However, Cavanagh and Geisler (2006) examined the P3 response of depressed and nondepressed students on an oddball task that presented alternating blocks of rare happy and fearful target expressions interspersed among standard neutral facial stimuli. The authors reported that, compared to the control group, the depressed group showed a reduced mean P3 to happy target faces but not fearful ones. Thus, extant P3 findings support the notion that depressotypic selective attention processing designates negative information as particularly salient and positive information as specifically non-salient. Depression vulnerability may be characterized by this same pattern of attention, which could promote extended periods of depressive cognition and mood. If this is the case, depression-susceptible groups may exhibit an increased P3 when attending to sad facial affect and an attenuated P3 to happy affect.

By comparison, the N2 has been scarcely studied with regard to depressive biases. One study reported that depressed individuals exhibited an attenuated right posterior N2 in reaction to mood-incongruent positive faces (Deldin, Keller, Gergen, & Miller, 2000). However this N2 was evoked by an affective valence identification task, not a selective attention or inhibition paradigm. Nonetheless, ERP research has the potential to help illuminate neural processes that underlie depressotypic attentional biases.

To summarize, behavioral information processing paradigms have provided strong evidence that depression and depression vulnerability can be associated with attentional biases toward affective facial stimuli. Also, there is mounting evidence that depression can be characterized by a specific inhibitory deficit for negative information. However, additional research is needed to determine whether depression susceptibility is associated with an inhibitory

deficit for sad facial affect. Reduced inhibition when encountering others' sad affect could give rise to prolonged patterns of rumination and perhaps the development and maintenance of depressive episodes. Also, abnormal attention to facial affect could potentially modulate other forms of depressogenic cognition and social behavior. Specifically, individuals whose attention is more focused on others' expressions of sadness and disappointment may be more likely to appraise and recall social interactions as more negative, to blame themselves for others' negative emotions, to criticize their own faults, and to isolate themselves from others (Frewen & Dozois, 2005; Joiner & Rudd, 1995; Lewinsohn, 1974; Persad & Polivy, 1993).

Furthermore, the mechanisms, or neural systems, that instantiate attentional biases are poorly understood and have been insufficiently studied with tools that can help elucidate processing patterns. Cognitive neuroscience can concurrently examine behavioral and psychophysiological correlates of attention in depression-prone groups, presenting the valuable opportunity to integrate knowledge from psychological and biological levels of analysis. Lastly, the question of whether state-, trait-, or state-and-trait-related operationalizations of risk present with homogeneous versus differential attention biases remains largely unresolved. Research that compares groups representing each of these operationalizations of risk on attentional measures is needed.

### *The Present Research*

In line with the previously identified empirical questions, the present study examined whether individuals from various groups at-risk for developing depression exhibit selective attention and inhibition biases for affective facial expressions as compared to low-risk individuals. A 2 x 2 x 2 mixed design yielded four sample groups with two levels of dysphoria status (nondysphoric vs. dysphoric) and history of depression (past major depression vs. none),

enabling comparisons of relative risk across groups and within groups (e.g., control vs. experimental task). Double-gated assessments were employed to determine the presence and stability of dysphoria and the existence of past major depressive episodes. Rumination and present emotional state were also assessed. Qualifying participants completed two visual oddball tasks, which elicited and measured behavioral and neuroelectric responses reflecting inhibition and activation aspects of selective attention. In the affective experimental task, participants were to respond or inhibit a response to infrequently presented sad or happy facial expressions (i.e., rare target or distracter) in the context of frequently presented neutral faces. A non-affective control oddball condition was also included to clarify whether any found inhibitory deficits would be specific to depressive affective information processing. This non-affective task required participants to respond only to facial stimuli that fit into one of three age categories. Behaviorally, faster reaction times (e.g., Joormann, 2004) or erroneous responses to a particular facial affect (i.e., responding to a sad face when instructed to respond only to happy faces) may identify a relative inhibitory deficit. Psychophysiologicaly, attenuated N2 or P3a or elevated P3b amplitudes may identify greater interference, inhibition processing, or focused attention. Therefore, given that the brain can recruit compensatory neural resources to minimize impairment in behavioral performance (Drummond, et al., 2001; Sumich, et al., 2006) depression vulnerable individuals' behavioral performance and neural activity linked to inhibitory processing were concurrently measured.

Because all groups who are susceptible to depression might not be characterized by similar attentional processing biases, this study included three vulnerable groups. A key question to be assessed was whether behavioral and neuroelectric indicators of cognitive bias are dependent on mood-state activation of (i.e., occurring in currently dysphoric groups) or whether trait-but-not-



state vulnerability status (e.g., formerly depressed but currently nondysphoric group) is related to biased processing when focusing on emotional content. Therefore, the present study examined possible state, trait, and state-trait interactive contributions to information processing.

### *Hypotheses*

Based on the reviewed literature, state-by-trait interaction effects were generally predicted such that the currently dysphoric formerly depressed group would show evidence of inhibitory deficits for sad distracter faces and facilitated processing of sad target faces compared to nondysphoric groups. Also, a small collection of findings suggests that currently dysphoric formerly depressed individuals would attend less to happy facial affect (e.g., Bradley, et al., 1998; Bradley, et al., 2000; Karparova, et al., 2005; Nandrino, Dodin, Martin, & Henniaux, 2004). Therefore, the present study expected that the currently dysphoric formerly depressed group would show evidence of reduced selective attention to happy target faces than the low-risk nondysphoric never depressed group. More specific predictions are discussed below and catalogued in Table 2 and Table 3 in Appendix B.

### *Behavioral Indicators of Inhibitory Bias*

For a depression susceptible individual, seeing the sad facial expression of another person might activate related depressive self-schemas, which could facilitate further relevant processing of depressive content or potentially cause interference when depressive content is irrelevant to the task at hand. In the context of a visual oddball paradigm where dysphoric participants are either to respond or inhibit responses to presented sad or happy faces, one might expect to see behaviorally facilitated responding to sad faces as compared to happy faces. This effect would be further pronounced if dysphoric participants were to insufficiently degrade task-irrelevant depressive content, as it could persist in working memory, leading to greater interference effects

when dysphoric participants must respond to subsequently presented happy faces. Thus, it was expected that the dysphoric formerly depressed group would respond more quickly to sad faces and more slowly to happy faces compared to nondysphoric never depressed individuals (e.g., Siegle, et al., 2002). Also, it was expected that dysphoric formerly depressed individuals would exhibit more errors of commission to sad faces when instructed to respond only to happy stimuli.

#### *Psychophysiological Indicators of Inhibitory Bias*

In terms of ERP analysis, the dysphoric formerly depressed group was predicted to evince a significantly higher amplitude P3b for task-appropriate responses to sad faces compared to happy faces. This pattern was not expected in the nondysphoric group. It was also predicted that the dysphoric formerly depressed group would exhibit a higher amplitude P3b for sad target trials compared to the nondysphoric never depressed group.

Additionally, it was thought that analysis of ERPs in dysphoric individuals might reveal a lower amplitude P3a for task-irrelevant sad faces than for task-irrelevant happy faces. This would be in line with the previously hypothesized deficit for inhibiting negative information during emotion-focused information processing (Joormann, 2004). Similarly, N2 amplitude was expected to be lower for task-irrelevant-sad than irrelevant-happy faces among dysphoric individuals.

#### *Indicator of Nonemotional Information Processing*

Consistent with research showing attenuated P3s in depressed individuals, dysphoric groups would be expected to exhibit P3b attenuation during the nonemotional oddball task. Alternatively, from a cognitive reactivity framework, P3b attenuation would only be expected when a diathesis (i.e., trait predisposition) and stress (depressed mood state) are present, in which case, only the currently dysphoric, formerly depressed group would show an attenuated P3b.

## Method

### *Participants*

In order to enroll the present qualifying sample, 155 participants were originally recruited. Ultimately, 55 qualifying participants met inclusion criteria and provided data that were adequate to analyze. Each diagnostic group included 14 participants, with the exception of the dysphoric never depressed group, which included 13 (see group characteristics below in Table 1). Groups of this size have provided sufficient statistical power to detect between-group differences in studies comparing P300 amplitudes of depressed and nondepressed samples (Diner, Holcomb, & Dykman, 1985; Murthy, Gangadhar, Janakiramaiah, & Subbakrishna, 1997; Pierson, et al., 1996; Torta, Borio, Cicolin, Vighetti, & Ravizza, 1994).

*Table 1. Descriptive characteristics of the sample*

	Dysphoric Formerly Depressed	Dysphoric Never Depressed	Nondysphoric Formerly Depressed	Nondysphoric Never Depressed	All Participants
N	14	13	14	14	55
Age in years	19.29 (1.20)	19.62 (2.47)	21.79 (7.74)	18.86 (0.86)	19.89 (4.20)
Female	50.00% (7)	61.53% (8)	71.42% (10)	57.14% (8)	60.00% (33)
African American/Black	7.14%	0%	0%	0%	1.82%
Asian American/Pacific Islander	0%	15.40%	0%	0%	3.64%
Caucasian American/White	92.86%	84.60%	100%	100%	94.54%
Prescreen BDI-II score	16.43 (7.59)	19.00 (6.15)	3.36 (4.40)	3.93 (5.62)	10.53 (9.25)
Experimental session BDI-II	17.43 (3.67)	16.31 (4.21)	3.79 (1.93)	2.07 (1.27)	9.78 (7.66)
Ruminative Response Scale	18.64 (4.91)	17.23 (3.24)	14.86 (5.72)	11.86 (4.37)	15.62 (5.24)
POMS Tension-Anxiety	5.71 (4.23)	4.54 (2.88)	1.57 (1.22)	1.23 (1.48)+	3.28 (3.30)++
POMS Depression-Dejection	3.85 (2.38)+	2.15 (2.04)	0.21 (0.58)	0.29 (0.61)	1.57 (2.16)++
POMS Anger-Hostility	0.93 (1.27)	1.15 (1.86)	0.00 (0.00)+	0.21 (0.58)	0.57 (1.22)++
POMS Vigor-Activity	5.50 (4.36)	3.85 (3.39)	7.29 (5.20)	6.64 (3.34)	5.85 (4.24)
POMS Fatigue-Inertia	6.86 (3.13)	6.62 (4.03)	2.57 (2.17)	3.29 (1.98)	4.80 (3.44)
POMS Confusion- Bewilderment	4.71 (2.73)	4.77 (1.79)	3.00 (0.78)	2.57 (1.16)	3.75 (1.99)

+Based on n = 13 due to missing data

++Based on n = 54 due to missing data

*Recruitment.* Participants were recruited from a pool of students completing research participation for introductory psychology course credit. Potential participants had already completed online demographic and psychological prescreening measures and had provided contact information. Potential participants who met preliminary eligibility criteria signed up to

participate via an online research system, or they were contacted by email or by phone. If they were interested in participating, a session was scheduled. To balance any gender effects across experimental groups, the proportion of each gender was kept roughly equal in each experimental group.

*Inclusion criteria.* To qualify, participants needed to be right-handed and have no history of neurological disorder or brain insult resulting in loss of consciousness. Also, participants were required to meet criteria for one of the following groups: currently dysphoric never depressed, currently dysphoric formerly depressed, currently nondysphoric formerly depressed, or currently nondysphoric never depressed. The currently dysphoric groups needed to score in the dysphoric range (i.e., 12-21) of the Beck Depression Inventory, Second Edition (BDI-II)(Beck, Steer, & Brown, 1996) at two time points (the first time point used projected scores from a 7-item short form BDI). This range maximized sensitivity to dysphoria, with a slightly elevated ceiling cut-point justifiable because current major depressive disorder was ruled out. The first time point was part of the previously mentioned online collection of measures that participants completed several weeks before the experimental session. The second time, in the experimental session, participants completed a modified version of the BDI-II (Life Stress Inventory; Hunt, Auriemma, & Cashaw, 2003), which includes additional items shown to limit underreporting of depressive symptoms. This version was chosen so as not to limit selection bias that can be introduced by preferentially enrolling students who *more openly* endorse depressive symptoms. Scoring in the dysphoric range at both time points demonstrated both the stability and presence of dysphoric mood in participants at the time of the experiment. To qualify in the nondysphoric never depressed group, participants needed to score lower than 7 on the BDI-II at both time points and fail to meet DSM-IV criteria for a past or present major depressive episode.

To qualify in either of the formerly depressed groups (i.e., with or without current dysphoria), participants needed to meet DSM-IV criteria for a past major depressive episode, based on an administration of the past mood disorders module of the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 2003). Participants were ineligible if they reported a history of mania/hypomania or any current psychiatric or health problems that might interfere with research participation. Lastly, participants needed to be at least 18 years old to participate in the experiment.

### *Experimental Stimuli*

#### *Selection Process*

Ethnically diverse facial stimuli were selected from the NimStim (Tottenham, Tanaka, Leon, McCarry, Nurse et al., in press), University of Pennsylvania (Erwin, Gur, Gur, Skolnick, Mawhinney-Hee et al., 1992; Gur, Sara, Hagendoorn, Marom, Hughett et al., 2002), Karolinska Directed Emotional Faces (Lundqvist, Flyt, & Ohman, 1998) and the Productive Aging Laboratory lifespan (Minear & Park, 2004) sets. Relatively unambiguous stimuli were pre-selected to minimize group interpretation differences. For the *non*-affective age group identification control task, each stimulus face falls into one of three age groupings of adults (younger: 18-25; middle: 40-60, older: 75-95). For the experimental task, piloting was conducted to select groups of faces that were consistently identified as falling in one of three affect groups (e.g., sad, happy, neutral), irrespective of dysphoria status or history of depression. In the piloting procedure, 417 facial stimuli were presented over four blocks to five participants representing each of the four cells of the previously described 2 x 2 design (current dysphoria status by past depression status). For any given stimulus to be incorporated into the experimental task set, it had to be coded accurately (i.e., consistent with established norms of the sets) at a rate

of at least 60% by each of the four groups (as in Hsieh & Ko, 2004), and its accuracy rate could not be significantly different based on dysphoria status, past depression status or an interaction of the two. Therefore, stimuli that met the minimum accuracy threshold were subjected to 2 x 2 analyses of variance, with dysphoria status and past depression status as independent variables and accuracy as the dependent variable. Because the small sample cells provided relatively weak power to detect between-group differences, stimuli indicating a difference, using a stringent alpha level of .15, were eliminated. From these procedures 60 happy, 60 neutral, and 60 sad qualifying facial expression stimuli were selected for the two affective valence identification task blocks (30 of each category per block). Therefore, happy and sad faces, whether presented as targets or distracters, were always novel to a participant completing the oddball tasks.

#### *Task-Relevant Characteristics*

The final set of stimuli was sufficiently large enough to enable each target and distracter stimulus to be presented only once during the oddball tasks so that behavioral and physiological measures derived from single (i.e., non-repeating) exposures to novel stimuli. Also, each set included sad, happy, and neutral expressions of the same actors. This controlled for level of attractiveness and other physical features extraneous to affect-related expression changes. Each of the four task blocks consisted of 30 target, 30 distracter, and 240 standard stimuli. Within each block, gender of depicted actors was balanced. The task blocks were administered in such an order that affective blocks alternated with non-affective age-related blocks (i.e., target = happy, older, sad, younger). The decision of which ordering permutations to use for each participant came from a pre-determined block design that minimized any order or fatigue effects and distributed them equally across the four risk groups. Also, two affective stimuli sets—each including 30 unique happy and 30 unique sad facial expressions—were incorporated in the

aforementioned block design so that each affective stimulus was equally likely to appear as a target or a distracter for participants in each of the four diagnostic groups (see Appendix A). Within each oddball task block the ordering of stimulus presentations was pseudo-random with overall proportions of 80% standard stimuli, 10% rare targets and 10% rare distracters.

### *Experimental Procedure*

Prior to arriving to participate, study participants had completed an online prescreen including short form measures assessing current dysphoria and past depression. The participant's responses on these measures were a preliminary indication of potential qualification into one of the four recruitment cells described earlier. Participants came in for a single experimental session. Upon arriving, participants were asked to provide informed consent to participate. Participants needed to meet eligibility criteria or they were debriefed and released. They completed measures of dysphoria (BDI-II), rumination (Response Styles Questionnaire; RSQ)(Nolen-Hoeksema, et al., 1993), and a clinician administered assessment of past or present depression (SCID). Qualifying participants were then fitted with an ERP net and were seated in front of a computer monitor in a sound-attenuated room. They were instructed to remain still during the experiment and used a chinrest to hold their head steady, 41.9 cm from the monitor.

Next, participants completed experimental and control visual oddball tasks with instructions to keep their eyes fixated on the cross in the middle of the screen during the experiment. For the oddball tasks, participants were instructed to respond as quickly as possible to rarely presented target stimuli and ignore rarely presented distracter stimuli in the context of frequently presented stimuli of a third type. Each of these task conditions was preceded by a short practice block to familiarize participants with the task. On practice blocks, the computer program provided feedback (e.g., "correct" or "incorrect") based on the participant's response.



During the task, a fixation cross appeared for 250ms. Each stimulus was presented in the center of the screen for 750 ms and was replaced by a mask for 1000ms. Each task block lasted approximately 11 minutes, and participants were given short breaks between the four blocks.

In the experimental condition blocks, the rare stimuli were happy and sad facial expressions, and the frequently presented stimuli were affectively neutral faces. Each participant completed one block where happy faces were targets and one where sad faces were targets. Each participant also completed two nonaffective control oddball condition blocks in which the rare stimuli were younger and older faces, while frequently presented stimuli were “middle” in age. Within each block, stimuli were presented pseudo-randomly so that rare targets were separated by between 4-16 non-target stimuli to maximize P3b responses to target stimuli (see Polich, 2007 review for discussion of probability and timing effects on P3). Also, in order to minimize lateralized pre-motor ERPs associated with button presses, laterality of response hand was counterbalanced across participants (see Appendix A).

