The Influence of Parental Bonding on Information Processing in Depression

By Melissa Van Veldhuizen

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CHAIR: Dr. Rick Ingram

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Dr. Sarah Kirk

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Dr. Nancy Hamilton

Date Defended: 05/07/2021

The thesis committee for Melissa Van Veldhuizen certifies that this is the approved version of the following thesis:

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CHAIR: Dr. Rick Ingram

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Abstract

The cognitive perspective on risk for depression and depression reoccurrence posits that individuals who suffer from multiple episodes are at-risk because of latent depressotypic schemas. Dysfunctional parental bonding is one potential explanation for how such schemas are developed, and has also been associated with risk for depression. Thus, depressotypic schemas may provide some explanation for the relationship between parental bonding and depressive symptoms. The purpose of the current study was to examine whether those who have dysfunctional parental bonding, but are not currently depressed will show evidence of negative self-referent schemas in an information processing task. The current study also examined whether participant mood during this task was important for endorsement and recall during this information processing task. Measures of parental bonding, depression, anxiety, current mood, and a self-referent encoding task were administered to participants. Results showed that participants with high levels of mother care endorsed more positive words. Sad mood was also a predictor of endorsement; as sad mood decreased, endorsed positive words increased. This study helps elucidate the potential relationships between parental bonding, depressotypic schemas, and mood. Additionally, these findings provide insight into how those with dysfunctional bonding might be more at risk for depressive symptoms.

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Introduction

Depression is a serious public health issue that affects more than 300 million people globally. It is the leading cause of disability worldwide (World Health Organization (WHO), 2019). The recurrent nature of the disorder further contributes its insidious, far-reaching effects. Clinically significant episodes of depression are a well-recognized marker of future depressive episodes. Upwards of 80% of individuals who have had a prior episode of depression will experience additional episodes at some point in the future (Kessler, 2002), and as the number of recurrent episodes of major depressive disorder increases, the chance of additional episodes increases as well (Bulloch et al., 2013). Thus, the effect of prior episodes of depression appears to be incremental rather than binary; rather than presence or absence of additional episodes of depression, the number of recurrent episodes could be more predictive of risk for additional episodes. The connection between past episodes of depression and mechanism for the risk of future episodes is somewhat unclear, and several theories have attempted to explain why depression is often recurrent, and what puts people at risk for developing depression in the first place.

The cognitive perspective on risk for depression and depression reoccurrence posits that individuals who suffer from multiple episodes are at-risk because of latent depressotypic schemas. Beck was the first to suggest such a framework for heightened risk for depression; in particular, among the various elements of his model, he emphasized the role of cognitive schemas (Beck, 1967). In Beck's model and others that emphasize cognitive factors schemas are defined as cognitive structures that interact with input from the world and impact attention, cognitions, and memory search (Beck, 1967; Segal, 1988). Among depressed individuals, these schemas are often negatively-toned, and influence self-referent views and appraisal of the

environment. As such, they are strongly implicated in the onset, maintenance, and relapse of depressive episodes (Beck 1963, 1967, 1987). Beck also emphasized a developmental foundation in which schemas are acquired in childhood through interaction with the environment and its stressors (Beck, 1967, 1987). For instance, if a child's environment is characterized by abuse, high levels of stress, or other forms of negativity, schemas may form that direct attention toward negative stimuli, enhance recall of negative events, or alter input from the environment to fit negative schemas (Ingram et al., 2000). Hence, in those vulnerable to depression, those who are currently depressed, and those who have been depressed in the past, negative schemas are dysfunctional and are thought to engender negative attitudes about oneself (self-schemas), the world in general, and the future, which is known as the "cognitive triad" in depression literature (Beck, 1967, 1987). Such schemas can be thought of as "depressotypic schemas."

Thus, depressotypic schemas (about the self, the world, and the future) offer a possible explanation for risk, onset, maintenance, and reoccurrence of depressive symptoms and episodes. But where do these depressotypic schemas come from? What influences whether someone develops them? One explanation put forth by many researchers is the influence of dysfunctional parental bonding. Bonding in general refers to behaviors that influence the nature of relationships, either bringing people closer together or driving them further apart (Parker et al., 1979). Although there are a variety of dimensions of bonding that have been conceptualized by researchers, one of the most prominent and widely accepted conceptualizations is the Parental Bonding Inventory (PBI) (Parker et al., 1979). Since the creation of this bonding instrument, some researchers have tested and published various factor structures for the PBI (Lizardi & Klein, 2002; Cappelli et al., 2015; Murphy, Brewin, & Silka, 1997). However, this project utilizes the original factor structure, as it is psychometrically sound and has been widely used for

several years. According to Parker, bonding occurs on two constructs or dimensions: care and overprotection. Care is the extent to which a caregiver is warm, loving, supportive, and affectionate toward a child; overprotection is the extent to which a caregiver is controlling, intrusive, and punitive toward a child. High or low levels of both of these dimensions influence the type and quality of parental bond.

