Understanding Risk and Symptoms: Parenting Styles, Symptoms of Depression, and Their Relationship to Automatic Thoughts

By
Joe Fulton
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Chair: Rick Ingram, Ph.D.

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Sarah Kirk, Ph.D., ABPP

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Steve Ilardi, Ph.D.

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The thesis committee for Joe Fulton
certifies that this is the approved version of the following thesis:

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Chair: Rick Ingram, Ph.D.

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Abstract

The aim of this study was to examine possible pathways through which parental bonding may relate to risk for depression. To examine perceptions of parenting style, current mood and levels of depressive symptoms, and the frequency of automatic thoughts, I collected data from 194 participants using the online survey service Amazon Mechanical Turk. Analyses revealed a mediational relationship between parental bonding levels and depressive symptoms through automatic thoughts. Overprotective parenting was not only mediated through negative thoughts, but also uniquely predicted somatic thoughts, whereas caring parenting did not. Caring parenting, mediated through positive automatic thoughts, uniquely predicted cognitive-affective symptoms, where overprotection did not. Overall this study suggests that the pathways through which parental bonding increases risk may not be as clear as originally assumed.

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Literature Review

Given the high prevalence of depression (Center for Behavioral Health Statistics and Quality, 2017), it is important to understand the factors that may play a role in vulnerability to the disorder. Understanding these factors not only allows for a better understanding of depression, it may also help lay the foundation for prevention of the disorder (Ingram, Miranda, & Segal, 1998). Although a number of variables (e.g., genetics) may predispose an individual to depression, developmental and environmental factors have also been recognized as potentially important risk factors (Sullivan, Neale, & Kendler, 2000; Ingram, 2003). Among developmental factors, dysfunctional parenting experiences have been identified as impactful. For example, dysfunctional parenting has been highlighted in a number of studies as a predictor for future depression (Alloy et al., 2001; Haugh, Miceli, & DeLorme, 2017; Ingram & Ritter, 2000; Lima, Mello, & de Jesus Mari, 2010; Parker, 1983). Although this relationship seems clear, the specific mechanisms by which dysfunctional parenting experiences are linked to the onset of depression are not yet well established (Ingram, 2003). This paper will examine the association between several variables that may be associated with vulnerability for depression. First, I explore developmental factors and the manner in which they may function in vulnerability to depression. Next, I examine the process through which these developmental factors may lead to differences in coping and views of self within a cognitive model of depression. Finally, I expand on these cognitive concepts by exploring how different profiles of depression may better explain the path from development to specific depressive symptoms.

Development and Depression

Early life experiences and development shape numerous aspects of an individual's functioning. One broadly recognized process that occurs in development is attachment. In brief,

attachment explains the process through which individuals form their first relationships, typically through interactions between parents and offspring. Attachment is viewed as an innate biological process and is typically conceptualized within an evolutionary context. Attachment is thought to serve as a way to maintain the security and safety of offspring. A number of factors can affect the degree of attachment such as the consistency of attachment behaviors, and the responsiveness of the attachment figure. As such, attachment can impact how children view the world and how they interact with others. Although attachment is rooted in biological systems, attachment theory also suggests that early interactions with caregivers provide the basis for cognitive processes that influence healthy adjustment, or in the case of disrupted attachment, unhealthy adjustment (Bowlby, 1969). Healthy attachment is linked to consistent beliefs in one's safety and the ability to develop independently from the attachment figure as well as a healthy view of self. Inconsistent or unhealthy attachment, on the other hand, is linked to a negative view of the world as unsafe, and a negative view of the self as unworthy of the attachment figure's affection and protection (Bowlby, 1980). This process reveals how the early interactions between children and attachment figures can shape the development of self, and influence how information is processed (Bowlby, 1969; Bowlby, 1980).