### *Measures*

*Beck Depression Inventory, Second Edition* (see Appendix C). The BDI-II (Beck, Steer, & Brown, 1996) is the most recent version of the BDI, a widely used self-report depression instrument with considerable reliability and validity data to support its use. Although the content of the 21 BDI-II items was updated to correspond with DSM-IV depression criteria, and total BDI-II scores tend to be significantly higher (i.e., by about 2 points) than BDI scores, psychometric properties of the two measures are very similar. As such, the BDI-II has demonstrated robust internal consistency, and its items correlate highly with BDI items that assess the same underlying symptoms (e.g., sadness, pessimism, indecisiveness) (Beck, Steer, Ball, & Ranieri, 1996; Dozois & Dobson, 1998). Likewise, the BDI-II has demonstrated a high

degree of convergent validity with other depression measures, but less impressive discriminative validity. That is, the BDI and BDI-II are sensitive measures of syndrome depression, but they are not specific to depression (Beck, Steer, Ball, & Ranieri, 1996; Kendall & Flannery-Schroeder, 1995). For example, aggregated high scores on the BDI and BDI-II are to a lesser extent correlated with anxiety. It has been proposed that the BDI is truly measuring dysphoria (Beck, Steer, & Garbin, 1988), which is a core element of depression (Frank et al., 1992; Ingram & Wisnicki, 1999). Thus, by extension, the BDI-II is also measuring dysphoria. Scores on the BDI-II can range from 0-63, with scores between 13 and 19 indicating dysphoria, and scores between 20 and 63 indicating depression (Dozois & Dobson, 1998).

*Life Stress Inventory* (see Appendix D). The Life Stress Inventory (LSI) is a modified “covert” version of the BDI-II that was created to examine whether socially desirable responding leads to underreporting of depressive symptoms (Hunt, et al., 2003). The LSI includes 14 additional items about relatively socially-benign stressors (e.g. “traffic often irritates me). Hunt and colleagues (2003) found that average sum scores from BDI-II items were three points higher on the covert LSI version than on an overt version (which included 14 additional items from the Zung Self-Rating Scale for Depression), suggesting that underreporting was curbed by the measure that evoked less fear of stigmatization.

*Ruminative Response Scale* (RRS; see Appendix E). The RRS is a self-report scale within the commonly used Response Styles Questionnaire (Nolen-Hoeksema, et al., 1993). The short form utilized in this study consisted of 8 factor-analyzed items that remained after items referring overtly to depressive symptoms were removed (e.g., Roberts, Gilboa, & Gotlib, 1998). Five items load onto an introspection/self-isolation factor, while the other three items load onto a self-blame factor. Each item lists a possible response to a sad mood state and asks the respondent

how frequently he or she acts this way. Item responses on the four-point Likert scale range from “almost never” to “almost always.” Scores on the short form can range from 0-24. The complete 21-item version of the RRS has demonstrated adequate internal consistency (Nolen-Hoeksema & Morrow, 1991), reliability, and validity (Roelofs, Muris, Huibers, Peeters, & Arntz, 2006). For example, participants’ RRS scores have been shown to correlate strongly with their use of ruminative responses to depressed moods in a 30-day diary (Nolen-Hoeksema, Morrow, & Fredrickson, 1990).

*Profile of Mood States-Brief (POMS-B; see Appendix F).* The POMS-B is a 30-item short form that is commonly used in medical settings to track short-term mood changes in patients. The POMS-B consists of the five original POMS items that load highest on the six mood factors derived from factor analyses (McNair, Loor, & Droppleman, 1992). Mood factors include tension-anxiety, depression, anger-hostility, vigor-activity, fatigue, and confusion-bewilderment. Items are rated on a five-point Likert scale (0-4) from “not at all” to “extremely.” Items are summed into their appropriate POMS-BF factor scores, which range from 0-20. The POMS-B has demonstrated reliability and validity as a sensitive instrument to short-term mood changes (Yeun & Shin-Park, 2006). Also, POMS subscales such as tension-anxiety, depression, and anger-hostility have been found to correlate with emotion-typical changes in physiology (Pollock, Cho, Reker, & Volavka, 1979).

*The Structured Clinical Interview for DSM-IV-I, Non-patient Edition (SCID-I/NP; see Appendix G).* The SCID-I/NP is a semi-structured interview used (First, Spitzer, Gibbon, & Williams, 2002) to make DSM-IV-TR diagnoses (American Psychiatric Association, 2000). The SCID consists of modules, which correspond with DSM-IV axes and classes of disorders. Each module is constructed as a schematic algorithm that leads to a diagnostic conclusion, based upon

the examinee's reporting and the clinician's judgment. As noted earlier, the SCID was used chiefly to assess for current and past mood episodes. Therefore, only the unipolar and bipolar depression modules of the SCID-I/NP were administered. The depression portion of the SCID has been shown to be reliable (Zanarini & Frankenburg, 2001; Zanarini, et al., 2000).

### *Collecting and Cleaning Oddball Task Data*

#### *Behavioral Data*

The visual oddball tasks were created with E-prime (Psychology Software Tools), which enables the precise measurement of reaction times and recording of correct and incorrect responses for later analysis. The tasks were presented on a Dell Dimension 8300 PC. With respect to response accuracy, participants needed to respond correctly to target stimuli considerably better than chance (60% correct) to be included in later analyses. This criterion was met by all participants who completed the affect identification tasks. For the reaction time analyses, only correct trials with response latencies between 100-1750 ms were included (participants only had a 2000ms inter-stimulus interval) to eliminate irrelevant outlying responses. This resulted in the removal of only 1 target trial, or .02% of target trials overall.

#### *Electrophysiological Data*

In line with a study by O'Hare (2007), EEG data were collected using a high-density, 128-channel Electrical Geodesics, Inc. system with Geodesic Sensor Nets, version 2.1. Electrodes were placed above and below the left eye and at the outer canthi to monitor blinks and eye movements (electro-oculogram; EOG). Electrode impedances were measured using a criterion of 50k ohms, per manufacturer guidelines. Data were recorded with bandpass cutoffs of .1 to 100 Hz, digitized online with a sampling rate of 250 Hz. Prior to segmentation, EEG data were filtered using a 30 Hz lowpass filter. EEG waveforms were time-locked to each stimulus onset,

beginning 200 ms before the stimulus onset and 1000 ms after stimulus onset. A baseline correction was applied to the 200 ms period before stimulus onset. An average reference transform was applied to estimate reference-independent ERP waveforms (Bertrand, Perrin, & Pernier, 1985; Dien, 1998a).

Eye blinks and movement artifacts were eliminated with an automatic independent components analysis (ICA) script using EEGLAB (Delorme & Makeig, 2004). Electrode channels were marked as “bad” for any trial if the fast average amplitude exceeded 200  $\mu\text{V}$ , if the differential average amplitude exceeded 100  $\mu\text{V}$ , or the channel lacked variance. A trial was identified as bad if it included more than 12 bad channels or had EOG activity (eye movement artifacts) in excess of 70  $\mu\text{V}$  even after the ICA routine had removed related variance. A channel was identified as bad across the whole session if it was documented to be bad in more than 25% of the trials. Bad channels were interpolated from nearby channels by using spherical splines. The described automated criteria were supplemented by visual analysis and editing. Per common convention, data from a participant was excluded from analyses if 60% or more of the trials in any block were marked bad. This resulted in the elimination of 6 participants from analyses.

#### *Data Analyses*

The first step of the analysis was to examine the collected data, checking for outliers, and ascertaining whether assumptions were met for appropriate statistical tests. Tests were conducted to ensure that experimental groups were roughly equivalent on appropriate baseline variables (e.g., age, gender, ethnicity). For any significant difference found, the variable in question was statistically controlled in subsequent analyses as a covariate and considered in interpretation of analyses.

### *Behavioral Analyses*

Response accuracy scores were examined to ensure engaged task performance as well as to rule out differential difficulty between the experimental and control tasks (task type) or within the experimental tasks (i.e. detection of sad versus happy targets). Thus, a 2 x 2 x 2 x 2 mixed ANOVA with gender, dysphoria status, depression history and task type as independent variables was conducted on response accuracy; a subsequent analysis substituted task type for valence as the within-subjects independent variable. Also, to examine affective response inhibition, errors of commission to sad distracters were compared across groups.

Reaction time data for analysis consisted of the median reaction times for each condition block (e.g. target: sad; target: happy; target: older; target: younger) for each participant. To account for any non-condition-specific effects, a preliminary 2 x 2 x 2 ANOVA with gender, dysphoria status, and depression history as independent variables was conducted on reaction times collapsed across all four condition blocks. Next, to examine general emotional versus nonemotional processing in the various groups, task was included as an additional within-subjects variable (i.e., affect vs. age evaluation). Following these preliminary analyses, valence (happy vs. sad) was substituted for task as a within-subjects factor in the 2 x 2 x 2 x 2 ANOVA. Finally, an exploratory ANOVA was used to examine possible links between reaction time and error of commission (responses to distracters) findings.

### *ERP Analyses*

As with all subsequent ERP component analyses, mean voltage was evaluated as a dependent variable across the temporal window and spatial location appropriate to each component.

*Identifying and assessing ERP components.* To isolate the primary ERP components associated with attention to emotional facial expressions, two analytic techniques were used. First, to examine the typically robust “P3 complex”, a traditional windowed analysis was conducted on individual average files. *A priori*, the time window of 350-650 ms was selected, and the spatial cluster encircling and including electrode 62 (roughly corresponding with a Pz location on a low-density EEG net) was selected for analysis. To examine the N2 component, the 210-330 ms temporal window and the spatial cluster centering on electrode 11 (also including 4, 5, 6, 12, 20) were selected. ANOVA was used to determine whether the general P3 or N2 components were robust enough for between-groups comparisons.

Additionally, to isolate the N2, P3a, and P3b components, a temporal-spatial principal components analysis (PCA) was conducted using the Matlab ERP PCA Toolbox 1.093. Voltage readings at each of the time points served as variables for the initial temporal PCA, which utilized promax rotation. The scree plot from this PCA suggested 6 temporal components be kept, accounting for 94.91%. A follow-up spatial Infomax independent components analysis (ICA) was conducted on each temporal component score to differentiate them (Dien, Spencer, & Donchin, 2003; Spencer, Dien, & Donchin, 1999), employing the script from EEGlab (Delorme & Makeig, 2004). This ICA suggested 8 spatial components and accounted for 72.66% of the variance. Combined the temporo-spatial PCA divided the data into 48 factors. Each factor was displayed on a net layout for visual inspection, and factors fitting a clear characteristic eye movement profile were removed from further analysis. This resulted in the elimination of 32 factors, leaving 16 remaining factors at that point. Next, it was necessary to match found factors to the documented spatial locations of the three ERP components of interest or to discard non-matching factors. The N200 was expected to derive from frontal electrode sites around 210–330

ms following distracter stimuli; the P300a from frontal and frontocentral sites 250-400 ms after distracter stimuli; and P300b from parietal and centroparietal sites 350-650 ms after target stimuli. Only 6 temporo-spatial factors fit broad spatial criteria of the ERP components of interest. The proportion of the grand average accounted for by each factor was reconstructed for interpretation and analysis (Dien, Tucker, Potts, & Hartry-Speiser, 1997).

For each participant, the P3(b) value consisted of the mean amplitude from standard trials subtracted from mean amplitude of target trials. The P3a value derived from subtracting standard trials from distracter trials. The N2 value derived from subtracting target trials from distracter trials. To be considered valid, each proposed component value's sign (+/-) needed to match its archetypal component's sign and to show significantly different values in appropriate trial contrast values. For instance, a P3b candidate factor needed to show significantly greater positive mean amplitude on target trials than standard trials. Thus, for each of the 6 factors, ANOVA and a Bonferroni correction for multiple comparisons were used, with an adjusted alpha of .008 (i.e., .05/6 factors).

*ERP comparisons based on risk factors.* To account for any between-groups non-condition-specific effects, a preliminary 2 x 2 x 2 ANOVA with gender, dysphoria status, and depression history as independent variables was conducted on targeted ERP components collapsed across all four condition blocks. After this, to examine general emotional versus nonemotional processing in the various groups, task (affect vs. age evaluation) was included as an additional within-subjects variable.

Planned analyses utilized two theory-driven approaches. First, a set of specific *a priori* between-groups contrasts were tested based on the assumptions that currently dysphoric formerly depressed group represents highest risk, and that the nondysphoric never depressed group



represents lowest risk. These contrasts (listed in the Table 2 and Table 3 of Appendix B) are theoretically linked by a common inhibitory process (hypotheses 1 & 5), a common mechanism of facilitated engagement with negative information (hypothesis 2 & 6), a common mechanism of attenuated orientation to positive information (hypothesis 3 & 6), and a common attenuation of nonaffective target-related activation (hypothesis 4). Secondly, the possibly differentiable and interacting effects on ERPs of dysphoria, depression history, and gender were explored via ANOVA. To start, a 2 x 2 x 2 x 2 ANOVA, with valence as a within-subjects variable and depression history, dysphoria status, and gender as between-subjects variables was conducted for ERP components of interest. Next, valence was removed from the ANOVA, and a 2 x 2 x 2 was conducted directly on specific ERP components of interest (e.g., sad P3).

## Results

### *Self-Report Measures*

#### *Demographic measures*

Analysis of variance indicated that age did not vary significantly on the basis of dysphoria status, depression history, gender, or any interaction among these variables. Regarding gender, the four subgroups ranged between 50-71% female. Despite these relative differences, a Pearson chi-square analysis indicated that females were not significantly more likely to be included in any one of the four recruited groups (dysphoria status x depression history) than the others (all  $p > .24$ ). Nonetheless, gender was included in analyses to examine potential gender effects. With respect to racial status endorsed, each of the four groups was predominantly Caucasian, with only three non-Caucasian participants. No qualifying participants self-identified as Hispanic/Latino, Native American, or multiracial. Fisher's exact tests indicated that no racial group was preferentially more likely to be included in any one the four recruited groups (all  $p \geq$

.50).

### *Dysphoria*

The dysphoric groups' mean experimental BDI-II score for the preceding two weeks did not differ significantly from their mean prescreen BDI-II score (see Table 1 for group means), supporting a moderate degree of stability across the intervening time period ( $M = 45.70$  days,  $SD = 27.56$ ). The level of BDI-II dysphoria endorsed did not significantly differ between the dysphoric formerly depressed group and the dysphoric never depressed group. Level of dysphoria also did not differ between the nondysphoric subgroups. No main or interaction effect of gender was found for BDI-II dysphoria.

### *Rumination*

A significant main effect of dysphoria status was found,  $F(1, 48) = 12.10, p = .001$ , with dysphoria associated with greater rumination (see dysphoric group means in Table 1). A nonsignificant trend was found for depression history,  $F(1, 48) = 3.23, p = .08$ , such that past depression status was linked to marginally greater levels of rumination.

### *Present Mood State*

Immediately before completing information processing tasks, dysphoric participants endorsed on the POMS significantly higher levels of tension-anxiety, depression, anger-hostility, fatigue, and confusion-bewilderment than nondysphoric participants (all  $p < .005$ ). Dysphoria was also associated with lower vigor-activity subscale scores,  $F(1, 47) = 4.02, p = .05$ . Currently dysphoric never depressed males endorsed the lowest vigor-activity levels ( $M = 2.80, SD = 2.39$ ). In addition, depression history by dysphoria status interaction trend was found for the POMS depression subscale,  $F(1, 48) = 3.55, p = .07$ . No main or interaction effect of gender was found for any POMS subscales.

## *Oddball Task Accuracy*

### *Preliminary Analyses*

*Was response accuracy acceptable and consistent across response types for all groups?*

The overall accuracy across oddball task conditions was 97.18% (2.11%). In accordance, rare target responses ( $M = 91.50\%$ ,  $SD = 5.67\%$ ) and distracter non-responses ( $M = 98.71\%$ ,  $SD = 1.29\%$ ) also exhibited robust accuracies. Interestingly, accuracy for frequent standard stimuli ( $M = 97.41\%$ ,  $SD = 2.43$ ) was lower than for distracters with a statistically significant, though relatively small, difference,  $F(1, 47) = 17.45$ ,  $p < .001$ . Accuracy across response types did not vary based on gender and other risk variables.

*Were risk variables associated with accuracy differences based on emotional valence of target stimuli?* Consistent with pilot data selection procedures, accuracy for specific sad and happy targets did not vary based on gender, dysphoria status, depression history or interactions among these variables.

### *Planned Analysis*

*Were risk variables associated with increased inhibitory failures (errors of commission) for sad distracter faces?* First, a significant main effect of affective task order needed to be controlled for first,  $F(1, 38) = 4.81$ ,  $p = .03$ . Consistent with predictable perseveration mistakes (i.e., correctly responding to a type of affect in one block, then incorrectly failing to inhibit a response to the same affect in a later block), more errors of commission to sad distracters occurred in happy target blocks that followed sad target blocks ( $M = 0.81$ ,  $SD = 0.86$ ) than vice versa ( $M = 0.35$ ,  $SD = 0.71$ ). Controlling for affective task order, a significant effect of dysphoria on commission errors was observed among females, but not males (notably, level of

dysphoria did not differ between dysphoric females and males,  $t(25) = .62, p = .54$ .

Specifically, dysphoric females committed more errors ( $M = 0.87, SD = 0.92$ ) than nondysphoric females ( $M = 0.28, SD = 0.57$ ) when presented sad distracter faces,  $F(1, 25) = 5.46, p = .03$ .

Although this difference only emerged in females, it appears consistent with a depressive emotion-specific inhibitory deficit. Based on the pilot study in which errors rarely involved a polar switch in affect appraisal (e.g., from sad to happy), these errors likely reflect inhibition failures to task-irrelevant sad faces. Further support is reported with reaction time results.

### *Oddball Task Reaction Time*

#### *Preliminary Analyses*

*Did general reaction time vary based on gender and other risk variables?* When collapsing across task variable levels (younger, older, happy, sad targets), a trend gender by dysphoria status by depression history interaction,  $F(1, 40) = 3.03, p = .09$ , was found. This interaction was further examined, revealing three significant simple main effects. Never depressed currently dysphoric males exhibited slower reaction times ( $M = 673.73$  ms,  $SD = 28.38$ ) than formerly depressed currently dysphoric males ( $M = 540.34$  ms,  $SD = 33.57$ ),  $F(1, 40) = 9.21, p = .004$ , never depressed nondysphoric males ( $M = 562.80$  ms,  $SD = 25.38$ ),  $F(1, 40) = 8.49, p = .006$ , and never depressed currently dysphoric females ( $M = 569.21$  ms,  $SD = 21.98$ ),  $F(1, 40) = 8.48, p = .006$ .