A secure (or optimal) bond is conceptualized as being low in overprotection and high in care (Parker et al, 1979). Caregiver affection, nurturing, and adequate protection promote independence and ability to form healthy relationships with others that presumably continues throughout life. A dysfunctional parental bond, on the other hand, is characterized by low levels of care and affection and high levels of overprotection, or "affectionless control" (Parker, 1983). Dysfunctional parental bonds have important implications for depression risk. High levels of parental control and overprotection are associated with depressive symptoms (Avagianou & Zafiropoulou, 2008; Hall et al., 2004; Parker, 1983), as are low levels of care and affection (Parker, Tupling, & Brown, 1995; Rapee, 1997). However, less is known about mechanisms of these associations.

Researchers have examined a number of potential mechanisms of this link between bonding and depressive symptoms, and depressotypic schemas as described above are one such mechanism. As discussed previously, research has demonstrated that depressotypic self-schemas are important in understanding cognitive vulnerability to depression (Segal, 1988; Ingram et al., 1998; McClain & Abramson, 1995). Negative, depressotypic self-schemas have been linked to dysfunctional parental bonding (Ingram, Overby, & Fortier, 2001). Cognitive theorists have posited that those with dysfunctional bonding develop schemas that give rise to various types of maladaptive cognitions about the self (self-schemas). Beck (1967) emphasized that depressotypic

schemas can lead to a negative bias in self- referential cognitions by impacting the way individuals attend to, process, interpret, and recall emotionally salient information. Such self-schemas have important implications for depression and are thought to result in a myriad of problems, including dysfunctional attitudes, negatively distorted information processing, and a negative cognitive triad – a set of beliefs regarding the self, the world, and the future.

Importantly, depressotypic, negative schemas in and of themselves are not sufficient to produce depression. Beck's model is an explicitly diathesis-stress approach; schemas are latent unless activated by appropriate stimuli (e.g. stressors in the environment that bring about a negative mood) (Beck, 1967). Relatedly, Teasdale (1988) proposed that dysfunctional, negative patterns of information processing become ingrained during initial depressive episodes and subsequently influence cognition when individuals experience future dysphoric mood states. Negative or dysphoric mood states are conceptualized as a stressor that activates such schemas. Dysphoric mood states 1) bias information processing, resulting in negative interpretations of events and the self, and 2) increase availability of negative self-referent constructs and dysfunctional attitudes, which increases the likelihood of recurrent episodes of depression (LeMoult et al., 2016).

Therefore, negative schemas do not have the potential to harmfully alter perception unless something occurs in the environment that triggers a negative mood state. Without activation of negative schemas, those who are at risk for depression and those who are not will appear to be similar on measures of maladaptive information processing and negative thoughts (Scher et al., 2005). Indeed, the importance of negative mood in activating negative schemas and negatively biased information processing has been demonstrated in recent research on depression (Fresco et al., 2006; Segal & Ingram, 1994; Raedt et al., 2010; Timbremont, & Braet, 2004;

Valle & Mateos, 2018). Although a variety of events may trigger negative self-schemas, events that lead to a negative mood state are featured prominently in cognitive models of depression. That is, induction of a negative mood is important because it activates schemas similar to those brought about by a negative, stressful life event. This activation facilitates access to the cognitive systems and structures of negative, harmful personal themes and information processing that is characteristic of depression (Ingram, 1984; Segal & Shaw, 1986; Teasdale, 1988). Such cognitive processing of stressful, negative events is often associated with self-blame, and interpreted through the lens of one's insufficiencies and inferiorities.

One way to measure activation of negative self-schemas is through self-referent encoding tasks (SRETs). This incidental recall task was developed from the depth-of-processing paradigm in experimental cognitive psychology and is built on the premise that when information is related to the self, it is encoded at deeper levels (Craik & Tulving, 1975). Participants are asked to rate the accuracy of self-referent adjectives and then are asked to recall these adjectives. The patterns of words recalled in this task are presumed to reflect the schemas that guide encoding and retrieval of information relevant to the content and activity of self-schemas (Ingram et al., 1994). Research using information processing tasks such as SRETs has also found that formerly depressed individuals exhibit similarly negative biases in memory and attention when in a negative mood state (for a review, see Joormann & Arditte, 2015). Individuals who have been depressed in the past (and are assumed to have latent depressive schemas according to Beck and other cognitive theorists) exhibit negative biases in memory and attention when in a negative mood state (for a review, see Joormann & Arditte, 2015) (Fritzsche et al., 2010; Kircanski, Mazur, & Gotlib, 2013). This supports the cognitive theory of depression that emphasizes schemas as important in risk, onset, maintenance, and relapse of depression; in those who have

been depressed in the past, their latent depressotypic schemas become active when in a negative mood state and influence the way they process information about themselves via depressotypic self-schemas (Fritzsche et al., 2010; Kircanski, Mazur, & Gotlib, 2013). However, some questions regarding depressotypic self-schemas and how they might help explain risk, onset, maintenance, and relapse of depression are still unanswered. What might influence whether people develop depressotypic schemas and information processing? Can self-referent encoding and information processing also tell us anything about whether these processes increase risk for developing depression, even in those who have not been depressed in the past and/or are not currently depressed?