A construct closely associated with attachment is parental bonding: those parental behaviors that facilitate or impede healthy attachment (Parker, Tupling, & Brown, 1979). Specifically, bonding can set the stage for healthy relationships in which needs are communicated and met and mutual care is expressed. Moreover, a strong bond between the parent and child allows for security and protection, while also facilitating social and emotional growth (Grossman, Grossman, Kindler, & Zimmerman, 2008; Parker, 1992). Alternatively, bonds can become unhealthy when needs are not met, or caregivers are too controlling. As such,

dysfunctional bonding is associated with restricted autonomy, lack of warmth, or ignoring needs, which can harm adaptive development (Parker, 1992). The results of problematic bonding can be pervasive throughout a person's life and, within an interpersonal context, can negatively affect future relationships. Increased risk of depression and anxiety is also related to dysfunctional bonding patterns (Ingram, 2003; Lima, Mello, & de Jesus Mari, 2010; Yap & Jorm, 2015).

Parker, Tupling, and Brown (1979), outlined two dimensions of parenting that can determine whether bonding develops in a healthy and adaptive way or is disrupted: care and overprotection. The care dimension includes aspects of compassion, support, and nurturance, and allows for the development of a sense of safety and autonomy. Moreover, high levels of care are associated with a more positive and functional sense of self and with healthier psychological functioning overall (Parker, 1993; Parker, Barrett, & Hickie, 1992; Miranda, Soares, Moraes, Fossaluza, Serafim, & Mello, 2012). Alternatively, low levels of parental care are characterized by more indifference and lack of involvement in caregiving and are associated with higher risk for psychopathology and dysfunctional behaviors (Ingram & Ritter, 2000; McGinn, Cukor, & Sanderson, 2005; Parker, 1993; Picardi et al., 2013). The other dimemsion of parenting, overprotection, is characterized by intrusiveness in the child's life and high levels of control. Like low levels of care, overprotective parenting is associated with an increased likelihood for developing psychopathology in the future (McGinn, Cukor, & Sanderson, 2005; Parker, 1993; Parker, Barrett, & Hickie, 1992). Depression in particular, has been shown to have a higher prevalence in those who perceived their parents' as overprotective, and have a lower prevalence in those who perceived their parents as more caring (Alloy et al., 2001; Haugh, Miceli, & DeLorme, 2017; Ingram & Ritter, 2000; Parker, 1993; Parker, 1983; Randolph & Dykman, 1998). This pattern suggests that specific aspects of parenting may lead to lasting effects that

impact the likelihood of developing depression. Although the presence and strength of the relationship between parenting and risk for depression has been well supported, the specific mechanisms underlying this relationship are have not been well established.

Cognitive Model

In an effort to explain the relationship between parenting and risk for depression, some research has focused on the connection between parenting behaviors and the emergence of dysfunctional cognitive patterns. Dysfunctional cognition is featured in a number of cognitive models, which assert that depression stems from maladaptive cognitive patterns that develop in childhood (Alloy, Salk, & Abramson, 2017; Beck, 1967; Ingram et al., 1998). More specifically, these models suggest that early interactions and relationships shape how people navigate and react to difficult situations. These early lessons may form adaptive skills that allow for healthy reactions to adversity, or alternatively, may teach unhealthy ways of viewing and navigating problematic situations.

Within the context of depression, these unhealthy thinking patterns, called schemas by Beck (1967), develop as a result of early childhood experiences and increase the likelihood of depressive thinking patterns. This model has been supported by a number of studies that have shown possible pathways through which early childhood interactions can predict dysfunctional thinking patterns. Some studies highlight how perception of parenting can predict schemas or cognitive style from a young age (Haugh, Miceli, & DeLorme, 2017; McGinn, Cukor, & Sanderson, 2005; Randolf & Dykman, 1998; Shah & Waller, 2000), while others highlight that cognitions themselves, including negative automatic thoughts and ruminative patterns, predict cognitive style (Acharya & Relojo, 2017; Ingram, Overbey, & Fortier, 2001; Williams, Harfmann, Ingram, Hagan, & Kramer, 2015).