Notably, the never depressed currently dysphoric group included only five participants, and the increased reaction times across all tasks were particularly influenced by two of these participants. However, these two participants' reaction time values were within three standard deviations of the full sample mean, indicating they did not meet a commonly accepted criterion to be considered outliers. Moreover, all five participants showed relatively greater mean reaction

times than the other seven subgroup means, implying the effect was not merely driven by outlying values. No other subgroup contrasts were significantly different.

*Were risk variables associated with different reaction times for emotional information versus nonemotional information?* The within-subjects variable of task type (emotional identification vs. age identification) showed no significant main effects or interactions with gender, dysphoria status, or depression history.

#### *Planned Analyses*

*Were risk variables associated with reaction time differences based on emotional valence?* Differences found were directly consistent with the general gender by depression history by dysphoria interaction reported earlier, with never depressed currently dysphoric individuals exhibiting significantly slowed reaction times for sad and happy targets. No other between-subgroup contrasts or within-subject contrasts (e.g., sad versus happy valence) were significantly different.

#### *Exploratory Analyses*

*For dysphoric females, was reaction time on sad errors of commission shorter than for correct affective targets?* Further indicating emotion-specific disinhibition (or impulsivity), dysphoric females' reaction times on sad distracter errors of commission ( $M = 531.89$  ms,  $SD = 52.28$ ) were relatively faster than their correct responses to sad targets ( $M = 562.98$ ,  $SD = 46.51$ ) and happy targets ( $M = 601.63$  ms,  $SD = 80.18$ ). The latter difference approached statistical significance,  $F(1, 8) = 4.61$ ,  $p = .06$ .

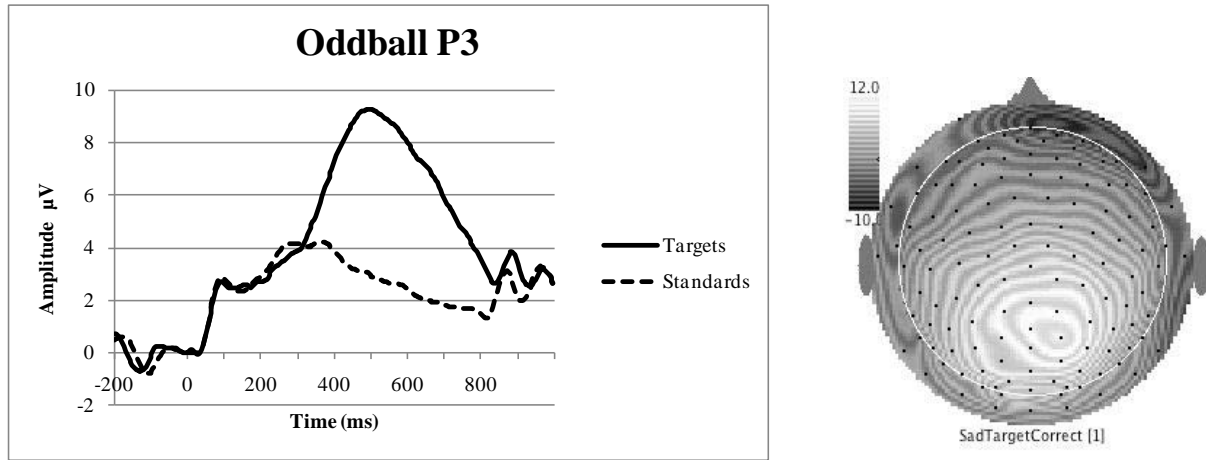
#### *Oddball Task Event-Related Potentials*

#### *Preliminary Analyses*

*Were ERP components of interest reliably found?* Some ERP components were reliably

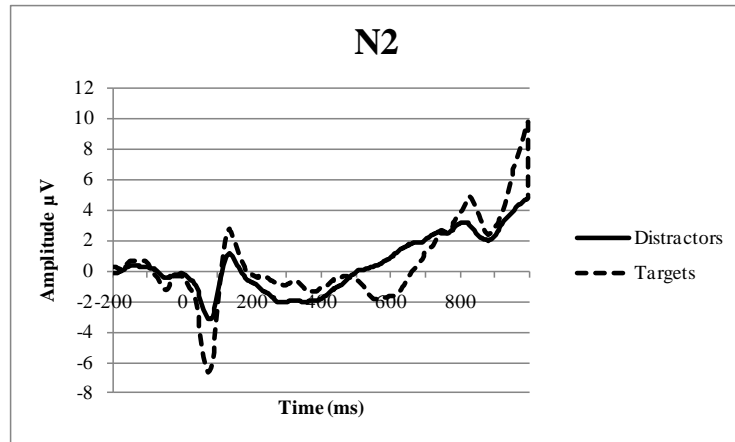
measured and analyzed, while others were not. Windowed and PCA-derived data identified what is consistent with a P3b (or the P3 complex of components). With windowed data, a 2 x 2 x 2 x 2 mixed ANOVA was conducted with dysphoria status, depression history, and gender as between-subjects variables and P3 as a within-subjects variable (amplitude to rare targets [ $M = 7.61 \mu\text{V}$ ,  $SD = 3.69$ ] versus frequent standards [ $M = 2.96\mu\text{V}$ ,  $SD = 2.14$ ]) revealed a significant main effect of the P3 component,  $F(1, 47) = 96.80$ ,  $p < .001$ , collapsing across task types (see Figure 1). Also, two unique parietally-located PCA factors that were positively responsive to rare targets compared to frequent standard stimuli were found. These will be referred to as *P3 Factor 1*,  $F(1, 47) = 61.11$ ,  $p < .001$ , and *P3 Factor 2*,  $F(1, 47) = 15.25$ ,  $p < .001$ . With respect to the P3a, neither windowed nor PCA-derived data in the present sample produced what could be considered a valid, significant component. Regarding the N2, windowed analyses revealed a significant frontal negative deflection in the appropriate temporo-spatial region (see Figure 2 below),  $F(1, 47) = 6.60$ ,  $p = .01$ . Also, a frontally-located PCA factor that was negatively responsive to rare distracters compared to rare targets was found,  $F(1, 47) = 23.91$ ,  $p < .001$ . This PCA factor may be consistent with the inhibitory N2. However, the N2 was not found to vary significantly with respect to any risk variables of interest.

Figure 1. Oddball P3 effect in waveform profile and scalp topography



A clear P3 effect is demonstrated by the subtraction of the mean amplitude to standard stimuli from task-relevant target stimuli, revealing a significantly larger response to the latter. Pre-selected P3 range was 350-650 ms in parietal scalp topography. As seen in graph and net layout on a head, data supported this temporo-spatial location.

Figure 2. N2 effect in waveform profile



A small N2 effect is demonstrated by the subtraction of the mean amplitude to rare target stimuli from task-irrelevant distracter stimuli, revealing a larger negative deflection of the waveform to the latter. Pre-selected N2 range was 210-330 ms in frontal midline scalp topography.

*Did P3b amplitude vary based on gender and other risk variables?* The analyses revealed a significant gender by dysphoria effect,  $F(1, 47) = 3.92, p = .05$ , on average P3 amplitude, collapsing across all task conditions. In particular, dysphoric males showed greater P3s ( $M = 6.15\mu\text{V}, SD = 3.61$ ) than dysphoric females ( $M = 3.45\mu\text{V}, SD = 3.47$ ),  $F(1, 47) = 4.27, p = .04$ . This effect was most pronounced for the nonemotional age identification tasks,  $F(1, 48) = 8.99, p = .004$  and was not statistically significant for the emotion identification blocks. As a result of these preliminary analyses, planned analyses incorporated gender as an additional risk variable. Also, where appropriate, follow up tests were performed separately for females and males. Similarly, due to dysphoria status's apparent influence on nonemotional processing and predicted influence on emotional processing, dysphoria was examined both as a grouping and continuous (i.e., total BDI-II score) variable for comparison purposes.

*Were past depression or current dysphoria associated with different P3 amplitude for general emotional information?* Neither past depression nor current dysphoria were associated with increased or decreased mean P3 amplitude for emotional targets compared to nonemotional targets. However, a main effect of emotionality was found,  $F(1, 47) = 19.06 < .001$ , such that across all groups, target P3s in the emotion identification blocks ( $M = 5.79\mu\text{V}, SD = 3.93$ ) were greater than in the age identification blocks ( $M = 3.68\mu\text{V}, SD = 3.80$ ).

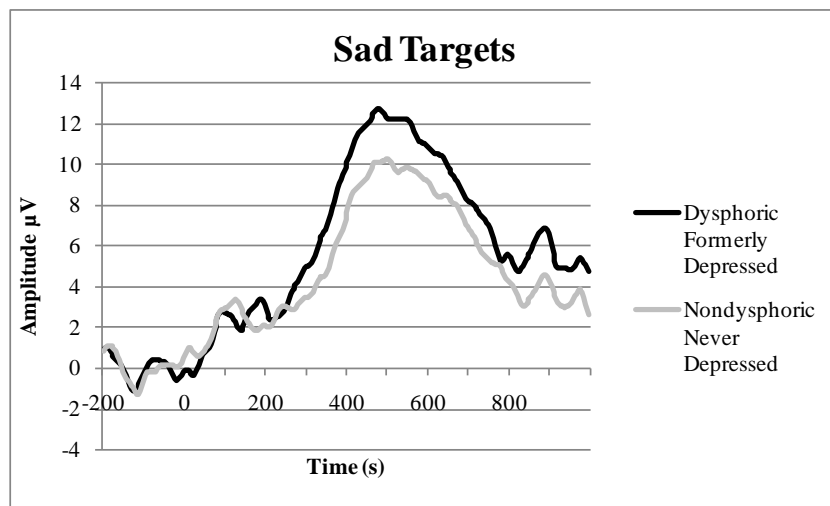
#### *Planned Analyses*

*Were specific predictions supported?* The windowed analysis indicated that the dysphoric formerly depressed group showed a greater P3 amplitude for sad target trials ( $M = 7.80, SD = 3.82$ ) than the nondysphoric never depressed group ( $M = 4.61\mu\text{V}, SD = 3.73$ ),  $t(26) = 2.23, p = .04$  (see Figure 3 below). However, significant between groups differences did not emerge with P3 factors 1 and 2. Also, as expected, the dysphoric formerly depressed group exhibited greater



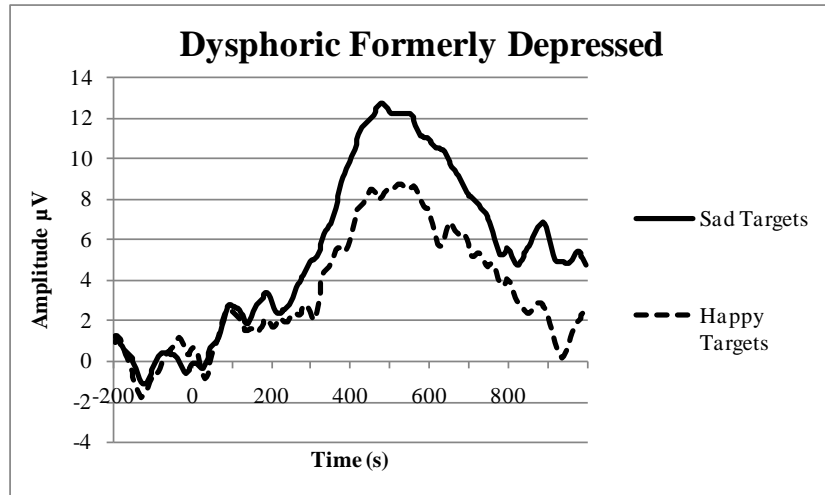
P3 amplitude for sad targets than for happy targets ( $M = 4.76 \mu\text{V}$ ,  $SD = 4.13$ ),  $F = (1, 13) = 7.33$ ,  $p = .02$  (see Figure 3). A significant valence effect did not occur in the other three subgroups. Counter to predictions, the dysphoric formerly depressed and the nondysphoric never depressed groups did not differ significantly in their P3 response to happy targets or targets from the age identification tasks.

Figure 3. Mean group ERP response to sad targets based on risk contrast



The dysphoric formerly depressed group showed a greater mean P3 response to sad targets than the nondysphoric never depressed group. P3 range was 350-650 ms in central-parietal scalp topography. The P3 was calculated by subtracting responses to neutral standard stimuli from responses to sad target stimuli.

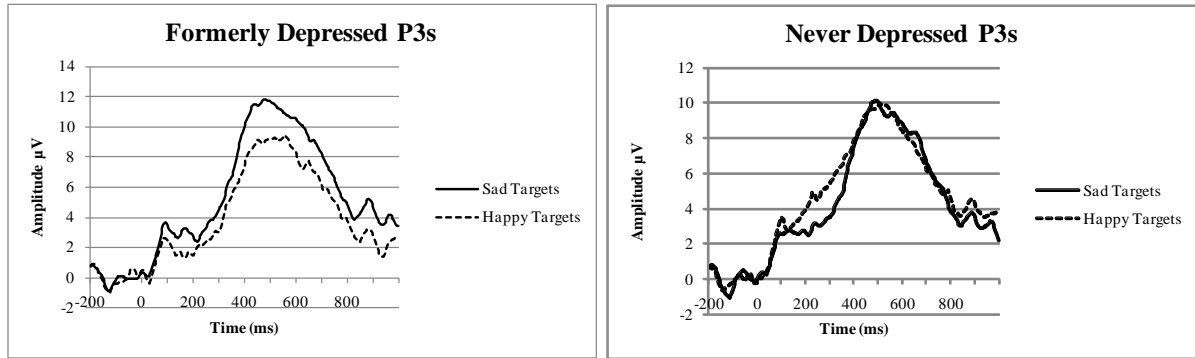
Figure 4. Mean ERP response to targets based on valence



The dysphoric formerly depressed group showed a greater mean P3 response to sad targets than to happy targets. The P3 was calculated by subtracting responses to neutral standard stimuli from responses to sad target stimuli.

*Were there risk variable P3 main effects or interactions based on emotional valence?* A 2 x 2 x 2 mixed ANOVA with dysphoria status, depression history, gender, and valence revealed a trend main effect of depression history,  $F(1, 47) = 3.69, p = .06$ . However, when dysphoria was allowed to covary as a continuous variable (i.e., total BDI-II score), the main effect of depression history became statistically significant,  $F(1, 47) = 3.95, p = .05$ . As represented in Figure 5, past depression was linked to increased P3 amplitude for sad targets and diminished P3 amplitude for happy targets, relative to the never depressed condition. Across the sample, this sad-happy P3 difference variable correlated weakly with endorsed rumination, not quite reaching statistical significance,  $r(n=55) = .25, p = .07$ . Follow up tests examined processing of sad and happy targets independently.

Figure 5. Mean group ERP response to valent targets based on depression history



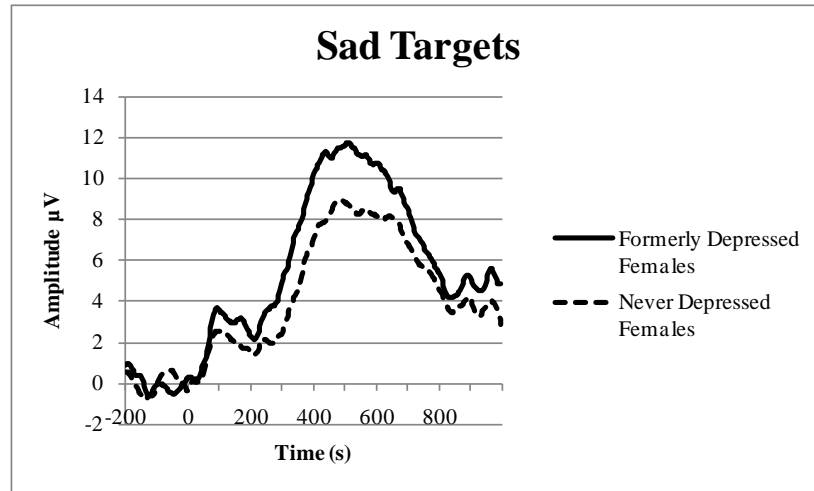
History of depression was associated with greater P3 response to sad targets than happy targets. No such effect associated with lack of depression history. P3 range was 350-650 ms in central-parietal scalp topography. The P3 was calculated by subtracting responses to neutral standard stimuli from responses to sad target stimuli.

*Were there risk variable P3 main effects or interactions for sad target faces? Yes.*

In windowed analyses, a dysphoria status by depression history 2 x 2 ANOVA revealed a main effect of past depression,  $F(1, 51) = 5.30, p = .03$ . Past depression was associated with greater P3s ( $M = 6.96 \mu V, SD = 3.89$ ) than no history of depression ( $M = 4.56 \mu V, SD = 3.79$ ).

Importantly, even among nondysphoric participants, the history of depression contrast effect was observed. With gender as an additional independent variable, a significant three-way gender by dysphoria by depression history interaction emerged,  $F(1, 47) = 5.98, p = .02$ . Examining gender groups separately, females showed a depression history main effect,  $F(1, 32) = 4.29, p = .05$ , while males exhibited a dysphoria by depression interaction,  $F(1, 21) = 4.74, p = .04$ . Formerly depressed women displayed greater P3 amplitude ( $M = 6.54 \mu V, SD = 3.59$ ) than never depressed women ( $M = 3.92 \mu V, SD = 3.13$ ) following sad target faces (see Figure 6). This result was corroborated by a parallel effect for P3 factor 1 from the PCA,  $F(1, 47) = 4.66, p = .04$  in women.