It is known that dysfunctional parental bonding is related to depression, but how and why? Does parental bonding have an effect on the way that people process information? Is there an association between dysfunctional parental bonding and depressive symptoms because people who experience dysfunctional parental bonding develop negative, depressotypic self-schemas, which then make them more likely to develop depression? Thus, one goal of this research is to test the role of negative, depressotypic self-schemas in dysfunctional parental bonding and depressive symptoms. A second goal is to examine whether a negative mood state is necessary or important in the relationship between dysfunctional parenting and negative self-schemas. Do people with dysfunctional bonding need to be in a negative mood in order to show encoding and retrieval of negative, depressotypic self-referential information?

To test this idea, participants completed a measure that captures 2 subscales of parental bonding for each parent (care and overprotection) which was compared with scores on a measure of information processing called the Self Referent Encoding Task (SRET). Current mood (sadness, anxiety, and hostility) was measured before administration of the SRET for

each participant. In order to rule out the possible confounding effects of current depression, only participants who did not report current depression were included in the sample. Past depression and anxiety were also measured, but not used as exclusionary criteria. Based on prior research, I hypothesized that participants who are not currently depressed but have dysfunctional parental bonds (indicated by low care and/or high overprotection) will exhibit negative self-referent information processing via encoding (endorsement) and recall negative words as descriptive of themselves on the SRET. Examining these questions would allow for the elucidation of a link between dysfunctional parental bonding and risk for depression, as well as help understand whether mood plays a role in this potential association between parental bonding, information processing, and risk for depression.

Method

Participants

The final sample was comprised of 141 undergraduate student participants who were recruited through the KU's Psychology 104 pool. For additional detail about sample characteristics, please see Table 1. Participants were selected for scores on the Beck Depression Inventory (BDI) of 7 or below using the SONA pre-screen. In order to ensure that every participant had accurate data for both caregivers, participants were also removed from the sample if either their mother or father was not least "somewhat present" while they were growing up. Two attention checks were also utilized. Participants were asked to select particular ratings in both the mother and father PBI (which were in the middle of the survey). For example, "Please select "very like" for this question." Any participant who did not pass both attention checks by selecting the correct option was removed from the sample.

Procedure

Participants from KU's 104 pool completed the survey online via Qualtrics. The Beck Depression Inventory I (BDI; Beck et al., 1967) was used twice: once during the SONA pre-screen, and again during the study itself as a part of the survey to ensure that depressive symptoms have not increased since the initial screening. Any participant that scored above a 7 on the BDI was removed from the sample to make certain that results on the self-referent encoding task were not accounted for by current depressive symptoms. Participants also completed the Structured Clinical Interview for DSM-5 – Self Report (SCID – SR; First et al., 2015) past depression module to assess for past depression over the participant's lifetime. Participants were not screened out based on past depression scores, but it was measured to ensure that the sample did not have high levels of past depression in order to help ensure that past depression did not account for SRET scores.

Participants also completed the Beck Anxiety Inventory (BAI; Beck & Steer, 1993) to determine presence of anxious symptoms. Scores on this measure were used to determine whether anxiety symptoms could account for variance in SRET scores. Participants completed the Parental Bonding Inventory (PBI; Parker et al., 1979) to assess characteristics of care and overprotection in caregiver relationships. To ensure that measurement of bonding is as consistent as possible, only participants who grew up with two parents present from the ages of 0-16 whose parents were present at least "some of the time" will be included in the sample. Scores on this measure are used to calculate two subscales (care and overprotection) for each parent, and these subscales will be examined against various outcomes on the SRET.

Participants also completed the Sadness, Anxiety, and Hostility scales of the Multiple Affect Adjective Checklist (MAACL; Zuckerman & Lubin, 1965) to assess their current mood

state during survey completion. The scale scores on this checklist (sadness in particular given its importance in prior literature on self-referent encoding) will be analyzed alongside SRET scores to determine whether present sad mood is related to results on the SRET. Lastly, all participants completed the SRET (Derry and Kuiper, 1981) and an incidental recall of the words presented. Participants viewed each word for a maximum of 5 seconds before automatically proceeding onto the next word in order ensure similar levels of exposure to words between participants.

Measures

The Beck Depression Inventory I (BDI; Beck et al., 1967) is a 21-item self-report questionnaire designed to assess symptoms of depression and asks the participant about common depressive symptoms they have experienced over the last two weeks. The items are rated on a 0 (no presence of a symptom) to 3 (strong presence of a symptom) point scale, with a possible range of scores from 0-63. The reliability and validity of the BDI are adequate (Beck, Steer, & Garbin, 1988; Beck et al., 1967. Participants were not asked the question about suicidal thoughts on this measure, as it is not connected to our research questions. In addition, this study was administered online and there was no way to follow up with participants who may have endorsed suicidal thoughts, so the question was not included.