Taken together, the evidence points to a possible pathway through which early childhood interactions shape future cognitive styles and cognitions. Along these lines, Oppenheimer, Hankin, and Young (2017) examined parenting behaviors of the parents of 275 children or adolescents in real time. The children or adolescents completed a stressful task, during which the parents were allowed to interact with the child, and the parental behaviors were rated as positive (e.g., saying "You did great") or negative (e.g., saying "That was dumb of you"). They then assessed depressive symptoms over 18 months and found a conditional indirect effect of low positive parenting on the likelihood of experiencing a depressive episode, through an increase of negative cognitive style, assessed by an adolescent cognitive style questionnaire. These findings support the cognitive schema models of depression and the idea that early childhood experiences, and specifically experiences with parents, may create negative schemas and thus increase risk for depression.

Negative schemas are also thought to underlie negative self-statements (Kendall, 1992; Ingram et al., 1998). Like increases in negative thoughts, evidence also indicates a lack of positive thoughts is indicative of the presence of schemas (Ingram & Wisnicki, 1988). Lower levels of positive thinking have been found to be associated with depression (Ingram et al., 1998), and can be reasonably thought of as part of the negative cognitive profile that characterizes the disorder (Burgess & Haaga, 1994; Ingram & Wisnicki, 1988). However, the exact role that decreased positive thinking plays in both vulnerability and the depressive syndrome itself is not entirely clear. It may be, for instance, that positive self-statements serve as a buffer against stressful events, and to the extent that this buffer becomes diminished, depression may occur in response to stress. Support for this idea was found in a study by Ingram, Trenary, Odom, Berry, and Nelson (2007). They assessed positive and negative thinking in the

context of risk and found that those with the least risk also showed more positive self-statements. Another possibility noted by Ingram and Wisnicki (1988) is that negative thinking may quickly increase with emotional distress, but that positive cognition may only decrease when the emotional distress worsens in a manner that leads to depression. Hence, increased negative thinking and decreased positive thinking likely characterize the depressive state, but they may function in different manner within that state. Given these perspectives and data, it is important in the current study to examine negative cognitive styles as they pertain to both past parenting styles and depressotypic thinking.

Dimensional Approaches to Depression

Depression is distinguishable from other disorders by its core symptoms of "depressed mood" or "loss of interest or pleasure" – at least one of which is necessary for a diagnosis of Major Depressive Disorder (American Psychiatric Association, 2013). However, there are seven other symptoms according to the Diagnostic Statistical Manual (DSM-V) that can be present or absent as long as there are at least five total symptoms. The differences in symptom presentation that can manifest from these combinations of symptoms alone can result in heterogeneous manifestations of depression. That is, a person diagnosed with depression can display a very different symptom pattern relative to another person diagnosed with the same disorder.

In order to counteract this heterogeneity, it may be useful to consider a symptom profile view of depression rather than a syndromal view. Costello (1993) points out in this regard the numerous problems that measuring and conceptualizing depression as a syndrome can create. He notes the considerable overlap between depression and other disorders, how unreliable diagnosis can be, and how the experience of depression is a subjective one, which makes it difficult to measure as an objective syndrome. A symptomatic lens avoids the pitfalls that accompany the

syndromic view of depression. For example, focusing on symptoms allows for unique markers to be traced to certain symptoms (Kendler, Aggen, & Neale, 2013), or can expose how distinct the experiences of depression are from one person to another (Fried & Nesse, 2017). Fried and Nesse (2017) revealed this distinctness when they examined the STAR*D study and found that there were over 1000 unique symptom profiles of depression and most of those profiles did not have significant overlap between patients. Thus, instead of the typical categorical view of depression, it may be more useful to explore symptoms more specifically and examine how those symptoms relate to factors such as risk or vulnerability.

Despite the clear advantages of a symptomatic view of depression, measuring and comparing individual symptoms may be difficult. This difficulty stems from the many obstacles to coherence of measurement including the heterogeneity of measurement and the variety of classifications of depressive symptoms. It may be useful, instead, to focus on certain groups of symptoms to avoid the detriments of syndromic analysis while not examining the minutia of each individual symptom's role. A middle ground between syndrome focus and symptom focus is to group symptoms into clusters. This clustering allows for differentiation between types of symptoms while still focusing on their role within depression. However, it can be difficult to distinguish how many clusters of symptoms might conceivably exist given that different studies have found differing symptoms and clusters depending on measurement and sample (e.g., Kendler, Aggen, & Neale, 2013; Ballard et al., 2018). Thus, it may be more useful to identify a cluster of symptoms that is unique and meaningful, and work from there.