Figure 6. Mean female ERP responses to sad targets based on history of depression

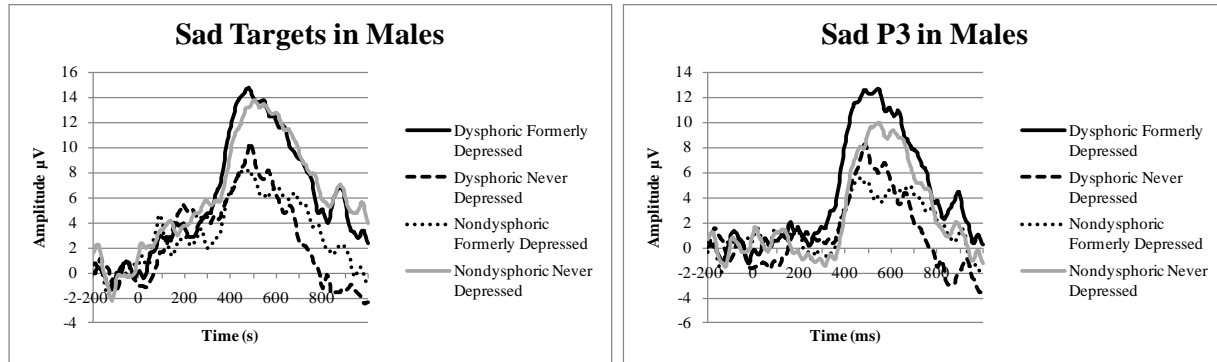


Formerly depressed females showed a greater mean P3 response to sad targets than never depressed females. The P3 was calculated by subtracting responses to neutral standard stimuli from responses to sad target stimuli.

In males, the dual risk (currently dysphoric formerly depressed) group showed greater P3 amplitude ( $M = 9.84 \mu\text{V}$ ,  $SD = 1.40$ ) than both the *nondysphoric* formerly depressed group ( $M = 3.69 \mu\text{V}$ ,  $SD = 1.85$ ),  $F(1, 47) = 7.03$ ,  $p = .01$ , and the currently dysphoric *never* depressed group ( $M = 4.59 \mu\text{V}$ ,  $SD = 1.66$ ),  $F(1, 47) = 5.86$ ,  $p = .02$ , for sad faces (see Figure 7).

Evaluating P3 factor 2 from the PCA revealed a parallel result with formerly depressed currently dysphoric males showing a greater P3 than never depressed currently dysphoric males,  $F(1, 47) = 3.95$ ,  $p = .05$ . Notably, previous depression and current dysphoria status on their own did not differentiate groups from the low-risk status (*nondysphoric never depressed*) males in terms of P3 mean amplitude. Also, consistent with findings for the gender-undifferentiated sample, dual-risk males showed relatively greater sad P3 mean amplitude ( $M = 9.84 \mu\text{V}$ ,  $SD = 3.16$ ) than the *nondysphoric never depressed* group ( $M = 6.21 \mu\text{V}$ ,  $SD = 5.18$ ). However, this difference only approached a statistical trend,  $t(11) = 1.55$ ,  $p = .15$ .

Figure 7. Mean male subgroup ERP responses to sad targets



Two graphs are shown. The graph on the left displays mean EEG amplitude in response to sad target stimuli. In the graph on the right, *P3 values* (i.e., responses to sad targets – responses to neutral standard stimuli) are plotted.

Among males, the dysphoric formerly depressed group showed a greater mean P3 response to sad targets than the three other subgroups. These effects are more evident in the graph on the right. P3 range was 350-650 ms in central-parietal scalp topography.

*Were risk variables associated with diminished P3 amplitude for happy target faces?* In windowed and PCA factor analyses, P3 amplitude for happy targets did not significantly vary based on dysphoria, depression history, or gender.

## Discussion

The present study examined relationships between depression-relevant facial affect processing and risk for depression. A well-established literature has linked negative attentional biases to major depression and recent empirical efforts have increasingly focused on depressive processing of interpersonal facial emotion (Gotlib, Kasch, et al., 2004; Gotlib, Krasnoperova, et al., 2004; Joormann & Gotlib, 2007; Karparova, et al., 2005). Accumulating evidence indicates that cognitive biases similar to those found in depressed groups can be also found in groups at risk for developing depression (Ingram, Steidtmann, & Bistricky, 2008). However, the present study is the first to examine behavioral and neurophysiological measures of selective attention to

depression-relevant facial affect in multiple at-risk groups. Moreover, this study investigated potential state (dysphoric mood), trait (past depression), and interactive effects on correlates of attention to address whether putative cognitive mechanisms might be chronically activated or only reactive to negative mood state. In general, results support that past depression and current dysphoria can affect attentional processing of sad facial affect, as indexed by amplitudes of the P3 complex derived from event-related potentials. By comparison, this study provides limited evidence to support the existence of deficient sad affect inhibition in at-risk groups.

Beyond design-imposed differences (e.g., dysphoria group status conferred greater BDI-II dysphoria score), risk variables were associated with differentiated levels of rumination and various mood states. Consistent with the idea that dysphoria is often characterized by multiple negative affects, current dysphoria was associated with greater depressive, angry, anxious, and fatigued mood states, as well as reduced vigor-activity and increased rumination. Past depression was also associated with marginally greater endorsed rumination.

Regarding the selective attention task, behavioral data indicated overall engaged and valid performance by participants. Dysphoria, depression history, and gender group differences in global accuracy for target stimuli were not predicted and were not found. However, it was predicted that an inhibitory deficit for depressive information might result in depression susceptible (dysphoric formerly depressed) participants mistakenly responding to rare sad distracter faces. Interestingly, dysphoria was linked to increased errors of commission for sad distracters in women, but not in men. Reaction time data appeared to corroborate that these false responses were made more hastily than correct responses. Combined, these findings implicate insufficient evaluation and/or inhibition in response to task-irrelevant sad facial stimuli in the dysphoric female portion of the sample. Possibly, these errors represent the accidental

confounding of personal affective salience (i.e., “that sad face is relevant to me in my dysphoric state”) with rule-dictated task relevance (i.e., “that sad face is relevant to me in the task I have been instructed to do”). However, the gender difference was unexpected and lacks a clear precedent or other support from the literature. Also somewhat unexpectedly, depression history was not related to any significant effects for errors of commission.

Further examining task reaction times, the never depressed currently dysphoric male group exhibited particularly slowed reaction times to targets of all valence and age types. This curious result might correspond with the fact that this group also endorsed the lowest level of vigor-activity and highest level of fatigue-inertia immediately preceding task performance, compared to other groups. Thus, low positive affect and concomitantly sluggish initiation may have contributed to this small group of never depressed currently dysphoric males’ slower response times. Such a finding is akin to cognitive deficits that characterize major depression (van Hoof, Jogems-Kosterman, Sabbe, Zitman, & Hulstijn, 1998), but it is not relevant to depressive cognitive biases. In this regard, depression risk variables were not associated with any valence-specific effects on reaction time. It had been predicted that the dysphoric formerly depressed group would exhibit facilitated processing of depressive faces and slowed processing of positive faces in comparison to the low-risk group, but reaction times revealed no such differences. Thus, neither dysphoria nor previous depression preferentially affected the speed of correctly responding to sad or happy affect.

Theory-driven examination of ERP indicators of attention revealed an interesting pattern of findings, implicating interactive effects among dysphoria, depression history, and gender on attention to facial affect. As predicted, the group presumed to be at greatest risk of future depression (currently dysphoric formerly depressed) showed greater P3 response to sad targets,

compared to the group likely to be at least risk (nondysphoric never depressed) and compared to their own happy P3 responses. When dysphoria was controlled for across the whole sample, past depression was associated with a greater P3 for sad targets than happy targets, an effect absent in the never depressed groups. This past depression effect was due to increased P3 response to sad target faces and not due to attenuated response to happy target faces; specific predicted between-group differences were found for the former and not for the latter.

To contextualize these results, the P3 is presumed to be a psychophysiological indicator of cognitive context-updating (Donchin & Coles, 1988) such that when new environmental stimuli are evaluated to be salient, the P3 signals the incorporation of the change in working memory. Moreover, variability in P3 amplitude is thought to measure attentional allocation (and perhaps affective encoding), and thereby relevance for a particular individual (Gasbarri, et al., 2007; Oliver-Rodriguez, Guan, & Johnston, 1999; Osterhout & Holcomb, 1995). The more personally relevant a stimulus is, the greater the P3 response it evokes, all else being equal. Among normal populations, pleasant and unpleasant stimuli are usually deemed more relevant and evoke greater P3 amplitude than neutral stimuli (Johnston, Miller, & Burleson, 1986). By extension, in this study past depression was associated with greater attentional allocation/perceived relevance for sad than happy target faces. This result was theoretically consistent with Joormann and Gotlib's (2007) behavioral finding in which formerly and currently depressed groups showed greater selective attention toward sad than happy facial expressions. In contrast, Iardi, Atchley, Enloe, Kwasny, and Garratt (2007) found that current but not past depression was associated with elevated attentional P3 responses to negative *words* compared to a never depressed group. Synthesizing the latter two studies and the current study findings, previous depression may be characterized by heightened attentional resource allocation to negative faces but not negative



words.

On the other hand, the influence of dysphoria in the current study was conditional on gender. In females, past depression—irrespective of dysphoria status—was linked to increased P3 amplitude to sad targets (consistent with the full sample effect of past depression). However, among males, dysphoria interacted with previous depression. Specifically, males with *dual risk* (past depression and current dysphoria) exhibited significantly greater P3s to sad targets than males with singular risk (either past depression or dysphoria), and relatively greater sad P3s than the low-risk males. Although supporting evidence of mood state by vulnerability trait-dependent cognitive reactivity has been abundant (Scher, et al., 2005), select findings (Atchley, et al., 2003; Hayward, Goodwin, Cowen, & Harmer, 2005; Joormann & Gotlib, 2007) suggest that a history of depression can be associated with certain depressive cognitive biases in the absence of significant dysphoric mood. Interestingly, the present set of findings seems to provide support for each of these scenarios, but in separate genders. In females, P3 hyper-reactivity to sad faces appeared trait-like, corresponding with solely past depression. In males, similar P3 hyper-reactivity to sad faces appeared trait-*and*-state-dependent, arising only in the group endorsing a history of depression and current dysphoria. By virtue of this study's design, this meant that approximately twice the proportion of females as males showed elevated P3s to sad targets, a theoretically meaningful ratio with respect to depression.

Epidemiological data have consistently reported a two-fold greater prevalence of depression in women than in men (Angst, et al., 2002; Kessler, McGonagle, Swartz, Blazer, & Nelson, 1993; Kuehner, 2003), identifying female gender as a risk factor associated with unique and/or more frequently activated vulnerability mechanisms (see Hyde, Mezulis, & Abramson, 2008 for review and integrative model). Although gender differences in ERP components were

not predicted, and the found elevated sad P3 in nondysphoric formerly depressed women may have been anomalous, at least one speculative account of this finding is worth considering. Beginning in adolescence, females more so than males engage in increased appearance-related *self-surveillance* (McKinley & Hyde, 1996), with an implicit goal of social desirability. Such increased self-critical comparison to perceived ideals tends to be associated with greater body shame and rumination, which in turn predict future depressive symptoms (as reviewed in Hyde, et al., 2008). A woman high in self-surveillance would also typically be more motivated to monitor how others respond to her, and direct affective facial expressions would provide a rich source of evaluative feedback about her social desirability. Seemingly disapproving expressions could be particularly salient, as they might trigger an intuitively perceived need to modify behavior or appearance. If the formerly depressed women in the current study were more likely to self-surveil than their never depressed counterparts, they may have more vigilantly attended to sad faces as an automatic, pre-conscious form of negative social feedback monitoring. Irrespective of the accuracy of this account, it seems significant that this mode of cognitive processing was active in formerly depressed females regardless of their current dysphoria status, but was restricted to dysphoric formerly depressed males. On a theoretical level, for any group in which it is found, an elevated attentional P3 to sad facial expressions may constitute a mechanism, rather than a passive marker, of risk for depression.

A disproportional attentional bias toward sad faces, as was found in the present study, could increase susceptibility to depression in several different ways that are consistent with cognitive and interpersonal models of depression (Beck, 1976; Ingram, 1984; Joiner & Coyne, 1999; Lewinsohn, 1974; Teasdale & Barnard, 1993). To begin with, a sad attentional bias could lead one to appraise social interactions more negatively, propagating depressive emotion and

cognition. If one's interaction partner smiles once and frowns once, a more intensively attended frown might receive prolonged processing and deeper affective encoding. The biased perceiver might leave and recall the interaction with feelings of distress, failure, or embarrassment.

Consistent with this reasoning, a stable negative attributional style for interpersonal events, triggered by ambiguous interactions, can elicit severe depressive symptoms (Joiner & Rudd, 1995). Moreover, depression, which is often characterized by negatively biased information processing and attributional style, has been associated with abnormally amplified sad emotional responses to others' negative facial affect (Persad & Polivy, 1993).

Selectively attending to negative emotional faces might also trigger depressive mood-maintaining thought patterns in depression-susceptible individuals. For example, Frewen and Dozois (2005) found that while viewing negative affective faces, dysphoric women were more likely than nondysphoric women to endorse having critical self-thoughts, feeling negatively evaluated by others, and feeling responsible for others' negative emotion. These types of automatic thoughts could spur other emotionally charged schema-related cognitions, ultimately leading to depressive rumination. A person might think, "His expression tells me he disapproved of my comment. Why did I say such a stupid thing? I screw everything up!" This kind of rumination may help explain how a combination of negative attention bias and life stressors can prospectively predict depressive symptoms (Beevers & Carver, 2003). Although the present study employed no measures of online thinking, dysphoria and past depression were related to greater endorsed rumination. In turn, rumination was weakly correlated with greater sad-to-happy P3 differential.

In addition, hyper-vigilant attention to sad facial feedback might help verify one's negative self-schema, perhaps providing relief from cognitive dissonance at the expense of motivation and

self-worth. Research indicates that some depression susceptible individuals seek out negative social evaluation—or “negative self-verification”—which tends to maintain or exacerbate depressive symptoms (Joiner, 1995; Swann, Wenzlaff, & Tafarodi, 1992). Similarly, hyper-attending to sad facial affect could verify one’s pessimistic view of the world. Derivative automatic thoughts such as, “a lot of other people seem dejected” or “other people appear judgmental and rejecting” might bolster a schema of the world as unwelcoming, unfulfilling, and unmanageable. As a consequence, disproportionately greater depressive cognition and emotion instigated by an attentional bias for sad facial affect might hinder normal social approach behaviors (Zauszniewski & Rong, 1999). Social isolation is a common depressive symptom that reduces social reinforcement and maintains depressive moods. In these ways, disproportionate attention directed toward sad facial emotion might elicit depressotypic cognitive, emotional, and interpersonal behavioral responses, possibly giving rise to the kind of cognitive structures and ruminative processing that promote and prolong depressed moods (Nolen-Hoeksema, et al., 1993). In fact, the combination of negative cognitive style and stress-reactive rumination appears potent enough that it can predict prospective onsets of depression (Robinson & Alloy, 2003). Although the present study could not test complex vulnerability pathways, it did identify in at-risk groups psychophysiological evidence of a biased attentional mechanism through which vulnerability pathways might extend. Moreover, this attentional bias was more reliably associated with past depression than with current dysphoria, supporting the notion that different operationalizations of depression risk may evince distinct mechanisms of vulnerability (Hyde, et al., 2008).

This investigation also attempted to address other conceptual questions of interest. Most importantly, based on past findings with currently and formerly depressed groups (Goeleven, et

al., 2006; Joormann, 2004), it was hypothesized that risk variables might be related to deficient inhibition of depressive facial affect. In theory, lingering negative content would become diffusely associated, triggering frequent rumination and prolonged periods of depressed mood. However, this study does not unequivocally support or refute abnormal affective inhibition processes. Although dysphoric women showed increased errors of commission for sad distracter faces, no between-group differences were found for the N2 potential, and the P3a was not reliably extracted. With respect to the P3a, full sample accuracy data indicate that this study's novelty oddball task may have required comparable inhibition for standard faces as distracter faces (when looking for a sad target face, the impulse could even be *greater* to respond to a neutral face than a happy face). This would have neutralized the probability that usually prompts a P3a response to the rare distracter. Fortunately, other recent studies (Eugene, Joormann, Cooney, Atlas, & Gotlib, 2010; Joormann, Nee, Berman, Jonides, & Gotlib, 2010) have provided further insight into possible inhibitory deficits in depression-susceptible populations through the use of different cognitive neuroscience methods. For example, Krompinger and Simons (2009) employed a go/no-go paradigm, and found that undergraduates who were elevated in depressive symptoms showed a decreased N2 for negative compared to positive pictures, unlike euthymic students. Because the go/no-go task predisposes a participant response on every trial, and the oddball task predisposes a non-response, distracter trials are significantly more challenging to response-inhibition resources in a go/no-go task than in the novelty oddball task (Bekker, Kenemans, & Verbaten, 2005; Donkers & van Boxtel, 2004). The present three-stimulus oddball paradigm provided the opportunity to assess selective attention and inhibition for depression-relevant affects mixed among predominantly neutral faces, arguably a valid analogue to naturalistic social settings. However, while the oddball task elicited selective attention

differences, it may have been less sensitive to inhibitory deficits than a two-stimulus go/no-go task. Future studies may more usefully employ the no-go N2 and the negative priming P2 potentials (Yao, et al., 2010) to examine affective inhibition and depression susceptibility.

Additionally, although previous depression research (Cavanagh & Geisler, 2006; Deldin, Keller, Gergen, & Miller, 2001) suggested that happy faces might evoke attenuated P3s in depression susceptible groups and increased P3s in low-risk groups, data from the present study did not support either scenario. This result was not particularly surprising, however. Although several behavioral studies have reported diminished attention to happy facial affect in depression susceptible populations (Joormann & Gotlib, 2007; Suslow, et al., 2004; Suslow, et al., 2001), others have reported normal processing of positive affect (Gotlib, Kasch, et al., 2004; Gotlib, Krasnoperova, et al., 2004; Karparova, et al., 2005). The present behavioral and psychophysiological findings suggest that current dysphoria and a history of depression may not be associated with selective attention deficits for happy facial affect.