The Beck Anxiety Inventory (BAI; Beck & Steer, 1993) is a 21-item self-report questionnaire designed to assess anxiety symptoms and severity levels in adults and adolescents in the past month. Items are rated on a scale of 0 (no presence of a symptom) to 3 (strong presence of a symptom) point scale, with a possible range of scores from 0-63. The reliability and validity for the BAI are adequate (Beck et al., 1988; Fydrich, Dowdall, & Chambless, 1992).

The Structured Clinical Interview for DSM-5 – Self-Report (SCID – SR) (First et al., 2015) is a structured clinical interview that has been adapted for self-report. Participants will

only complete the SCID-SR for past depression, which is comprised of 11 questions about the presence or absence of depressive symptoms over the participant's lifetime based on the DSM-5 criteria. The range of scores is 0-9, with participants earning 1 point for each depressive symptom endorsed (some items map onto the same criteria for depression). Scores of 0-4 indicate absence of past depressive symptoms, and scores 5-9 indicate presence of past depressive symptoms. This variable was dichotomized into presence vs. absence of past depression based on these scores.

The Parental Bonding Instrument (PBI; Parker et al., 1979) assesses memory of parental behavior and attitudes toward the participant during his or her first 16 years of childhood. The questionnaire asks participants to recall memories of each parent separately (25 questions for each parent) and is comprised of two factors: care and overprotection. The care dimension assesses degree of empathetic and caring behavior on the part of the parent and the overprotection dimension assesses degree of intrusion and control on the part of the parent. The scale has demonstrated good reliability and validity (Parker et al., 1979; Parker et al., 1990; Wilhelm & Parker, 1990).

To measure current affect, participants completed the Sadness, Anxiety, and Hostility scales of the Multiple Affect Adjective Checklist (MAACL; Zuckerman & Lubin, 1965). The MAACL contains 132 items and participants are instructed to check each item that describes how they feel in the present moment. The psychometric properties of the MAACL have been evaluated and have demonstrated both reliability and validity (Lubin, Zuckerman, & Woodward, 1985).

The self-referent encoding task (SRET) (Derry and Kuiper, 1981) is a task that is thought to assess schema-related processing and recall of self-referent information. Participants made

decisions about whether positive and negative adjectives described them. Participants viewed one word at a time on screen and are asked to make rapid judgments about whether each word presented describes them, spending no more than 5 seconds on each word before being shown the next item automatically. Participants were not permitted to go back to previous words. The scale which participants will rate words from will range from 1 “no” to 7 “very much.” Participants will view a total of 42 words, one at a time and only once. The first 3 words and last 3 words were not included in scoring to control for primacy and recency effects. All participants were shown the same first and last words in the same exact order, but all of the other words were randomized for every participant. After completing the task, participants were unexpectedly asked to recall as many words as possible. Spelling of words was deemphasized to participants and they were given a minimum of 1 minute and a maximum of 3 minutes to recall words.

The words that were used are as follows: Positive words: joyful, brilliant, great, nice, excited, pleased, excellent, wonderful, loved, fun, friendly, helpful, confident, fantastic, content, playful, kind, funny. Negative words: angry, annoyed, ashamed, depressed, guilty, horrible, lonely, lost, mad, sad, scared, stupid, terrible, unhappy, unloved, unwanted, upset, worried. Neutral (primacy/recency): cooperative, healthy, realistic, caring, trustworthy, capable.

Results

A total of twelve linear regression models were conducted. All model coefficients were standardized for ease of comparison and are specified in Tables 2 through 5. For additional reference, Table 8 specifies the outcome and predictors for each model. Model 1 regressed Mother subscales (care and overprotection), MAACL Sad, and their interactions on the percentage of positive words that were endorsed by participants during the SRET. Overall, Model 1 was significant, $F(5,135)=16.94$, $p<.001$, and the adjusted $R^2 = .36$. In Model 1, mother

care was a significant predictor of the percent of positive words endorsed. That is, as mother care increased, the percentage of positive words endorsed by participants also increased. Participant mood during SRET ratings was also a significant predictor in Model 1. As sad mood measured by the MAACL decreased, the percentage of positive words endorsed by participants increased. Mother overprotection and the interaction between bonding variables and mood were not significant predictors of the percentage of positive words endorsed.

Model 2 regressed Father subscales (care and overprotection), MAACL Sad, and their interactions on the percentage of positive words that were endorsed by participants during the SRET. Overall, Model 2 was significant, $F(5,135)=11.10$, $p<.001$, and the adjusted $R^2 = .27$. In Model 2, sad mood as measured by the MAACL was a significant predictor of the percent of positive words endorsed. As sad mood decreased, the percentage of positive words endorsed by participants increased. All other variables in this model (Father care and overprotection and the interaction between bonding variables and mood) were not significant predictors of the percentage of positive words endorsed.