Because somatic symptoms have been identified somatic symptoms as unique and distinguishable from the other symptoms of depression, they are an ideal candidate for focus (e.g., Bekhuis, Boschloo, Rosmalen, & Schoevers, 2015; Bohman, Jonsson, Von Knorring,

Pareen, Olson, 2010; Bohman, Laftman, Cleland, Lundberg, Paaren, Jonsson, 2018; Lamela et al., 2017; Silverstein & Levin, 2014). Somatic symptoms are associated with physical issues including sleep difficulties, fatigue, and appetite changes and have been found to be unique from other depressive symptoms, while still being distinct from other disorders (Bekhuis et al., 2015). Somatic symptoms have also been associated with developmental patterns (Lamela et al., 2017; Silverstein & Levin, 2014) and have been found to be unique predictors for depression severity (Bohman et al., 2014; 2018). For instance, Bohman et al. (2018), conducted a longitudinal study with approximately 600 teens either suffering with depression or who were nondepressed controls. Results showed that children experiencing more somatic symptoms at baseline were more likely to be hospitalized for mood related disorders later in life. Somatic symptoms also predicted mental health hospitalizations for those who did not have depression but did have at least some somatic symptoms. Clearly, this subset of symptoms can be differentiated and may be important in understanding risk for depression. The remaining symptom cluster consists of cognitive symptoms, which are made up of problems with concentration, lack of energy, and restlessness, and affective or mood-related symptoms, which include sadness, guilt, and anhedonia. These two types of symptoms round out the remaining symptoms that are found in depression, according to the DSM-V. Using this clustering allows for a focus on the unique role that somatic symptoms may play while still examining all of the other symptoms within the disorder.

Indeed, these symptoms have distinct characteristics evidenced by the specific areas of functionality they impact. However, they can also be distinguished through statistical means. Both factor analysis and other structural equations have been able to identify that the cognitive-affective and somatic subtypes are distinct from one another (Fountoulakis, Iacovides,

Nimatoudis, Kaprinis, & Ierodiakonou, 1999; Killgore, 1999; Shafer, 2006; Lamela, Jongenelen, Morais, & Figueiredo, 2017). Within the Beck Depression Inventory (BDI) in particular, studies have shown clear distinctions between these two clusters of symptoms (i.e. Killgore, 1999; Helm & Boward, 2003). With these functionally and statistically based distinctions, we can examine different aspects of depression symptoms without focusing on every individual symptom that someone may present with. Indeed, using these clusters may allow for clearer delineation of symptoms and could allow for an expansion in our ability to explore the unique aspects of parenting and negative cognitions that influence vulnerability for depression as a whole. This separation may also allow for an examination of the different pathways through which parenting impacts risk.

Summary and the Present Study

In sum, parental bonding has been shown to be associated with a person's level of risk for developing depression. However, the pathways through which parenting impacts risk are not fully understood. One way to explain the pathway is poor parental bonding may lead to the development of dysfunctional cognitive patterns and negative attitudes toward the self, due to the lasting impact that parental bonding creates on how a person sees his or herself and the world. Though research has examined a mediation of parental bonding by cognitive style to predict risk for depression, there has been less exploration of this relationship in regard to specific profiles of depressive symptoms. Namely, studies have not explored how this pathway is related to somatic or cognitive-affective symptoms rather than the disorder as a whole. Given the relationship between parental bonding and cognitive style and the lack of clarity in their pathways, an exploration of the specific pathways through which these mechanisms are acting is important. More specifically, examining how parental bonding and cognitive style are associated with

depression from a symptom-focused lens may allow for an elucidation of the specific relationships between these concepts, and allow for a clearer view of the pathways of risk for depressive symptoms.