This investigation also sought to examine whether psychophysiological indicators of a depressotypic attentional bias might be detectable in presumed at-risk (dysphoric) individuals who had never experienced a past episode of depression. If so, this cognitive factor's precedence to a first onset would be established, more strongly implicating it in etiological processes. However, independent of depression history, dysphoria was not associated with elevated P3s following sad targets. Only groups with a past history of depression, a significant risk factor for future depression (American Psychiatric Association, 2000), showed this effect. One possibility is that those who are prone to a full-fledged major depressive syndrome are distinguished by underlying trait-like (genetic, temperamental, or epigenetic) factors that, when combined with stressful events, can result in depression (Hyde, et al., 2008). By reciprocal reasoning, if sad

attentional bias is one such factor, it would be most frequently detected in the formerly depressed groups in the present study. Additional supportive cross-sectional evidence has indicated that abnormally biased attention toward negative facial affect can be elicited in never-depressed children with inherited trait-susceptibility to depression (Joormann, Talbot, & Gotlib, 2007). A promising future direction would be to compare never-depressed individuals who disproportionately direct attention to negative facial affect with non-biased individuals on longitudinally-tracked measures of mood, cognition, life stressors, and interpersonal functioning. If biased attention to sad facial affect is a depressogenic factor, it might predict greater depressive symptoms in conjunction with these other psychosocial factors.

Inconsistent with study predictions, the dysphoric formerly depressed group did not show attenuated P3s on the nonaffective age-evaluation oddball task. A large collection of studies has indicated that major depression is associated with an attenuation of the P3 on oddball tasks involving nonaffective stimuli (Ancy, et al., 1996; Kemp, et al., 2009), which is theoretically linked to a depressive attentional deficit. The present result may indicate that this attentional deficit does not occur in dysphoric individuals below the threshold of major depression. Consistent with this interpretation, remission of depressive symptoms has been linked to P3 normalization (Neuhaus, et al., 2007). Alternatively, it could be that faces, irrespective of their affect, are sufficiently relevant and engaging to dysphoric or depressed human observers so as to elicit a normal target P3. This possibility is worth investigating in future research.

On a related note, this study provided evidence that affective faces receive greater attentional allocation than nonaffective faces. That is, regardless of diagnostic status, participants exhibited greater P3s for affect-evaluation targets than age-evaluation targets. This finding appears consistent with the notion that facial affect has always been an indispensable source of

information for humans solving problems related to reproduction and survival (Cosmides & Tooby, 2000; Wilson, 1999). To translate the present finding to a naturalistic setting, detecting a frown or a smile better informs an immediate behavioral response than identifying the age range of a neutral face. In short, facial affect is salient and receives preferential attention.

The present study had several limitations worth considering. First, the present sample became undersized once unexpected gender interactions necessitated analyses with three rather than two dichotomous participant variables. Given that four participant cells turned to eight, power to detect true differences may have been hindered. Conversely, small cell sizes (e.g., five in dysphoric formerly depressed male group) may have increased the likelihood of extreme but not statistically outlying values driving a group effect. Certainly, replication of the gender-differentiated P3 results will be needed before any strong conclusions can be drawn from them. Importantly, however, the elevated sad P3 associated with past depression was derived from the whole sample. As such, this result may be more reliable. Second, the present sample was relatively homogeneous with respect to age, race, and ethnicity. Therefore, it cannot be known whether the present results generalize beyond this largely Caucasian, college-aged sample. To assess this, similar studies enrolling various age, racial, and ethnic groups will be needed. Third, the study's cross-sectional design tacitly implies that participants with dysphoria or past depression are at greater risk of depression based on epidemiological risk ratio data. However, this design cannot confirm whether these individuals will eventually develop depressive episodes at an abnormally elevated rate. Similarly, as previously mentioned, the present study cannot confirm whether the found attentional bias for sad affect contributes to any of the speculative psychosocial repercussions discussed earlier. Fourth, BDI-II scores extracted from Life Stress Inventory (LSI) responses may be inflated. The present study prioritized limiting participants'



social desirability response set in order to reduce false negative assessments of dysphoric individuals who might deny symptoms on a more obvious clinical measure. Nevertheless, past LSI findings would suggest that the dysphoric groups in the present study would fall within the range of dysphoria traditionally measured by the stand-alone BDI-II (Hunt, et al., 2003). Finally, given that the temporo-spatial principal components analysis failed to extract a clear P3a potential, the study was unable to examine possible between-groups differences related to depression susceptibility.

Despite its limitations, this study provides some intriguing evidence and raises important questions for future research. Most significantly, this is the first study to report an association between past depression and preferentially elevated P3 amplitude in response to sad facial affect. The presence of current dysphoria may increase the likelihood of this response pattern in males. These psychophysiological findings suggest that depression vulnerable individuals may be more likely to allocate disproportionately increased attentional resources toward sad facial affect in the social environment, even without exhibiting behavioral indicators of bias. Theoretically, increased attention toward sad expressions could cultivate overly negativistic conceptions of the self and proximal social world, induce greater interpersonal stress, and promote rumination. In turn, these factors might instigate frequent, prolonged depressed moods and, perhaps, depressive episodes.

Future research will need to answer key questions to pave empirical gaps along theorized pathways. For instance, is attention bias to sad *facial affect* part of a distinctive vulnerability pathway or part of a global (i.e., modality agnostic) depressive attention bias that generalizes to affective words and pictures as well? Few studies have compared, in the same depressed sample, the processing of facial affect and other affective stimulus modalities. However, some evidence

indicates that biases to specific facial affects may be dissociable from biases to words of the same affect (Deldin et al., 2000; Karparova, et al., 2005). Conceivably, between two depression-susceptible individuals, one whose sense of worth derives predominantly from social validation might be more vigilant to disapproving facial feedback than a person whose self-perceived value comes from individual accomplishment (Beck, 1987). Conversely, the achievement-oriented individual might attend more to loaded words, such as “loser” or “failure.”

Further considering modality congruence, how is an attentional bias for sad facial affect related to depressive social and cognitive phenomena, such as reassurance-seeking, negative self-verification, rejection-elicitation, self-surveillance and negative attributional style? In particular, does a facial affect bias lead to increased depressive symptoms and increased likelihood of a depressive episode via these cognitive-interpersonal avenues? Prospective longitudinal studies incorporating relevant factors could help address these important questions. Lastly, it remains unknown whether depression susceptibility is characterized by deficient inhibition of negative information and related neural correlates. Relevant studies should be informed by the growing literature regarding affect inhibition in depression.

In sum, the present study provides psychophysiological evidence indicating that depression vulnerability is linked to increased selective attention to sad facial affect. Future research is needed to replicate, clarify, and extend the present findings toward an understanding of the role of facial affect processing in depression vulnerability pathways. In this context, the present findings may be a modest but important step.

## References

- American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition Text Revision*. Washington, DC: American Psychiatric Association.
- Ancy, J., Gangadhar, B. N., & Janakiramaiah, N. (1996). "Normal" P300 amplitude predicts rapid response to ECT in melancholia. *Journal of Affective Disorders, 41*(3), 211-215.
- Angst, J., Gamma, A., Gastpar, M., Lepine, J. P., Mendlewicz, J., & Tylee, A. (2002). Gender differences in depression. Epidemiological findings from the European DEPRES I and II studies. *European Archives of Psychiatry and Clinical Neuroscience, 252*(5), 201-209.
- Atchley, R. A., Ilardi, S. S., & Enloe, A. (2003). Hemispheric asymmetry in the processing of emotional content in word meanings: the effect of current and past depression. *Brain and Language, 84*, 105-119.
- Atchley, R. A., Stringer, R., Mathias, E., Ilardi, S. S., & Minatrea, A. D. (2007). The right hemisphere's contribution to emotional word processing in currently depressed, remitted depressed, and never depressed individuals. *Journal of Neurolinguistics, 20*(2), 145-160.
- Beck, A. T. (1967). *Depression: Causes and treatment*. Philadelphia, PA: University of Philadelphia Press.
- Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York: International Universities Press.
- Beck, A. T. (1987). Cognitive models of depression. *Journal of Cognitive Psychotherapy, 1*, 5-37.
- Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Beck Depression Inventory manual (2nd ed.)*. San Antonio, TX: Psychological Corporation.

- Beevers, C. G., & Carver, C. S. (2003). Attentional bias and mood persistence as prospective predictors of dysphoria. *Cognitive Therapy and Research*, 27, 619-637.
- Beevers, C. G., Rohde, P., Stice, E., & Nolen-Hoeksema, S. (2007). Recovery from major depressive disorder among female adolescents: a prospective test of the scar hypothesis. *J Consult Clin Psychol*, 75(6), 888-900.
- Bekker, E. M., Kenemans, J. L., & Verbaten, M. N. (2004). Electrophysiological correlates of attention, inhibition, sensitivity and bias in a continuous performance task. *Clinical Neurophysiology*, 115(9), 2001-2013.
- Bekker, E. M., Kenemans, J. L., & Verbaten, M. N. (2005). Source analysis of the N2 in a cued Go/NoGo task. *Cognitive Brain Research* 22(2), 221-231.
- Bertoli, S., & Probst, R. (2005). Lack of standard N2 in elderly participants indicates inhibitory processing deficit. *Neuroreport*, 16(17), 1933-1937.
- Bistricky, S. L., Ingram, R. E., & Atchley, R. A. (2010). Facial affect processing and depression susceptibility: Cognitive biases and cognitive neuroscience. *Manuscript submitted for publication*, 45.
- Bokura, H., Yamaguchi, S., & Kobayashi, S. (2001). Electrophysiological correlates for response inhibition in a Go/NoGo task. *Clinical Neurophysiology*, 112(12), 2224-2232.
- Bokura, H., Yamaguchi, S., & Kobayashi, S. (2005). Event-related potentials for response inhibition in Parkinson's disease. *Neuropsychologia*, 43(6), 967-975.
- Bradley, B. P., Mogg, K., Falla, S. J., & Hamilton, L. R. (1998). Attentional bias for threatening facial expressions in anxiety: Manipulation of stimulus duration. *Cognition and Emotion*, 12(6), 737-753.
- Bradley, B. P., Mogg, K., & Lee, S. C. (1997). Attentional biases for negative information in

- induced and naturally occurring dysphoria. *Behavioral Research and Therapy*, 35(10), 911-927.
- Bradley, B. P., Mogg, K., & Millar, N. H. (2000). Covert and overt orienting of attention to emotional faces in anxiety. *Cognition and Emotion*, 14(6), 7899-7808.
- Bruin, K. J., Wijers, A. A., & van Staveren, A. S. (2001). Response priming in a go/nogo task: do we have to explain the go/nogo N2 effect in terms of response activation instead of inhibition? *Clinical Neurophysiology*, 112(9), 1660-1671.
- Cavanagh, J., & Geisler, M. W. (2006). Mood effects on the ERP processing of emotional intensity in faces: a P3 investigation with depressed students. *International Journal of Psychophysiology*, 60, 27-33.
- Clark, D. A., Beck, A. T., & Stewart, B. (1990). Cognitive specificity and positive-negative affectivity: Complementary or contradictory views on anxiety and depression? *Journal of Abnormal Psychology*, 99, 148-155.
- Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, 100(3), 316-336.
- Cosmides, L., & Tooby, J. (2000). Evolutionary Psychology and the Emotions. In M. Lewis & J. M. Haviland-Jones (Eds.), *Handbook of Emotions*. New York: Guilford Press.
- Cosmides, L., Tooby, J., & Barkow, J. (1992). Introduction: Evolutionary psychology and conceptual integration. . In J. Barkow, L. Cosmides & J. Tooby (Eds.), *The adapted mind: Evolutionary psychology and the generation of culture* (pp. 3-15). New York: Oxford University Press.
- Coyne, J. C. (1976). Toward an interactional description of depression. *Psychiatry*, 39(1), 28-40.
- Cuijpers, P., & Smit, F. (2004). Subthreshold depression as a risk indicator for major depressive

- disorder: a systematic review of prospective studies. *Acta Psychiatrica Scandinavica*, *109*(5), 325-331.
- De Houwer, J., & Hermans, D. (1994). Differences in the affective processing of words and pictures. *Cognition and Emotion*, *8*, 1-20.
- Debener, S., Kranczioch, C., Herrmann, C. S., & Engel, A. K. (2002). Auditory novelty oddball allows reliable distinction of top-down and bottom-up processes of attention. *International Journal of Psychophysiology*, *46*, 77-84.
- Deldin, P. J., Keller, J., Gergen, J. A., & Miller, G. A. (2000). Right-posterior face processing anomaly in depression. *Journal of Abnormal Psychology*, *109*(1), 116-121.
- Deldin, P. J., Keller, J., Gergen, J. A., & Miller, G. A. (2001). Cognitive bias and emotion in neuropsychological models of depression. *Cognition and Emotion*, *15*(6), 787-802
- Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, *134*(1), 9-21.
- Dien, J., Spencer, K. M., & Donchin, E. (2003). Localization of the event-related potential novelty response as defined by principal components analysis. *Brain Research: Cognitive Brain Research*, *17*(3), 637-650.
- Dien, J., Tucker, D. M., Potts, G., & Hartry-Speiser, A. (1997). Localization of auditory evoked potentials related to selective intermodal attention. *Journal of Cognitive Neuroscience*, *9*(6), 799-823.
- Diner, B. C., Holcomb, P. J., & Dykman, R. A. (1985). P300 in major depressive disorder. *Psychiatry Research*, *15*(3), 175-184.
- Donchin, E., & Coles, M. G. (1988). Is the P300 component a manifestation of *context updating*?

*Behavioral and Brain Sciences, 11(3).*

- Donkers, F. C., & van Boxtel, G. J. (2004). The N2 in go/no-go tasks reflects conflict monitoring not response inhibition. *Brain and Cognition, 56(2)*, 165-176.
- Drummond, S. P., Gillin, J. C., & Brown, G. G. (2001). Increased cerebral response during a divided attention task following sleep deprivation. *Journal of Sleep Research, 10(2)*, 85-92.
- Eaton, W. W., Badawi, M., & Melton, B. (1995). Prodromes and precursors: epidemiologic data for primary prevention of disorders with slow onset. *American Journal of Psychiatry, 152(7)*, 967-972.
- Erwin, R. J., Gur, R. C., Gur, R. E., Skolnick, B., Mawhinney-Hee, M., & Smailis, J. (1992). Facial emotion discrimination: I. Task construction and behavioral findings in normal subjects. *Psychiatry Research, 42(3)*, 231-240.
- Eugene, F., Joormann, J., Cooney, R. E., Atlas, L. Y., & Gotlib, I. H. (2010). Neural correlates of inhibitory deficits in depression. *Psychiatry Research, 181(1)*, 30-35.
- Fallgatter, A. J., Ehlis, A. C., Seifert, J., Strik, W. K., Scheuerpflug, P., Zillessen, K. E., et al. (2004). Altered response control and anterior cingulate function in attention-deficit/hyperactivity disorder boys. *Clinical Neurophysiology, 115(4)*, 973-981.
- Fox, A. M., Michie, P. T., Wynne, C. D., & Maybery, M. T. (2000). ERP correlates of response inhibition to elemental and configural stimuli in a negative patterning task. *Clinical Neurophysiology, 111(6)*, 1045-1053.
- Gasbarri, A., Arnone, B., Pompili, A., Pacitti, F., Pacitti, C., & Cahill, L. (2007). Sex-related hemispheric lateralization of electrical potentials evoked by arousing negative stimuli. *Brain Research, 1138*, 178-186.

- Gilboa-Schechtman, E., Erhard-Weiss, D., & Jeczemien, P. (2002). Interpersonal deficits meet cognitive biases: memory for facial expressions in depressed and anxious men and women. *Psychiatry Research, 113*(3), 279-293.
- Glaser, W. R., & Glaser, M. O. (1989). Context effects in Stroop-like word and picture processing. *Journal of Experimental Psychology: General, 118*, 13-42.
- Goeleven, E., De Raedt, R., Baert, S., & Koster, E. H. W. (2006). Deficient inhibition of emotional information in depression. *Journal of Affective Disorders, 93*, 149-157.
- Gollan, J. K., Pane, H. T., McCloskey, M. S., & Coccaro, E. F. (2008). Identifying differences in biased affective information processing in major depression. *Psychiatry Research, 159*(1-2), 18-24.
- Gotlib, I. H., Kasch, K. L., Traill, S., Joormann, J., Arnow, B. A., & Johnson, S. L. (2004). Coherence and specificity of information-processing biases in depression and social phobia. *Journal of Abnormal Psychology, 113*(3), 386-398.
- Gotlib, I. H., Krasnoperova, E., Yue, D. N., & Joormann, J. (2004). Attentional biases for negative interpersonal stimuli in clinical depression. *Journal of Abnormal Psychology, 113*, 121-135.
- Gur, R. C., Erwin, R. J., Gur, R. E., Zvil, A. S., Heimberg, C., & Kraemer, H. C. (1992). Facial emotion discrimination: II. Behavioral findings in depression. *Psychiatry Research, 42*(3), 241-251.
- Gur, R. C., Sara, R., Hagendoorn, M., Marom, O., Hughett, P., Macy, L., et al. (2002). A method for obtaining 3-dimensional facial expressions and its standardization for use in neurocognitive studies. *Journal of Neuroscience Methods, 115*(2), 137-143.
- Hartlage, S., Alloy, L. B., Vazquez, C., & Dykman, B. (1993). Automatic and effortful



- processing in depression. *Psychological Bulletin*, 113(2), 247-278.
- Hayward, G., Goodwin, G., Cowen, P. J., & Harmer, C. J. (2005). Low-dose tryptophan depletion in recovered depressed patients induces changes in cognitive processing without depressive symptoms. *Biological Psychiatry*, 57, 517-524.
- Houghton, G., & Tipper, S. P. (1994). A model of inhibitory mechanisms in selective attention. In D. Dagenbach & T. Carr (Eds.), *Inhibitory Mechanisms in Attention, Memory and Language*. San Diego, CA: Academic Press.
- Hsieh, Y. H., & Ko, H. C. (2004). Cognitive biases for emotional faces in high- and low-trait depressive participants. *Kaohsiung Journal of Medical Science*, 20(10), 478-483.
- Hunt, M., Auriemma, J., & Cashaw, A. C. (2003). Self-report bias and underreporting of depression on the BDI-II. *Journal of Personality Assessment*, 80(1), 26-30.
- Hyde, J. S., Mezulis, A. H., & Abramson, L. Y. (2008). The ABCs of depression: integrating affective, biological, and cognitive models to explain the emergence of the gender difference in depression. *Psychological Review*, 115(2), 291-313.
- Ilardi, S. S., Atchley, R. A., Enloe, A., Kwasny, K., & Garratt, G. (2007). Disentangling attentional biases and attentional deficits in depression: An event-related potential P300 analysis. *Cognitive Therapy and Research*, 31(175-187).
- Ingram, R. E. (1984). Toward an information-processing analysis of depression. *Cognitive Therapy and Research*, 8(5), 443-478.
- Ingram, R. E., & Hamilton, N. A. (1999). Evaluating precision in the social psychological assessment of depression: Methodological considerations, issues, and recommendations. *Journal of Social and Clinical Psychology*, 18(2), 160-180.
- Ingram, R. E., Miranda, J., & Segal, Z. V. (1998). *Cognitive vulnerability to depression*. New

York: Guilford.