Model 3 regressed Mother subscales (care and overprotection), MAACL Sad, and their interactions on the percentage of positive words endorsed by participants that were recalled during the incidental recall on the SRET. Overall, Model 3 was not significant $F(5,135)=.95$, $p=.45$), and the adjusted $R^2 = .03$. None of the independent variables in this model were significant predictors of the percentage of positive words endorsed that were recalled by participants. Model 4 regressed Father subscales (care and overprotection), MAACL sad mood, and their interactions on the percentage of positive words endorsed by participants that were recalled on the SRET. Overall, Model 4 was not significant, $F(5,135)=.56$, $p=.73$, and the adjusted $R^2 = -.02$. As was the case in Model 3, none of the independent variables in this model

were significant predictors of the percentage of positive words endorsed that were recalled by participants.

Model 5 regressed Mother subscales (care and overprotection), MAACL Sad, and their interactions on the percentage of negative words endorsed by participants that were recalled during the incidental recall on the SRET. Overall, Model 5 was not significant, $F(5,135)=1.57$, $p=.17$), and the adjusted $R^2 = .02$. None of the independent variables in this model were significant predictors of the percentage of negative words endorsed that were recalled by participants. Model 6 regressed Father subscales (care and overprotection), MAACL Sad, and their interactions on the percentage of negative words endorsed by participants that were recalled during the incidental recall on the SRET. Overall, Model 6 was not significant, $F(5,135)=1.47$, $p=.20$), and the adjusted $R^2 = .02$. Sad mood as measured by the MAACL was the only significant predictor of percentage of negative words endorsed that participants recalled; as sad mood increased, so did the number of negative words recalled. No other independent variables in this model were significant predictors of the percentage of negative words endorsed that were recalled by participants.

Models 7 through 12 were conducted to determine whether introducing a measure of anxious symptoms would remove any significance found in prior models, which would have indicated that anxious symptoms were perhaps better explain variation in several components of SRET scores. Anxious symptoms (as measured by the BAI) were introduced to all models (1-6). All models' coefficients that were previously significant were not altered by the introduction of anxious symptoms with the exception of Model 6. Model 12 introduced anxious symptoms into Model 6, and showed that including anxious symptoms did change the coefficient significance for sad mood in Model 6, which was the only significant predictor in this model. It is of note that

this predictor's significance in Model 6 was not as robust as others predictors in other models, and this model was not significant overall before and after introduction of anxious symptoms. All other predictors remained nonsignificant when anxiety was introduced as a variable. For additional details on Models 7 through 12, please see Table 5.

Discussion

This study examined whether parental bonding was a significant predictor of the way that participants would endorse and recall self-referent information. Mood was also examined to determine whether participant sad mood predicted the way that participants endorsed and recalled self-referent information. As previously noted, these questions may have important implications for understanding a potential mechanism for how dysfunctional parental bonding increases risk for depression and whether sad mood is important when examining self-schemas and information processing. Note: a reference for each model is located in Table 8 at the end of the document.

As discussed previously, negative self-schemas are thought to be potential risk factors for depression and are also indicated in maintenance and re-occurrence of depression according to the cognitive perspective (Beck, 1967; Segal, 1988; Ingram et al., 1998; McClain & Abramson, 1995). Results in Model 1 showed that maternal care was a significant predictor of the endorsement of positive words as self-descriptive. This is in line with the idea that high levels of maternal care is perhaps an important protective factor against depressive symptoms through the power to buffer against negative schemas about the self. Existing literature supports maternal care as a protective factor against depressive symptoms (Ingram et al, 2001; Miranda et al., 2012), and the current findings suggest that one potential mechanism for this protection against

depressive symptoms is the influence of mother care on positive self-schemas and self-referent information processing.

Results in Models 1 and 2 showed that sad mood was a significant predictor of endorsement of words during the SRET, and showed an inverse relationship with the number of positive words endorsed by participants. These results suggest that participant mood during tasks like the SRET is important for how they will rate themselves during the task; when participants are in a sad mood, they endorsed fewer positive words as self-referent. These findings are in support of existing theories that emphasize a diathesis-stress approach to understanding cognitive vulnerability; in order for people to access negative schemas (self-schemas in this case), a sad mood is necessary for activation of negatively toned self-schemas (Beck, 1967; Teasdale, 1988; LeMoult et al., 2016; Fresco et al., 2006; Segal & Ingram, 1994; Raedt et al., 2010; Timbremont, & Braet, 2004; Valle & Mateos, 2018; Ingram, 1984; Segal & Shaw, 1986).