Given questions about how problematic bonding may be related to depression, the current study addressed three goals in regard to its focus on the relationship between automatic thoughts, current depressive symptoms, and parental bonding. The first goal was to examine whether there is an association between different parenting styles and the two depressive symptom profiles: somatic and cognitive-affective. The second goal was to better understand the relationship between these specific symptom profiles and current negative cognitive style as measured by both positive and negative automatic thoughts. The final goal was to examine the pathway through which parenting style may predict current depressive symptoms, specifically through mediation by automatic thoughts. In order to address these questions data on reports of parenting, levels of positive and negative automatic thoughts, and severity of depressive symptoms were collected.

Based on findings from past research (e.g., Ingram & Ritter, 2000; McLeod, Weisz, & Wood, 2007) I predicted that care would correlate negatively, and overprotection would correlate positively with depressive symptoms. I also hypothesized that higher levels negative automatic and lower levels positive automatic thoughts would be associated with higher levels of depressive symptoms, as has been found in the past (e.g., Hollon & Kendall, 1980; Ingram, Kendall, Siegle, Guarino, & McLaughlin, 1995; Ingram, Overbey, & Fortier, 2001). Finally, automatic thoughts were expected to fully mediate care and overprotection when predicting depressive symptoms (e.g., Haugh et al., 2017; Williams et al., 2015).

Method

Participants

The final sample consisted of 194 participants who were recruited from the online survey service offered through Amazon Mechanical Turk (MTurk). Each participant first completed a pre-screen questionnaire, and in order to qualify for the study they had to score between 5 and 25 on the Beck Depression Inventory. This screening measure was used in order to include people with at least some symptoms of depression and exclude those with a large number of depressive symptoms. Within the final population, the mean age was 40.94 (SD=13.79), and the sample was 80 percent female and 81 percent white.

Measures

Abbreviated Profile of Mood States (POMS-A; Shachams, 1983). The POMS-A is a 37-item self-report measure that assesses current affect. Participants are asked to rate which number best describes how they currently feel based on presented adjectives on a scale of 0-4 (Not at all – Extremely). The measure has a range of 0 to 148, with higher scores indicating increased presence of different mood states. Within the total score there are six subscales: Depression (8 words), Vigor (6 words), Confusion (5 words), Tension/Anxiety (6 words), Anger (7 words), and Fatigue (5 words) that can be used to describe current levels of specific mood states. Curran, Andrykowski, and Studts (1995), showed the measure to have good reliability and validity and found that the abbreviated version was still able to measure overall mood and the 6 mood states at least as well as the full-length measure. This measure was administered in order to control for current mood states and help verify that the depressive symptoms, and not current mood, were the underlying mechanisms behind the results.

Beck Depression Inventory (BDI; Beck, 1967). The BDI is a 21-item self-report measure that assesses current depressive symptom severity on a 4-point Likert scale. Each item is rated on a scale from 0-3 (0= absent symptom, 1=mild symptom, 2=moderate symptom, 3=severe symptom) with a range of 0-63. Higher scores on the measure reflect a presence of more symptoms of depression with higher severity. Meta-analysis of the measure has found it to have good internal reliability and validity (Beck et al., 1988).

Beck Anxiety Inventory (BAI; Beck et al., 1988). The BAI is a 21-item self-report measure that assesses current anxiety symptom on a 4-point Likert scale. Similar to the BDI, the BAI is rated on a 0-3 scale with a range of 0-63. Higher scores indicate a higher number and frequency of symptoms of anxiety that a person may be experiencing. Steer et al. (1993), found the measure to have high internal reliability and validity. This measure was administered in order to control for anxiety symptoms that are often found to be comorbid with depression symptoms in order to focus solely on depressive symptoms.

Parental Bonding Instrument (PBI; Parker, Tupling, and Brown, 1979). The PBI is a 50-item self-report questionnaire that assesses perceived parenting attitudes and behaviors for both mothers and fathers from the first 16 years of life. The measure describes a behavior that a parent may have exhibited and asks participants to rate how similar that behavior is to behaviors of their parents on a 4-point Likert scale (very like, moderately like, moderately unlike, very unlike). The instrument consists of two subscales: a caring subscale (comprising 12 items; range 0-36) and an overprotection subscale (with 13 items; range 0-39) for each parent. The 12 care items describe positive, caring parental behaviors and higher scores are associated with healthy bonding. The 13 overprotection items describe negative, controlling parental behaviors and higher scores are

associated with negative bonding. Wilhelm et al. (2004) found the measure to have good validity and reliability.