- Ingram, R. E., Steidtmann, D., & Bistricky, S. L. (2008). Information Processing: Attention and Memory. In K. S. Dobson & D. Dozois (Eds.), *Risk factors in depression*. Oxford, U.K.: Elsevier/Academic Press.
- Jermann, F., van der Linden, M., & D'Argembeau, A. (2008). Identity recognition and happy and sad facial expression recall: influence of depressive symptoms. *Memory*, *16*(4), 364-373.
- Jodo, E., & Kayama, Y. (1992). Relation of a negative component to response inhibition in a Go/No-Go task. Electroencephalograph. *Clinical Neurophysiology*, *82*, 477-482.
- Johnston, V. S., Miller, D. R., & Burlison, M. H. (1986). Multiple P3s to emotional stimuli and their theoretical significance. *Psychophysiology*, *23*, 684-693.
- Johnstone, S. J., Dimoska, A., Smith, J. L., Barry, R. J., Pleffer, C. B., Chiswick, D., et al. (2007). The development of stop-signal and Go/Nogo response inhibition in children aged 7-12 years: performance and event-related potential indices. *International Journal of Psychophysiology*, *63*(1), 25-38.
- Joiner, T. E., & Coyne, J. C. (1999). *The interactional nature of depression: Advances in interpersonal approaches*. Washington, D.C.: American Psychological Association.
- Joiner, T. E., Jr. (1995). The price of soliciting and receiving negative feedback: self-verification theory as a vulnerability to depression theory. *Journal of Abnormal Psychology*, *104*(2), 364-372.
- Joiner, T. E., Jr., & Rudd, M. D. (1995). Negative attributional style for interpersonal events and the occurrence of severe interpersonal disruptions as predictors of self-reported suicidal ideation. *Suicide and Life Threatening Behavior*, *25*(2), 297-304.
- Jonkman, L. M. (2006). The development of preparation, conflict monitoring and inhibition from

- early childhood to young adulthood: a Go/Nogo ERP study. *Brain Research*, 1097, 181-193.
- Joormann, J. (2004). Attention bias in dysphoria: The role of inhibitory processes. *Cognition and Emotion*, 18(1), 125-147.
- Joormann, J., & Gotlib, I. H. (2007). Selective attention to emotional faces following recovery from depression. *Journal of Abnormal Psychology*, 116(1), 80-85.
- Joormann, J., Nee, D. E., Berman, M. G., Jonides, J., & Gotlib, I. H. (2010). Interference resolution in major depression. *Cognitive, Affective, & Behavioral Neuroscience*, 10(1), 21-33.
- Joormann, J., Talbot, L., & Gotlib, I. H. (2007). Biased processing of emotional information in girls at risk for depression. *Journal of Abnormal Psychology*, 116(1), 135-143.
- Kaiser, S., Unger, J., Kiefer, M., Markela, J., Mundt, C., & Weisbrod, M. (2003). Executive control deficit in depression: event-related potentials in a Go/Nogo task. *Psychiatry Research*, 122(3), 169-184.
- Karparova, S. P., Kersting, A., & Suslow, T. (2005). Disengagement of attention from facial emotion in unipolar depression. *Psychiatry and Clinical Neurosciences*, 59(6), 723-729.
- Kemp, A. H., Hopkinson, P. J., Hermens, D. F., Rowe, D. L., Sumich, A. L., Clark, C. R., et al. (2009). Fronto-temporal alterations within the first 200 ms during an attentional task distinguish major depression, non-clinical participants with depressed mood and healthy controls: a potential biomarker? *Human Brain Mapping*, 30(2), 602-614.
- Kessler, R. C., McGonagle, K. A., Swartz, M., Blazer, D. G., & Nelson, C. B. (1993). Sex and depression in the National Comorbidity Survey. I: Lifetime prevalence, chronicity and recurrence. *Journal of Affective Disorders*, 29(2-3), 85-96.

- Koster, E. H., De Raedt, R., Goeleven, E., Franck, E., & Crombez, G. (2005). Mood-congruent attentional bias in dysphoria: maintained attention to and impaired disengagement from negative information. *Emotion*, 5(4), 446-455.
- Kuehner, C. (2003). Gender differences in unipolar depression: an update of epidemiological findings and possible explanations. *Acta Psychiatrica Scandinavica*, 108(3), 163-174.
- Lavric, A., Pizzagalli, D. A., & Forstmeier, S. (2004). When 'go' and 'nogo' are equally frequent: ERP components and cortical tomography. *European Journal of Neuroscience*, 20(9), 2483-2488.
- Leppanen, J. M., Milders, M., Bell, J. S., Terriere, E., & Hietanen, J. K. (2004). Depression biases the recognition of emotionally neutral faces. *Psychiatry Research*, 128(2), 123-133.
- Lewinsohn, P. M. (1974). A behavioral approach to depression. In R. J. Friedman & M. M. Katz (Eds.), *The psychology of depression: Contemporary theory and research* (pp. 157-185). Washington, DC: Winston-Wiley.
- Lewinsohn, P. M., Steinmetz, J. L., Larson, D. W., & Franklin, J. (1981). Depression-related cognitions: antecedent or consequence? *Journal of Abnormal Psychology*, 90(3), 213-219.
- Linville, P. (1996). Attention Inhibition: Does it Underlie Ruminative Thought? In R. S. Wyer & T. K. Srull (Eds.), *Ruminative Thoughts: Advances in Social Cognition* (Vol. IX, pp. 121-133). Mahwah, NJ: Lawrence Erlbaum Associates
- Lundqvist, D., Flyt, A., & Ohman, A. (1998). Karolinska Directed Emotional Faces - KDEF [CD-ROM]. Stockholm: Department of Clinical Neuroscience, Psychology Section, Karolinska Institutet.

- McCabe, S. B., Gotlib, I. H., & Martin, R. A. (2000). Cognitive vulnerability for depression: Deployment of attention as a function of history of depression and current mood state. *Cognitive Therapy and Research, 24*(4), 427-444.
- McKinley, N. M., & Hyde, J. S. (1996). The Objectified Body Consciousness Scale: Development and validation. *Psychology of Women Quarterly, 20*(181-215).
- McNair, D. M., Loo, M., & Droppleman, L. F. (1992). *Manual for the Profile of Mood States*. . San Diego, CA: Educational and Industrial Testing Service.
- Minear, M., & Park, D. C. (2004). A lifespan database of adult facial stimuli. *Behavior Research Methods, Instruments, & Computers, 36*(4), 630-633.
- Murthy, P. J., Gangadhar, B. N., Janakiramaiah, N., & Subbakrishna, D. K. (1997). Normalization of P300 amplitude following treatment in dysthymia. *Biological Psychiatry, 42*(8), 740-743.
- Nandrino, J., Dodin, V., Martin, P., & Henniaux, M. (2004). Emotional information processing in first and recurrent major depressive episodes. *Journal of Psychiatric Research, 38*, 475-484.
- Neill, W. T., Valdes, L. A., & Terry, K. M. (1995). Selective attention and the inhibitory control of cognition. In F. N. Dempster & C. J. Brainerd (Eds.), *Interference and inhibition in cognition* (pp. 207-261). New York: Academic Press.
- Neuhaus, A. H., Luborzewski, A., Rentzsch, J., Brakemeier, E. L., Opgen-Rhein, C., Gallinat, J., et al. (2007). P300 is enhanced in responders to vagus nerve stimulation for treatment of major depressive disorder. *Journal of Affective Disorders, 100*(1-3), 123-128.
- Nieuwenhuis, S., Yeung, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: effects

- of response conflict and trial type frequency. *Cognitive Affective and Behavioral Neuroscience*, 3(1), 17-26.
- Nikendei, C., Dengler, W., Wiedemann, G., & Pauli, P. (2005). Selective processing of pain-related word stimuli in subclinical depression as indicated by event-related brain potentials. *Biological Psychology*, 70, 52-60.
- Nolen-Hoeksema, S., & Morrow, J. (1991). A prospective study of depression and posttraumatic stress symptoms after a natural disaster: the 1989 Loma Prieta Earthquake. *Journal of Personality and Social Psychology*, 61, 115-121.
- Nolen-Hoeksema, S., Morrow, J., & Fredrickson, B. L. (1990). The effects of response styles on the duration of depressed mood: A field study. Stanford University.
- Nolen-Hoeksema, S., Morrow, J., & Fredrickson, B. L. (1993). Response styles and the duration of episodes of depressed mood. *Journal of Abnormal Psychology*, 102, 20-28.
- O'Hare, A. J. (2007). *The Fear Survey Schedule as a sensitive measure of stress: Evidence from ERPs*. The University of Kansas, Lawrence, KS.
- Ohira, H. (1996). Assessability of negative constructs in depression: An event-related brain potential and reaction time analysis. *Japanese Journal of Experimental Social Psychology*, 35(3), 304-316.
- Oliver-Rodriguez, J. C., Guan, Z., & Johnston, V. S. (1999). Gender differences in late positive components evoked by human faces. *Psychophysiology*, 36(2), 176-185.
- Osterhout, L., & Holcomb, P. J. (1995). Event-related potentials and language comprehension. In M. D. Rugg & M. G. H. Coles (Eds.), *Electrophysiology of Mind: Event-Related Brain Potentials and Cognition*. Oxford, NY, Tokyo: Oxford University Press.
- Perez-Edgar, K., Fox, N. A., Cohn, J. F., & Kovacs, M. (2006). Behavioral and

- electrophysiological markers of selective attention in children of parents with a history of depression. *Biological Psychiatry*, 60(10), 1131-1138.
- Persad, S. M., & Polivy, J. (1993). Differences between depressed and nondepressed individuals in the recognition of and response to facial emotional cues. *Journal of Abnormal Psychology*, 102(3), 358-368.
- Pierson, A., Ragot, R., Van Hooff, J., Partiot, A., Renault, B., & Jouvent, R. (1996). Heterogeneity of information-processing alterations according to dimensions of depression: an event-related potentials study. *Biological Psychiatry*, 40(2), 98-115.
- Polich, J. (2007). Updating P300: an integrative theory of P3a and P3b. *Clinical Neurophysiology*, 118(10), 2128-2148.
- Pollock, V., Cho, D. W., Reker, D., & Volavka, J. (1979). Profile of Mood States: the factors and their physiological correlates. *Journal of Nervous and Mental Disease*, 167(10), 612-614.
- Raes, F., Hermans, D., & Williams, J. M. (2006). Negative bias in the perception of others' facial emotional expressions in major depression: the role of depressive rumination. *Journal of Nervous and Mental Disorders*, 194(10), 796-799.
- Rich, B. A., Schmajuk, M., Perez-Edgar, K. E., Pine, D. S., Fox, N. A., & Leibenluft, E. (2005). The impact of reward, punishment, and frustration on attention in pediatric bipolar disorder. *Biological Psychiatry*, 58(7), 532-539.
- Ridout, N., Astell, A., Reid, I. C., Glen, T., & O'Carroll, R. E. (2003). Memory bias for emotional facial expressions in major depression. *Cognition and Emotion*, 17, 101-122.
- Robinson, M. S., & Alloy, L. B. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research*, 27(3), 275-292.

- Roelofs, J., Muris, P., Huibers, M., Peeters, F., & Arntz, A. (2006). On the measurement of rumination: a psychometric evaluation of the ruminative response scale and the rumination on sadness scale in undergraduates. *Journal of Behavior Therapy and Experimental Psychiatry, 37*(4), 299-313.
- Ruchsow, M., Reuter, K., Hermle, L., Ebert, D., Kiefer, M., & Falkenstein, M. (2007). Executive control in obsessive-compulsive disorder: event-related potentials in a Go/Nogo task. *Journal of Neural Transmission.*
- Ruys, K. I., & Stapel, D. A. (2008). Emotion elicitor or emotion messenger? Subliminal priming reveals two faces of facial expressions. *Psychological Science, 19*(6), 593-600.
- Scher, C. D., Ingram, R. E., & Segal, Z. V. (2005). Cognitive reactivity and vulnerability: empirical evaluation of construct activation and cognitive diatheses in unipolar depression. *Clinical Psychology Review, 25*(4), 487-510.
- Segal, Z. V., & Ingram, R. E. (1994). Mood priming and construct activation in tests of cognitive vulnerability to unipolar depression. *Clinical Psychology Review, 14*, 663-695.
- Segal, Z. V., & Shaw, B. F. (1986). Cognition in depression: A reappraisal of Coyne & Gotlib's critique. *Cognitive Therapy and Research, 10*, 671-694.
- Segrin, C. (2001). *Interpersonal Processes in Psychological Problems*. New York, NY: Guilford Press.
- Segrin, C., & Abramson, L. Y. (1994). Negative reactions to depressive behaviors: a communication theories analysis. *Journal of Abnormal Psychology, 103*(4), 655-668.
- Shea, M. T., Leon, A. C., Mueller, T. I., Solomon, D. A., Warshaw, M. G., & Keller, M. B. (1996). Does major depression result in lasting personality change? *Am J Psychiatry, 153*(11), 1404-1410.



- Siegle, G. J., Ingram, R. E., & Matt, G. E. (2002). Affective interference: An explanation for negative attention biases in dysphoria? *Cognitive Therapy and Research*, 26, 73-87.
- Simons, R. F., Graham, F. K., Miles, M. A., & Chen, X. (2001). On the relationship of P3a and the Novelty-P3. *Biological Psychology*, 56(3), 207-218.
- Smith, J. L., Johnstone, S. J., & Barry, R. J. (2007). Links Response Priming in the Go/NoGo Task: The N2 Reflects Neither Inhibition Nor Conflict. *Clinical Neurophysiology*, 118(2), 343-355.
- Spencer, K. M., Dien, J., & Donchin, E. (1999). A componential analysis of the ERP elicited by novel events using a dense electrode array. *Psychophysiology*, 36(3), 409-414.
- Steidtmann, D., Ingram, R. E., & Siegle, G. J. (2010). Pupil response to negative emotional information in individuals at risk for depression. *Cognition and Emotion*, 24, 480-496.
- Sumich, A. L., Kumari, V., Heasman, B. C., Gordon, E., & Brammer, M. (2006). Abnormal asymmetry of N200 and P300 event-related potentials in subclinical depression. *Journal of Affective Disorders*, 92(2-3), 171-183.
- Surguladze, S., Young, A. W., Senior, C., Brebion, G., Travis, M. J., & Phillips, M. L. (2004). Recognition accuracy and response bias to happy and sad facial expressions in patients with major depression. *Neuropsychology*, 18(2), 212-218.
- Suslow, T., Dannlowski, U., Lalee-Mentzel, J., Donges, U. S., Arolt, V., & Kersting, A. (2004). Spatial processing of facial emotion in patients with unipolar depression: a longitudinal study. *Journal of Affective Disorders*, 83, 59-63.
- Suslow, T., Junghanns, K., & Arolt, V. (2001). Detection of facial expressions of emotions in depression. *Perceptual and Motor Skills*, 92, 857-868.
- Swann, W. B., Jr., Wenzlaff, R. M., Krull, D. S., & Pelham, B. W. (1992). Allure of negative

- feedback: self-verification strivings among depressed persons. *Journal of Abnormal Psychology*, 101(2), 293-306.
- Swann, W. B., Jr., Wenzlaff, R. M., & Tafarodi, R. W. (1992). Depression and the search for negative evaluations: more evidence of the role of self-verification strivings. *Journal of Abnormal Psychology*, 101(2), 314-317.
- Teasdale, J. D., & Barnard. (1993). *Affect, cognition, and change: Re-modelling depressive thought*. Hove, U.K.: Erlbaum.
- Torta, R., Borio, R., Cicolin, A., Vighetti, S., & Ravizza, L. (1994). S-adenosyl-L-methionine normalizes P300 latency in patients with major depression: A preliminary report. *Current Therapeutic Research*, 55(7), 864-874.
- Tottenham, N., Tanaka, J., Leon, A. C., McCarry, T., Nurse, M., Hare, T. A., et al. (in press). The NimStim set of facial expressions: judgments from untrained research participants. *Psychiatry Research*.
- van Hoof, J. J., Jogems-Kosterman, B. J., Sabbe, B. G., Zitman, F. G., & Hulstijn, W. (1998). Differentiation of cognitive and motor slowing in the digit symbol test (DST): differences between depression and schizophrenia. *Journal of Psychiatry Research*(33), 99-103.
- Vanderploeg, R. D., Brown, W. S., & Marsh, J. T. (1987). Judgments of emotion in words and faces: ERP correlates. *International Journal of Psychophysiology*, 5(3), 193-205.
- Warner, V., Weissman, M. M., Fendrich, M., Wickramaratne, P., & Moreau, D. (1992). The course of major depression in the offspring of depressed parents. Incidence, recurrence, and recovery. *Archives of General Psychiatry*, 49(10), 795-801.
- White, M. (1995). Preattentive analysis of facial expressions of emotion. *Cognition and Emotion*, 9, 439-460.

- Wilson, E. O. (1999). *Consilience: The Unity of Knowledge*. New York: Vintage Books.
- Yao, S., Liu, M., Liu, J., Hu, Z., Yi, J., & Huang, R. (2010). Inhibition dysfunction in depression: event-related potentials during negative affective priming. *Psychiatry Research, 182*(2), 172-179.
- Yeun, E. J., & Shin-Park, K. K. (2006). Verification of the profile of mood states-brief: cross-cultural analysis. *Journal of Clinical Psychology, 62*(9), 1173-1180.
- Zanarini, M. C., & Frankenburg, F. R. (2001). Attainment and maintenance of reliability of axis I and II disorders over the course of a longitudinal study. *Comprehensive Psychiatry, 42*(5), 369-374.
- Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., et al. (2000). The Collaborative Longitudinal Personality Disorders Study: reliability of axis I and II diagnoses. *Journal of Personality Disorders, 14*(4), 291-299.
- Zauszniewski, J. A., & Rong, J. R. (1999). Depressive cognitions and psychosocial functioning: a test of Beck's cognitive theory. *Archives of Psychiatric Nursing, 13*(6), 286-293.