As discussed previously, results that suggest mood as an important predictor of results on self-referent encoding tasks may have important implications for understanding cognitive models of depression. Activation of negative schemas through negative mood facilitates access to the cognitive systems and structures of negative, harmful personal themes and information processing that is characteristic of depression (Ingram, 1984; Segal & Shaw, 1986; Teasdale, 1988). Such cognitive processing is thought to be similar to how one may process a stressful, negative event, and is often associated with self-blame and interpreted through the lens of one's insufficiencies and inferiorities. The findings of this study support that idea that a negative mood is indeed important for facilitation of cognitive processing through negative schemas, and showed that when sad mood was low, participants rated themselves much more positively on the SRET. It is also of note that this effect was detected without manipulation of participant mood –

mood was measured, but a negative or sad mood induction was not conducted. An even stronger affect may have been detected if mood had been experimentally manipulated.

Models 3 and 4 examined whether mother and father care and overprotection, sad mood, and their interactions had an effect on the percentage of positive words that were endorsed and later recalled during incidental recall. These models were not significant, and neither were any of their model coefficients. The lack of significance could be due to a myriad of reasons, including that there may simply no relationship between these predictors and self-referent recall. Largely the same was true for Models 5 and 6, which examined whether mother and father bonding dimensions, sad mood, and their interactions had an effect on the percentage of negative words that were endorsed and later recalled. Sad mood was a significant predictor in Model 6 (the model using father care and overprotection), but this significant coefficient became nonsignificant with the introduction of anxious symptoms into the model (Model 12). Lack of an activating stimulus (no induction of sad mood), and insufficient power to detect these affects are also possible explanations for nonsignificant effects for these models, but it is impossible to say for certain. As stated previously, nonsignificant effects of these models could be due to a myriad of reasons, including that there is simply no relationship among these variables.

Introducing a measure of anxious symptoms did not significantly change any results. As stated previously, it is of note that introducing anxious symptoms into Model 6 (Model 12) did alter the previously significant coefficient for mood as a predictor of the percentage of negative items endorsed by participants that were later recalled in the model for father bonding variables. However, it is important to consider that the model itself was not significant from the beginning. All other models were unaffected by the introduction of anxious symptoms. At the same time, it is also perhaps important to also consider the fact that depression and anxiety are somewhat

related in that they have overlapping diagnostic criteria, and both fall under the larger umbrella of negative affectivity. Therefore, controlling for anxious symptoms and attempting to rule it out as a potential predictor of results in this study may not be as meaningful when considering this perspective. Nevertheless, it is notable that the more robust findings regarding influence of mother care and mood on the percentage of positive words endorsed by participants remained unaffected by the introduction of anxious symptoms. This helps rule out the possibility that these results are not due to any shared variance between anxious symptoms, mother care, or sad mood.

Several limitations of the study must be noted. One limitation was the inability to recruit participants for high vs. low levels of mother care. Making such a comparison with a sample that had a wide range of care scores would have perhaps been more informative in examining the relationships between the variables in this study. Means and standard deviations on several variables were computed for each group and can be found in Table 7. The N for participants with low care (using the cutoff created by the author of the PBI) was 27, and the N for participants with high care was 114. Such disparate numbers in high vs. low care did not allow for a statistically sound or accurate comparison using regression or ANOVA. In the future, it would be important to ensure that a sample addressing these research questions have a wider range of scores and more comparable numbers in higher vs. lower care and overprotection. Another limitation of the study was the lack of an activating stimulus for sad mood. Given that sad mood was central to research questions, it may have been more advantageous to use a mood manipulation. Future research that examines questions related to self-referent encoding should endeavor to examine mood using activating stimuli (mood manipulation).

In addition, it may have also been more informative to include a range of BDI scores in this study in order to assess a full spectrum of depressive symptoms together with self-referent

encoding, mood, and parental bonding. It would afford the opportunity to learn more about how self-referent recall and parental bonding may differ among those with high and low levels of depression. Including a more complete range of depressive symptoms would provide richer data, and future studies on this topic should aim to do so. Lastly, it is important to consider the affect of the COVID-19 pandemic on the individuals who participated in this research and on research activities in general. Not only did this negatively impact recruitment, given the high levels of stress and exhaustion that are likely common for many amid the pandemic, completing a research study online may have been different than it would be normally and, as a results, may have led to higher-than-average levels of inattention or careless responding.

This study, along with abundant research in this area (Beck, 1967; Teasdale, 1988; LeMoult et al., 2016; Fresco et al., 2006; Segal & Ingram, 1994; Raedt et al., 2010; Timbremont, & Braet, 2004; Valle & Mateos, 2018; Ingram, 1984; Segal & Shaw, 1986) also suggest that sad mood is important for accessing negative self-schemas, which has important implications for how research assessing bonding should be conducted in the future, and how cognitive vulnerability to depression is understood. Future research should continue to explore these relationships with larger samples, more variability in parental bonding and depressive symptoms, and the inclusion of a mood manipulation to help elucidate the relationship between mood, parental bonding, cognitive vulnerability to depression, and self-referent encoding.

In sum, this study provides preliminary evidence to support the fact that levels of maternal care influence self-referent encoding, and thus the way that people see themselves. Results suggest that those who have high levels of maternal care have more positive schemas about themselves, which may in turn make them less likely to develop depression. This additionally suggests a potential mechanism for *how* mother care protects against depression,

furthering understanding about *how* higher levels of maternal care might be protective. Although one may not be able to change the type of parental bond experienced as a child, it *is* possible to affect change upon cognitive structures that may have developed as a result of dysfunctional parental bonding. Results presented in this study support the use of therapies for depression that include a cognitive component, like Beck's Cognitive Behavioral Therapy (CBT).

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Appendix 1: Tables

Table 1: Sample Characteristics

Variable	*N* (%)	*M* (*SD*)
Age	141	18.62(0.90)
Gender		
Male	79 (56%)	
Female	62 (44%)	
Ethnicity/Race	141	
White	117 (83%)	
Asian	8 (5.7%)	
Hispanic/Latino(a)	6 (4.3%)	
Black or African American	5 (3.5%)	
American Indian or Alaska Native	1 (0.7%)	
Other	4 (2.8%)	
Prefer not to specify	0 (0%)	

Table 2: Model Parameters – Positive Endorsement

Model	β	SE β	*p*	Adjusted R^2
Model 1				.36
Intercept	-.02	.07	.775	
PBI Mother Care	.28	.08	<.001***	
PBI Mother Overprotection	-.17	.08	.032*	
MAACL Sad	-.42	.07	<.001***	
PBI MC*MAACL	-.02	.09	.839	
PBI MO*MAACL	.09	.07	.224	
Model 2				.27
Intercept	-.01	.07	.884	
PBI Father Care	.14	.08	.089	
PBI Father Overprotection	-.01	.08	.865	
MAACL Sad	-.50	.07	<.001***	
PBI FC*MAACL	-.07	.07	.316	
PBI FO*MAACL	.01	.09	.875	

Table 3: Percent Positive Endorsed Recalled

Model	β	SE β	*p*	Adjusted R^2
Model 3				-.002
Intercept	.05	.09	.560	
PBI Mother Care	-.07	.10	.509	
PBI Mother Overprotection	-.11	.10	.267	
MAACL Sad	-.01	.09	.901	
PBI MC*MAACL	.20	.11	.075	
PBI MO*MAACL	.07	.09	.474	
Model 4				-.02
Intercept	.003	.09	.967	
PBI Father Care	-.14	.10	.145	
PBI Father Overprotection	-.12	.09	.197	
MAACL Sad	-.01	.09	.890	
PBI FC*MAACL	.03	.08	.693	
PBI FO*MAACL	.01	.10	.916	

Table 4: Percent Negative Endorsed Recalled

Model	β	SE β	*p*	Adjusted R^2
Model 5				.02
Intercept	-.007	.09	.933	
PBI Mother Care	-.13	.10	.184	
PBI Mother Overprotection	.01	.09	.890	
MAACL Sad	.13	.09	.162	
PBI MC*MAACL	.0004	.11	.997	
PBI MO*MAACL	.05	.09	.610	
Model 6				.02
Intercept	-.003	.08	.965	
PBI Father Care	-.09	.09	.322	
PBI Father Overprotection	-.14	.09	.142	
MAACL Sad	.18	.08	.032*	
PBI FC*MAACL	-.0005	.08	.995	
PBI FO*MAACL	.05	.10	.644	

Table 5: Models Examining BAI in Each Model

Model	β	SE β	*p*	Adjusted R^2
Model 7				.38
Intercept	-.02	.07	.801	
PBI Mother Care	.27	.08	<.001***	
PBI Mother Overprotection	-.15	.08	.060	
MAACL Sad	-.39	.07	<.001***	
BAI	-.13	.07	.056	
PBI MC*MAACL	-.01	.09	.881	
PBI MO*MAACL	.08	.07	.252	
Model 8				.29
Intercept	-.007	.07	.922	
PBI Father Care	.13	.08	.097	
PBI Father Overprotection	-.006	.08	.930	
MAACL Sad	-.46	.07	<.001***	
BAI	-.17	.07	.028*	
PBI FC*MAACL	-.06	.07	.425	
PBI FO*MAACL	-.006	.09	.944	
Model	β	SE β	*p*	Adjusted R^2
Model 9				-.005

Intercept	.05	.09	.570	
PBI Mother Care	-.06	.10	.527	
PBI Mother Overprotection	-.12	.10	.232	
MAACL Sad	-.02	.09	.791	
BAI	.06	.09	.481	
PBI MC*MAACL	.19	.11	.079	
PBI MO*MAACL	.07	.09	.457	
Model 10				-.02
Intercept	.002	.09	.977	
PBI Father Care	-.14	.10	.150	
PBI Father Overprotection	-.12	.09	.191	
MAACL Sad	-.02	.09	.790	
BAI	.05	.09	.565	
PBI FC*MAACL	.03	.08	.736	
PBI FO*MAACL	.02	.11	.870	
Model	β	SE β	*p*	Adjusted R^2
Model 11				.02
Intercept	-.009	.09	.914	
PBI Mother Care	-.13	.10	.198	
PBI Mother Overprotection	-.003	.10	.969	