Appendix A Block Design to Minimize Ordering Effects, Stimulus Condition Effects, and to Distribute Equally Among Groups

	<i>Response</i>		<i>Target 1</i>		<i>Target 2</i>		<i>Target 3</i>		<i>Target 4</i>	<i>MemTask</i>
1	Right	SPracR	SAR	OPracR	OAR	HPracR	HBR	YPracR	YBR	MemTask
2	Left	SPracL	SAL	OPracL	OAL	HPracL	HBL	YPracL	YBL	MemTask
3	Right	HPracR	HAR	YPracR	YBR	SPracR	SBR	OPracR	OAR	MemTask
4	Left	HPracL	HAL	YPracL	YBL	SPracL	SBL	OPracL	OAL	MemTask
5	Right	OPracR	OAR	SPracR	SBR	YPracR	YBR	HPracR	HAR	MemTask
6	Left	OPracL	OAL	SPracL	SBL	YPracL	YBL	HPracL	HAL	MemTask
7	Right	YPracR	YAR	HPracR	HAR	OPracR	OBR	SPracR	SBR	MemTask
8	Left	YPracL	YAL	HPracL	HAL	OPracL	OBL	SPracL	SBL	MemTask
9	Right	SPracR	SBR	OPracR	OBR	HPracR	HAR	YPracR	YAR	MemTask
10	Left	SPracL	SBL	OPracL	OBL	HPracL	HAL	YPracL	YAL	MemTask
11	Right	HPracR	HBR	YPracR	YAR	SPracR	SAR	OPracR	OBR	MemTask
12	Left	HPracL	HBL	YPracL	YAL	SPracL	SAL	OPracL	OBL	MemTask
13	Right	YPracR	YBR	SPracR	SAR	OPracR	OAR	HPracR	HBR	MemTask
14	Left	YPracL	YBL	SPracL	SAL	OPracL	OAL	HPracL	HBL	MemTask
15	Right	OPracR	OBR	HPracR	HBR	YPracR	YAR	SPracR	SAR	MemTask
16	Left	OPracL	OBL	HPracL	HBL	YPracL	YAL	SPracL	SAL	MemTask
17	Right	SPracR	SAR	OPracR	OBR	HPracR	HBR	YPracR	YAR	MemTask
18	Left	SPracL	SAL	OPracL	OBL	HPracL	HBL	YPracL	YAL	MemTask
19	Right	HPracR	HAR	YPracR	YAR	SPracR	SBR	OPracR	OBR	MemTask
20	Left	HPracL	HAL	YPracL	YAL	SPracL	SBL	OPracL	OBL	MemTask
21	Right	OPracR	OAR	SPracR	SAR	YPracR	YBR	HPracR	HBR	MemTask
22	Left	OPracL	OAL	SPracL	SAL	YPracL	YBL	HPracL	HBL	MemTask
23	Right	YPracR	YAR	HPracR	HBR	OPracR	OBR	SPracR	SAR	MemTask
24	Left	YPracL	YAL	HPracL	HBL	OPracL	OBL	SPracL	SAL	MemTask
25	Right	SPracR	SBR	OPracR	OAR	HPracR	HAR	YPracR	YBR	MemTask
26	Left	SPracL	SBL	OPracL	OAL	HPracL	HAL	YPracL	YBL	MemTask
27	Right	HPracR	HBR	YPracR	YBR	SPracR	SAR	OPracR	OAR	MemTask
28	Left	HPracL	HBL	YPracL	YBL	SPracL	SAL	OPracL	OAL	MemTask
29	Right	YPracR	YBR	SPracR	SBR	OPracR	OAR	HPracR	HAR	MemTask
30	Left	YPracL	YBL	SPracL	SBL	OPracL	OAL	HPracL	HAL	MemTask
31	Right	OPracR	OBR	HPracR	HAR	YPracR	YAR	SPracR	SBR	MemTask
32	Left	OPracL	OBL	HPracL	HAL	YPracL	YAL	SPracL	SBL	MemTask

The top row represents the target stimulus type that participants were instructed to respond to in experimental blocks 1, 2, 3, and 4. Letters represent blocks of facial expression target stimuli. S = sad, H = happy, O = older, Y = younger. Thus, S1A indicates that sad facial expressions from part 1 of the “A” list of stimuli were the target for a particular block, whereas H2B indicates that happy expressions from part 2 of the “B” list were the target for a particular block. To control for ordering effects and stimulus condition effects, a separate chart was utilized when recruiting each of the four diagnostic groups.

## Appendix B

### Tables of ERP Planned Contrasts from Oddball Task

<b>Table 2. Between-Groups Planned Contrasts of ERP Component Mean Voltages</b>							
	<i>Group 1 (lesser magnitude)</i>	<	<i>Group 2 (greater magnitude)</i>	<i>Trial Type</i>	<i>N200</i>	<i>P300a</i>	<i>P300b</i>
1	Dysphoric formerly depressed		Nondysphoric never depressed	Sad distracter	no	N/A	
2	Nondysphoric never depressed		Dysphoric formerly depressed	Sad target			yes
3	Dysphoric formerly depressed		Nondysphoric never depressed	Happy targets			no
4	Dysphoric formerly depressed		Nondysphoric never depressed	Old & young targets			no

Table 1 outlines *between*-groups planned contrasts that were hypothesized to exhibit significant differences in mean voltage with respect to particular ERP components. Expected differences are highlighted in gray with an indication of whether or not the prediction was supported. The P300a was not reliably extracted and could not be tested for differences.

<b>Table 3. Within-Group Planned Contrast of ERP Component Mean Voltages</b>								
	<i>Group</i>		<i>Trial Type 1 (lesser)</i>	<	<i>Trial Type 2 (greater)</i>	<i>N200</i>	<i>P300a</i>	<i>P300b</i>
5	Dysphoric formerly depressed		Sad distracter		Happy distracter	no	N/A	
6	Dysphoric formerly depressed		Happy target		Sad target			yes

Table 2 outlines *within*-groups planned contrasts that were hypothesized to exhibit significant differences in mean voltage with respect to particular ERP components. Expected differences are highlighted in gray with an indication of whether or not the prediction was supported. The P300a was not reliably extracted and could not be tested for differences.

## Beck Depression Inventory, Second Edition

**BDI-II**

Date: \_\_\_\_\_

Name: \_\_\_\_\_ Marital Status: \_\_\_\_\_ Age: \_\_\_\_\_ Sex: \_\_\_\_\_

Occupation: \_\_\_\_\_ Education: \_\_\_\_\_

**Instructions:** This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

**1. Sadness**

- 0 I do not feel sad.
- 1 I feel sad much of the time.
- 2 I am sad all the time.
- 3 I am so sad or unhappy that I can't stand it.

**2. Pessimism**

- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to be.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

**3. Past Failure**

- 0 I do not feel like a failure.
- 1 I have failed more than I should have.
- 2 As I look back, I see a lot of failures.
- 3 I feel I am a total failure as a person.

**4. Loss of Pleasure**

- 0 I get as much pleasure as I ever did from the things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from the things I used to enjoy.
- 3 I can't get any pleasure from the things I used to enjoy.

**5. Guilty Feelings**

- 0 I don't feel particularly guilty.
- 1 I feel guilty over many things I have done or should have done.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

**6. Punishment Feelings**

- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

**7. Self-Dislike**

- 0 I feel the same about myself as ever.
- 1 I have lost confidence in myself.
- 2 I am disappointed in myself.
- 3 I dislike myself.

**8. Self-Criticalness**

- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all of my faults.
- 3 I blame myself for everything bad that happens.

**9. Suicidal Thoughts or Wishes**

- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

**10. Crying**

- 0 I don't cry anymore than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying, but I can't.

Subtotal Page 1

**Continued on Back**

**11. Agitation**

- 0 I am no more restless or wound up than usual.
- 1 I feel more restless or wound up than usual.
- 2 I am so restless or agitated that it's hard to stay still.
- 3 I am so restless or agitated that I have to keep moving or doing something.

**12. Loss of Interest**

- 0 I have not lost interest in other people or activities.
- 1 I am less interested in other people or things than before.
- 2 I have lost most of my interest in other people or things.
- 3 It's hard to get interested in anything.

**13. Indecisiveness**

- 0 I make decisions about as well as ever.
- 1 I find it more difficult to make decisions than usual.
- 2 I have much greater difficulty in making decisions than I used to.
- 3 I have trouble making any decisions.

**14. Worthlessness**

- 0 I do not feel I am worthless.
- 1 I don't consider myself as worthwhile and useful as I used to.
- 2 I feel more worthless as compared to other people.
- 3 I feel utterly worthless.

**15. Loss of Energy**

- 0 I have as much energy as ever.
- 1 I have less energy than I used to have.
- 2 I don't have enough energy to do very much.
- 3 I don't have enough energy to do anything.

**16. Changes in Sleeping Pattern**

- 0 I have not experienced any change in my sleeping pattern.

---

- 1a I sleep somewhat more than usual.
- 1b I sleep somewhat less than usual.

---

- 2a I sleep a lot more than usual.
- 2b I sleep a lot less than usual.

---

- 3a I sleep most of the day.
- 3b I wake up 1-2 hours early and can't get back to sleep.

**17. Irritability**

- 0 I am no more irritable than usual.
- 1 I am more irritable than usual.
- 2 I am much more irritable than usual.
- 3 I am irritable all the time.

**18. Changes in Appetite**

- 0 I have not experienced any change in my appetite.

---

- 1a My appetite is somewhat less than usual.
- 1b My appetite is somewhat greater than usual.

---

- 2a My appetite is much less than before.
- 2b My appetite is much greater than usual.

---

- 3a I have no appetite at all.
- 3b I crave food all the time.

**19. Concentration Difficulty**

- 0 I can concentrate as well as ever.
- 1 I can't concentrate as well as usual.
- 2 It's hard to keep my mind on anything for very long.
- 3 I find I can't concentrate on anything.

**20. Tiredness or Fatigue**

- 0 I am no more tired or fatigued than usual.
- 1 I get more tired or fatigued more easily than usual.
- 2 I am too tired or fatigued to do a lot of the things I used to do.
- 3 I am too tired or fatigued to do most of the things I used to do.

**21. Loss of Interest in Sex**

- 0 I have not noticed any recent change in my interest in sex.
- 1 I am less interested in sex than I used to be.
- 2 I am much less interested in sex now.
- 3 I have lost interest in sex completely.

13 14 15 16 17 18 19 20 A B C D E

## Appendix D

### Life Stress Inventory

Please read each group of statements carefully, then pick out the one statement in each group which best describes the way that you have been feeling during the **past two weeks including today!** Circle the number next to the statement you have picked. **Do not** leave any statements blank.

If several statements in the group seem to apply equally well, simply circle the largest number. Be sure that you do not mark more than one statement for item 34 (change in sleeping pattern) and item 35 (change in appetite).

#### **Item 1.**

- 0 My life is not stressful.
- 1 My life is as stressful as it used to be.
- 2 My life is more stressful than usual.
- 3 My life is unbearably stressful.

#### **Item 2**

- 0 I am not busy
- 1 I am as busy as others.
- 2 I am busier than other people.
- 3 I am busy all the time.

#### **Item 3**

- 0 I am not pressured at work.
- 1 I have an average amount of pressure on me at work.
- 2 I have more pressure on me at work than most people.
- 3 I have an extraordinary amount of pressure on me at work.

#### **Item 4**

- 0 I am no more restless or wound up than usual.
- 1 I feel more restless or wound up than usual.
- 2 I am so restless or agitated that It's hard to stay still.
- 3 I am so restless or agitated that I have to keep moving or doing something.

#### **Item 5**

- 0 I am not discouraged about my future.
- 1 I feel more discouraged about my future than I used to be.
- 2 I do not expect things to work out for me.
- 3 I feel my future is hopeless and will only get worse.

#### **Item 6**

- 0 I do not feel like a failure
- 1 I have failed more than I should have.



- 2 As I look back, I see a lot of failure.
- 3 I feel that I am a total failure as a person.

**Item 7**

- 0 I get as much pleasure as I ever did from the things I enjoy.
- 1 I don't enjoy things as much as I used to.
- 2 I get very little pleasure from the things I used to enjoy.
- 3 I can't get any pleasure from the things I used to enjoy.

**Item 8**

- 0 I don't feel particularly guilty.
- 1 I feel guilty over many things I have done or should have done.
- 2 I feel quite guilty most of the time.
- 3 I feel guilty all of the time.

**Item 9**

- 0 My life does not change much.
- 1 there are a normal number of changes occurring in my life right now.
- 2 There are a large number of changes occurring in my life right now.
- 3 There are an excessive number of changes in my life right now and I cannot control them.

**Item 10**

- 0 I don't feel I am being punished.
- 1 I feel I may be punished.
- 2 I expect to be punished.
- 3 I feel I am being punished.

**Item 11**

- 0 I feel the same about myself as ever.
- 1 I have lost confidence in myself.
- 2 I am disappointed in myself.
- 3 I dislike myself.

**Item 12**

- 0 I don't criticize or blame myself more than usual.
- 1 I am more critical of myself than I used to be.
- 2 I criticize myself for all my faults.
- 3 I blame myself for everything bad that happens.

**Item 13**

- 0 I do not have bad luck.
- 1 I have the same amount of luck as everyone else.
- 2 I have worse luck than normal.
- 3 I have bad luck frequently.

**Item 14**

- 0 I do not have a lot of responsibilities
- 1 I have an average number of responsibilities
- 2 I have more than an average number of responsibilities
- 3 I have an extraordinary number of responsibilities.

**Item 15**

- 0 I don't cry anymore than I used to.
- 1 I cry more than I used to.
- 2 I cry over every little thing.
- 3 I feel like crying but I can't.

**Item 16**

- 0 People are not rude to me.
- 1 People are rude to me on occasion.
- 2 People are rude to me quite often.
- 3 People are always rude to me.

**Item 17**

- 0 I have control over how I spend my time.
- 1 I have some control over how I spend my time.
- 2 I have little control over how I spend my time.
- 3 I have almost no control over how I spend my time.

**Item 18**

- 0 My home life is calm and peaceful.
- 1 My home life sometimes gets hectic.
- 2 My home life sometimes feels difficult to manage.
- 3 My home life feels out of control.

**Item 19**

- 0 I have not lost interest in other people or activities.
- 1 I am less interested in other people or activities than before.
- 2 I have lost most of my interest in other people or things.
- 3 It's hard to get interested in anything.

**Item 20**

- 0 I make decision about as well as ever.
- 1 I find it more difficult to make decisions than usual.
- 2 I have much greater difficulty in making decisions that I used to.
- 3 I have trouble making any decisions.

**Item 21**

- 0 My family is a source of support.
- 1 My family is a source of concern.
- 2 It can be stressful dealing with my family.
- 3 My family is a major source of stress.

**Item 22**

- 0 I do not feel I am worthless.
- 1 I don't consider myself as worthwhile or useful as I used to be.
- 2 I feel more worthless compared to other people.
- 3 I feel utterly worthless.

**Item 23**

- 0 I do not have any unexpected events in my life.
- 1 I have as many unexpected events in my life as others do.
- 2 I have more many unexpected events in my life than others do.
- 3. Unexpected events constantly occur in my life.

**Item 24**

- 0 I have as much energy as ever.
- 1 I have less energy than I used to have.
- 2 I don't have enough energy to do very much.
- 3 I don't have enough energy to do anything.

**Item 25**

- 0 I am no more irritable than usual.
- 1 I am more irritable than usual.
- 2 I am much more irritable than usual.
- 3 I am irritable all the time.

**Item 26**

- 0 My bills are easy to manage.
- 1 My bills sometimes concern me.
- 2 It can be stressful dealing with my bills.
- 3 My bills are a major source of stress.

**Item 27**

- 0 I do not feel sad.
- 1 I feel sad much of the time.
- 2 I am sad all the time.
- 3 I am so sad or unhappy that I can't stand it.

**Item 28**

- 0 I can concentrate as well as ever.
- 1 I can't concentrate as well as ever.
- 2 It's hard to keep my mind on anything for very long.
- 3 I find I can't concentrate on anything.

**Item 29**

- 0 I am no more tired or fatigued than usual.
- 1 I get tired or fatigued more easily than usual.
- 2 I am too tired or fatigued to do a lot of the things I used to do.
- 3 I am too tired or fatigued to most of the things I used to do.

**Item 30**

- 0 I have as many opportunities in my life as anyone else does.
- 1 My opportunities sometimes seem limited.
- 2 I do not have many opportunities.
- 3 I am trapped by my life.

**Item 31**

- 0 I have not noticed any recent changes in my interest in sex.
- 1 I am less interested in sex than I used to be.
- 2 I am much less interested in sex now.
- 3 I have lost interest in sex completely.

**Item 32**

- 0 Traffic does not bother me.
- 1 Traffic is no more annoying to me than it is to anyone else.
- 2 Traffic often irritates me.
- 3 Traffic is a major source of stress in my life.

**Item 33**

- 0 I don't have any thoughts of killing myself.
- 1 I have thoughts of killing myself, but I would not carry them out.
- 2 I would like to kill myself.
- 3 I would kill myself if I had the chance.

**Item 34**

- 0 I have not experienced any change in my sleeping pattern.
  - 1a I sleep somewhat more than usual OR
  - 1b I sleep somewhat less than usual.
  - 2a I sleep a lot more than usual OR
  - 2b I sleep a lot less than usual.
  - 3a I sleep most of the day OR
  - 3b I wake up 1-2 hours early and can't get back to sleep.

**Item 35**

0 I have not experienced any changes in my appetite.

---

1a My appetite is somewhat less than usual OR

1b My appetite is somewhat greater than usual.

---

2a My appetite is much less than before OR

2b My appetite is much greater than usual.

---

3a I have no appetite at all OR

3b I crave food all the time.

## Appendix E

### Ruminative Response Scale short form from the Response Styles Questionnaire

People think and do many things when they feel down. Please read each of the items below and indicate whether you never, sometimes, often, or always think or do each of the following things when you feel down, sad, or depressed. Please indicate what you *generally* do, not what you think you should do.