MAACL Sad	.10	.09	.261	
BAI	.11	.09	.217	
PBI MC*MAACL	.0003	.11	.974	
PBI MO*MAACL	.05	.09	.573	
Model 12				.03
Intercept	-.006	.08	.938	
PBI Father Care	-.09	.09	.341	
PBI Father Overprotection	-.14	.09	.126	
MAACL Sad	.15	.09	.079	
BAI	.13	.09	.140	
PBI FC*MAACL	-.01	.08	.881	
PBI FO*MAACL	.06	.10	.539	

Table 6: Variable Means and Standard Deviations

Variable	M *(SD)*
BDI Total	2.16 (2.18)
BAI Total	7.74 (5.69)
MAACL Sad	10.8 (5.36)
PBI Mother Care	30.9 (5.28)
PBI Mother Overprotection	10.9 (6.04)
PBI Father Care	29.0 (7.32)
PBI Father Overprotection	7.94 (5.71)
SCID Past Depression Score	6.08 (1.87)
SRET Percent Endorsed Positive	0.74 (0.16)
SRET Percent Positive Endorsed Recalled	0.25 (0.13)
SRET Percent Negative Endorsed Recalled	0.22 (0.23)

Table 7: Variable Means and Standard Deviations by Low vs. High Care

Variable	*Low Care (N=27)* M *(SD)*	*High Care (N=114)* M *(SD)*
BDI Total	3.07 (2.54)	1.94 (2.04)
BAI Total	8.33 (4.34)	7.60 (5.98)
MAACL Sad	14.22 (5.15)	9.98 (5.10)
PBI Mother Care	21.78 (3.34)	33.04 (2.73)
PBI Mother Overprotection	15.04 (7.50)	9.93 (5.22)
PBI Father Care	22.37 (8.13)	30.56 (6.19)
PBI Father Overprotection	10.04 (6.73)	7.44 (5.35)
SCID Past Depression Score	6.0 (2.12)	6.12 (1.80)
SRET Percent Endorsed Positive	0.59 (0.08)	0.78 (0.16)
SRET Percent Positive Endorsed Recalled	0.24 (0.11)	0.26 (0.13)
SRET Percent Negative Endorsed Recalled	0.28 (0.16)	0.20 (0.24)

Table 8: Models

Model 1 = Percent Endorsed Positive ~ 1 + PBI MC + PBI MO + MAACL SAD + PBI MC x MAACL SAD + PBI MO x MAACL SAD
Model 2 = Percent Endorsed Positive ~ 1 + PBI FC + PBI FO + MAACL SAD + PBI FC x MAACL SAD + PBI FO x MAACL SAD
Model 3 = Percent Positive Endorsed Recalled ~ 1 + PBI MC + PBI MO + MAACL SAD + PBI MC x MAACL SAD + PBI MO x MAACL SAD
Model 4 = Percent Positive Endorsed Recalled ~ 1 + PBI FC + PBI FO + MAACL SAD + PBI FC x MAACL SAD + PBI FO x MAACL SAD
Model 5 = Percent Negative Endorsed Recalled ~ 1 + PBI MC + PBI MO + MAACL SAD + PBI MC x MAACL SAD + PBI MO x MAACL SAD
Model 6 = Percent Negative Endorsed Recalled ~ 1 + PBI FC + PBI FO + MAACL SAD + PBI FC x MAACL SAD + PBI FO x MAACL SAD
Model 7 = Percent Endorsed Positive ~ 1 + PBI MC + PBI MO + MAACL SAD + BAI + PBI MC x MAACL SAD + PBI MO x MAACL SAD
Model 8 = Percent Endorsed Positive ~ 1 + PBI FC + PBI FO + MAACL SAD + BAI + PBI FC x MAACL SAD + PBI FO x MAACL SAD
Model 9 = Percent Positive Endorsed Recalled ~ 1 + PBI MC + PBI MO + MAACL SAD + BAI + PBI MC x MAACL SAD + PBI MO x MAACL SAD
Model 10 = Percent Positive Endorsed Recalled ~ 1 + PBI FC + PBI FO + MAACL SAD + BAI + PBI FC x MAACL SAD + PBI FO x MAACL SAD
Model 11 = Percent Negative Endorsed Recalled ~ 1 + PBI MC + PBI MO + MAACL SAD + BAI + PBI MC x MAACL SAD + PBI MO x MAACL SAD
Model 12 = Percent Negative Endorsed Recalled ~ 1 + PBI FC + PBI FO + MAACL SAD + BAI + PBI FC x MAACL SAD + PBI FO x MAACL SAD