1. Go someplace alone to think about your feelings

Almost never          Sometimes          Often          Almost always

2. Isolate yourself and think about the reasons why you feel sad

Almost never          Sometimes          Often          Almost always

3. Go away by yourself and think about why you feel this way

Almost never          Sometimes          Often          Almost always

4. Write down what you are thinking about and analyze it

Almost never          Sometimes          Often          Almost always

5. Listen to sad music

Almost never          Sometimes          Often          Almost always

6. Think “Why do I always react this way?”

Almost never          Sometimes          Often          Almost always

7. Think about a recent situation, wishing it had gone better

Almost never          Sometimes          Often          Almost always

8. Think about how angry you are with yourself

Almost never          Sometimes          Often          Almost always

Appendix F

Profile of Mood States questionnaire

Below is a list of words that describe feelings that people have. Please read each word carefully. Then circle the number that best describes how you feel RIGHT NOW.

		Not at all	A little	Moderately	Quite a bit	Extremely
1.	Tense	0	1	2	3	4
2.	Angry	0	1	2	3	4
3.	Worn out	0	1	2	3	4
4.	Lively	0	1	2	3	4
5.	Confused	0	1	2	3	4
6.	Shaky	0	1	2	3	4
7.	Sad	0	1	2	3	4
8.	Active	0	1	2	3	4
9.	Grouchy	0	1	2	3	4
10.	Energetic	0	1	2	3	4
11.	Unworthy	0	1	2	3	4
12.	Uneasy	0	1	2	3	4
13.	Fatigued	0	1	2	3	4
14.	Annoyed	0	1	2	3	4
15.	Discouraged	0	1	2	3	4
16.	Nervous	0	1	2	3	4
17.	Lonely	0	1	2	3	4
18.	Muddled	0	1	2	3	4
19.	Exhausted	0	1	2	3	4
20.	Anxious	0	1	2	3	4
21.	Gloomy	0	1	2	3	4
22.	Sluggish	0	1	2	3	4
23.	Weary	0	1	2	3	4
24.	Bewildered	0	1	2	3	4
25.	Furious	0	1	2	3	4
26.	Efficient	0	1	2	3	4
27.	Full of pep	0	1	2	3	4
28.	Bad-tempered	0	1	2	3	4
29.	Forgetful	0	1	2	3	4
30.	Vigorous	0	1	2	3	4

*Please ensure you have answered every item.*

Appendix G

The Structured Clinical Interview for DSM-IV-I

<p><b>PAST MAJOR DEPRESSIVE EPISODE</b></p> <p>→ IF NOT CURRENTLY DEPRESSED: Have you <u>ever</u> had a period when you were feeling depressed or down most of the day nearly every day? (What was that like?)</p> <p>→ IF CURRENTLY DEPRESSED BUT FULL CRITERIA ARE NOT MET, SCREEN FOR PAST MDE: Has there ever been <u>another</u> time when you were depressed or down most of the day nearly every day? (What was that like?)</p> <p>IF YES: When was that? How long did it last? (As long as two weeks?)</p> <p>→ IF PAST DEPRESSED MOOD: During that time, did you lose interest or pleasure in things you usually enjoyed? (What was that like?)</p> <p>→ IF NO PAST DEPRESSED MOOD: What about a time when you lost interest or pleasure in things you usually enjoyed? (What was that like?)</p> <p>→ IF YES: When was that? Was it nearly every day? How long did it last? (As long as two weeks?)</p> <p>Have you had more than one time like that? (Which time was the worst?)</p> <p>IF UNCLEAR: Have you had any times like that in the past year?</p>	<p><b>MDE CRITERIA</b></p> <p>A. Five or more of the following symptoms have been present during the same two-week period and represent a change from previous functioning; at least one of the symptoms was either</p> <p>(1) depressed mood or</p> <p>(2) loss of interest or pleasure.</p> <p>(1) depressed mood most of the day, nearly every day, as indicated by either subjective report (e.g., feels sad or empty) or observation made by others (e.g., appears tearful). Note: in children and adolescents, can be irritable mood.</p> <p>(2) Markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day (as indicated either by subjective account or observation made by others)</p> <p><b>NOTE: IF MORE THAN ONE PAST EPISODE IS LIKELY, SELECT THE "WORST" ONE FOR YOUR INQUIRY ABOUT A PAST MAJOR DEPRESSIVE EPISODE. HOWEVER, <u>IF THERE WAS AN EPISODE IN THE PAST YEAR, ASK ABOUT THAT EPISODE EVEN IF IT WAS NOT THE WORST.</u></b></p>	<p>? 1 2 3</p> <p>? 1 2 3</p> <p>IF NEITHER ITEM (1) NOR (2) IS CODED "3," GO TO CURRENT MANIC EPISODE,</p>
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? = inadequate information    1 = absent or false    2 = subthreshold    3 threshold or true



<p>FOR THE FOLLOWING QUESTIONS, FOCUS ON THE WORST TWO WEEKS OF THE PAST MAJOR DEPRESSIVE EPISODE THAT YOU ARE INQUIRING ABOUT</p> <p>During that (TWO WEEK PERIOD)...</p> <p>...did you lose or gain any weight? (How much?) (Were you trying to lose weight?)</p> <p>IF NO: How was your appetite? (What about compared to your usual appetite?) (Did you have to force yourself to eat?) (Eat [less/more] than usual?) (Was that nearly every day?)</p> <p>...how were you sleeping? (Trouble falling asleep, waking frequently, trouble staying asleep, waking too early, OR sleeping too much? How many hours a night compared to usual? Was that nearly every night?)</p> <p>..were you so fidgety or restless that you were unable to sit still? (Was it so bad that other people noticed it? What did they notice? Was that nearly every day?)</p> <p>IF NO: what about the opposite – talking or moving more slowly than is normal for you? (Was it so bad that other people notice it? What did they notice? Was it nearly every day?)</p> <p>...what was your energy like? (Tired all the time? Nearly every day?)</p>	<p>NOTE: WHEN RATING THE FOLLOWING ITEMS, CODE "1" IF CLEARLY DIRECTLY DUE TO A GENERAL MEDICAL CONDITION, OR TO MOOD INCONGRUENT DELUSIONS OR HALLUCINATIONS</p> <p>(3) significant weight loss when not dieting, or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day. NOTE: in children, consider failure to make expected weight gains.</p> <p>Check if:  <input type="checkbox"/> Weight loss or decreased appetite  <input type="checkbox"/> Weight gain or increased appetite</p> <p>(4) insomnia or hypersomnia nearly every day  Check if:  <input type="checkbox"/> insomnia  <input type="checkbox"/> hypersomnia</p> <p>(5) psychomotor agitation or retardation nearly every day (observable by others, not merely subjective feelings of restlessness or being slowed down)  Check if  <input type="checkbox"/> psychomotor agitation  <input type="checkbox"/> psychomotor retardation</p> <p>(6) fatigue or loss of energy nearly every day</p>	<p>? 1 2 3</p> <p>? 1 2 3</p> <p>? 1 2 3</p> <p>? 1 2 3</p>
---	--	---

? = inadequate information    1 = absent or false    2 = subthreshold    3 = threshold or true

<p>During that time...</p> <p>...how did you feel about yourself? (Worthless?) (Nearly every day?)</p> <p>IF NO: What about feeling guilty about things you had done or not done? (Nearly every day?)</p> <p>...did you have trouble thinking or concentrating? (what kinds of things did it interfere with?) (Nearly every day?)</p> <p>IF NO: was it hard to make decisions about everyday things? (Nearly every day?)</p> <p>...were things so bad that you were thinking a lot about death or that you would be better off dead? What about thinking of hurting yourself?</p> <p>IF YES: did you do anything to hurt yourself?</p>	<p>(7) feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick) NOTE: CODE "1" OR "2" FOR LOW SELF-ESTEEM BUT NOT WORTHLESSNESS Check if: __worthlessness __inappropriate guilt</p> <p>(8)diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others) Check if: __diminished ability to think __indecisiveness</p> <p>(9) recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide NOTE: CODE "1" FOR SELF-MUTILATION W/O SUICIDAL INTENT Check if: __thoughts of own death __suicidal ideation __specific plan __suicide attempt</p>	<p>? 1 2 3</p> <p>? 1 2 3</p> <p>? 1 2 3</p>
--	---	--

? = inadequate information    1 = absent or false    2 = subthreshold    3 = threshold or true





<p>(did this begin soon after someone close to you died?)</p> <p>IF UNKNOWN: has there been any other time when you were (depressed/OWN WORDS) like this that did not occur after someone close to you died?</p> <p>→IF YES: GO TO *PAST MAJOR DEPRESSIVE EPISODE, 8A. 12 AND CHECK WHETHER THERE HAS BEEN ANY OTHER MAJOR DEPRESSIVE EPISODE THAT WAS NOT BETTER ACCOUNTED FOR BY BEREAVEMENT. IF SO, ASK ABOUT THAT EPISODE.</p> <p>→IF NO: TO TO *CURRENT MANIC EPISODE, *A. 18</p> <p>How old were you when (PAST MAJOR DEPRESSIVE EPISODE) started?</p> <p>How many separate times in your life have you been (depressed/ OWN WORDS) nearly every day for at least two weeks and had several of the symptoms that you described, like (SXS OR WORST EPISODE)?</p>	<p>E. The symptoms are not better accounted for by [simple] Bereavement, i.e., after the loss of a loved one, the symptoms persist for longer than 2 months or are characterized by marked functional impairment morbid preoccupation with worthlessness, suicidal ideation, psychotic symptoms or psychomotor retardation.</p> <p>MAJOR DEPRESSIVE EPISODE CRITERIA A, B, D, AND E ARE CODED "3"</p> <p>Age at onset of Past Major Depressive Episode coded above</p> <p>Total number of Major Depressive Episodes (CODE 99 IF TOO NUMEROUS OR INDISTINCT TO COUNT)</p> <p>NOTE: TO RECORD DETAILS OF OTHER PAST EPISODES, GO TO J. 9 (OPTIONAL)</p>	<p>1 SIMPLE BEREAVEMENT</p> <p>3 AT LEAST ONE EPISODE NOT SIMPLE BEREAVEMENT</p> <p>CONTINUE</p> <p>1 GO TO *CURRENT MANIC EPISODE *A. 18</p> <p>3 PAST MAJOR DEPRESSIVE EPISODE</p> <p>_____</p> <p>_____</p>
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**\*PAST MANIC EPISODE\***

**MANIC EPISODE CRITERIA**

NOTE: IF CURRENTLY ELEVATED OR IRRITABLE MOOD BUT FULL CRITERIA ARE NOT MET FOR A MANIC EPISODE, SUBSTITUTE THE PHRASE "Has there ever been another time..." IN EACH OF THE SCREENING QUESTIONS BELOW.

Have you ever had a period of time when you were feeling so good, "high," excited, or hyper that other people thought you were not your normal self or you were so hyper that you got into trouble?

(Did anyone say you were manic?) (Was that more than just feeling good?)

IF NO: What about a period of time when you were so irritable that you found yourself shouting at people or starting fights or arguments? (Did you find yourself shouting at people you really didn't know?)

A. A distinct period of abnormally and persistently elevated, expansive, or irritable mood...

Check if:  
 elevated, expansive mood  
 irritable mood

? 1 2 3

GO TO
*DYS-
THYMIC
DISOR-
DER,*
A. 38

A12

A12  
A12

When was that?

What was it like?

How long did that last? (as long as one week?) (Did you have to go into a hospital?)

...lasting at least one week (or any duration if hospitalization is necessary)

? 1 2 3

GO TO
*PAST
HYPOMANIC
EPISODE*
A. 33

A12

Have you had more than one time like that? (Which time was the most extreme?)

IF UNCLEAR: Have you had any times like that in the past year?

NOTE: IF THERE IS EVIDENCE FOR MORE THAN ONE PAST EPISODE, SELECT THE "WORST" ONE FOR YOUR INQUIRY ABOUT PAST MANIC EPISODE. IF THERE WAS AN EPISODE IN THE PAST YEAR, ASK ABOUT THAT EPISODE EVEN IF IT WAS NOT THE WORST.

?=inadequate information    1=absent or false    2=subthreshold    3=threshold or true

SCID-I (DSM-IV) **Fast Manic (FEB 1996 FINAL) Mood Episodes A. 29.**

FOCUS ON THE WORST PERIOD OF THE EPISODE THAT YOU ARE INQUIRING ABOUT.

IF UNCLEAR: During (EPISODE), when were you the most (OWN WORDS FOR MANIA)?

During that time...

..how did you feel about yourself?

(More self-confident than usual?) (Any special powers or abilities?)

3. During the period of mood disturbance, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:

(1) inflated self-esteem or grandiosity

? 1 2 3

A130

..did you need less sleep than usual?

IF YES: Did you still feel rested?

(2) decreased need for sleep (e.g., feels rested after only three hours of sleep)

? 1 2 3

A131

..were you much more talkative than usual? (Did people have trouble stopping you or understanding you? Did people have trouble getting a word in edgewise?)

(3) more talkative than usual or pressure to keep talking

? 1 2 3

A132

..were your thoughts racing through your head?

(4) flight of ideas or subjective experience that thoughts are racing

? 1 2 3

A133

..were you so easily distracted by things around you that you had trouble concentrating or staying on one track?

(5) distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli)

? 1 2 3

A134

..how did you spend your time? (Work, friends, hobbies?) (Were you so active that your friends or family were concerned about you?)

(6) increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation

? 1 2 3

A135

IF NO INCREASED ACTIVITY: Were you physically restless? (How bad was it?)

Check if:  
 increase in activity  
 psychomotor agitation

A136

A137

?=inadequate information    1=absent or false    2=subthreshold    3=threshold or true

During this time...

..did you do anything that could have caused trouble for you or your family? (Buying things you didn't need?) (Anything sexual that was unusual for you?) (Reckless driving?)

(7) excessive involvement in pleasurable activities which have a high potential for painful consequences (e.g., the person engages in unrestrained buying sprees, sexual indiscretions, or foolish business investments) ? 1 2 3

A138

AT LEAST THREE "B" SXs ARE CODED "3" (FOUR IF MOOD ONLY IRRITABLE)

1

3

A139

IF NOT ALREADY ASKED: Has there been any other time when you were (high/irritable/OWN WORDS) and had even more of the symptoms that I just asked you about?

-> IF YES: RETURN TO \*PAST MANIC EPISODE,\* A. 28, AND INQUIRE ABOUT WORST EPISODE.

-> IF NO: GO TO \*DYSTHYMIC DISORDER,\* A. 38.

NOTE: DSM-IV criterion C (i.e., does not meet criteria for a Mixed Episode) has been omitted from the SCID.

CONTINUE

IF NOT KNOWN: At that time, did you have serious problems at home or at work (school) because you were (SYMPTOMS) or did you have to go into a hospital?

D. The mood disturbance is sufficiently severe to cause marked impairment in occupational functioning or in usual social activities or relationships with others, or to necessitate hospitalization to prevent harm to self or others, or there are psychotic features.

1

3

A140

IF NOT ALREADY ASKED: Has there been any other time when you were (high/irritable/OWN WORDS) and had (ACKNOWLEDGED MANIC SYMPTOMS) and you got into trouble with people or were hospitalized?

DESCRIBE:

-> IF YES: RECODE CRITERION D as "3"

-> IF NO: GO TO \*PAST HYPOMANIC CRITERION C,\* A. 35.

CONTINUE ON NEXT PAGE

?=inadequate information 1=absent or false 2=subthreshold 3=threshold or true



Just before this began, were you physically ill?

IF YES: What did the doctor say?

Just before this began, were you taking any medications?

IF YES: Any change in the amount you were taking?

Just before this began, were you drinking or using any street drugs?

E. The symptoms are not due to the direct physiological effects of a substance (e.g., a drug of abuse, medication) or to a general medical condition

1 3  
DUE TO SUB-  
STANCE USE  
OR GMC

A14

IF THERE IS ANY INDICATION THAT THE MANIA MAY BE SECONDARY (I.E., A DIRECT PHYSIOLOGICAL CONSEQUENCE OF A GMC OR SUBSTANCE, GO TO \*GMC/SUBSTANCE,\* A.43, AND RETURN HERE TO MAKE A RATING OF "1" OR "3."

NOTE: MANIC-LIKE EPISODES THAT ARE CLEARLY CAUSED BY SOMATIC ANTIDEPRESSANT TREATMENT (E.G., MEDICATION, ECT, LIGHT THERAPY) SHOULD NOT COUNT TOWARD A DIAGNOSIS OF BIPOLAR I DISORDER BUT ARE CONSIDERED SUBSTANCE-INDUCED MOOD DISORDERS, A. 45.

REFER TO LIST OF GENERAL MEDICAL CONDITIONS AND SUBSTANCES, A. 21.

IF UNKNOWN: Has there been any other time when you were (high/irritable/ OWN WORDS) and were not (using SUBSTANCE/ill with GMC)?

- > IF YES: RETURN TO \*PAST MANIC EPISODE,\* A. 28, AND INQUIRE ABOUT OTHER EPISODE.
- > IF NO: GO TO \*DYSTHYMIC DISORDER,\* A. 38.

PRIMARY  
MOOD  
EPISODE

CONTINUE

**MANIC EPISODE CRITERIA**  
A, B, D, AND E ARE CODED "3"

1	3
GO TO *DYSTHY- MIC DIS- ORDER,* A. 38	PAST MANIC EPI- SODE

A14

How old were you when (PAST  
MANIC EPISODE) started?

Age at onset of Past Manic  
Episode coded above

—	—
---	---

A14

How many separate times in your  
life were you (HIGH/OWN WORDS)  
and had [ACKNOWLEDGED  
MANIC SYMPTOMS] for a period  
of time (or were hospita-  
lized)?

Number of Manic Episodes  
(CODE 99 IF TOO INDISTINCT  
OR NUMEROUS TO COUNT)

—	—
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A14

NOTE: TO RECORD DETAILS OF  
OTHER PAST EPISODES, GO TO  
J. 14 (OPTIONAL)

GO TO NEXT MODULE
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?=inadequate information    1=absent or false    2=subthreshold    3=threshold or true