Automatic Thoughts Questionnaire-Negative (ATQ-N; Hollon & Kendall, 1980). The ATQ-N is a 30 item self-report measure that assesses the frequency of negative thoughts that a person may experience. The questionnaire presents the participant with negative self-statements that are then rated on a 5-point Likert scale for how frequently that person has those thoughts on a scale from 1 “not at all” to 5 “all the time.” The range of scores is from 30-150; higher scores indicating more negative thoughts over the past week. Hill et al. (1989) found the measure to be appropriately reliable and valid.

Automatic Thoughts Questionnaire-Positive (ATQ-P; Ingram & Wisnicki, 1988). The ATQ-P is a 30 question self-report questionnaire that measures the frequency of positive cognitions about the self. The questionnaire asks participants to identify how often they have experienced each positive thought on a 5-point Likert scale from 1 “never” to 5 “all the time.” The range of scores is from 30-150, with higher scores indicating more positive thoughts throughout the last week. Ingram et al. (1995) showed the study to be adequately valid and reliable.

Procedure

Participants were collected using Amazon Mechanical Turk (Litman, Robinson, & Abberbock, 2016), a public survey site. Before the study, participants were asked to read an information statement and required to click a button signaling agreement to the procedures and requirements of the study. Participants were also required to complete a validity measure to confirm they were not using a computer program to automatically complete the survey. The participants first completed a pre-screen to determine current level of depressive symptoms, then

if eligible, were contacted with the chance to complete the full study. After being contacted, the participants were included regardless of their current BDI score, which resulted in a slight shift of scores from between 5 and 25 to between 0 and 51. Each participant was compensated two cents for the short screening survey and those who continued were compensated $2.50 for completing the full survey. The full survey consisted of questionnaires that collected demographic information, and assessed current affect, current depressive and anxiety symptoms, past depressive symptoms, perceptions of their parental bond, and presence of dysfunctional thoughts and attitudes. All measures were completed online with the participants' personal devices through Amazon Mechanical Turk.

Results

Data Analysis

Prior to conducting analyses, I examined the data for inconsistencies in general answering patterns and on answers to validity measures imbedded in the surveys. Participants who failed to answer any validity measures were removed, bringing the total number of participants from 251 to 224. The sample was then narrowed to include only those who had indicated on the demographic questionnaire that both parents were present at least "some of the time" during their childhood. This restriction was used to avoid missing answers for some of the measures that were specified by parent and to increase consistency within the population. After removing these cases the final number of participants used for analysis was 194.

Analysis of the data was completed using SPSS version 23 along with an SPSS macro in order to examine the mediating relationship between variables. The PROCESS macro allows for an analysis of the direct and indirect relationship between predictors and mediators and uses

bootstrapping and SOBEL testing to determine the significance of these mediation affects when compared to zero.

Model Fit

Before conducting regression and mediation analysis, I conducted an analysis of the model fit and the possible impact of confounding variables. I first examined the patterns of responses and explored the overall pattern of variance. After finding the variance, skewness, and kurtosis in an acceptable range (see Table 1 for all descriptive statistics), I examined the relationships between the variables using bivariate and partial correlations. The variables, even when controlling for extraneous variables including age, anxiety symptoms, and current mood state, followed a predictable pattern. Parental care and positive automatic thoughts had a negative correlation with depression symptoms, whereas parental overprotection and negative automatic thoughts had a positive correlation with depressive symptoms (see Table 2 for correlations).

I next examined the overall fit of the models using regression analysis. Regression of depression symptoms onto the model revealed a high collinearity between the predictors than is acceptable for these analyses. The eigenvalues for the models were close to zero (Eigenvalues for total BDI scores regressed on the model: .02, .12, .16) indicating that small changes in the data would have an inflated their effect on results. However, after converting the scores into Z scores the eigenvalues increased into a normal range indicating the collinearity lowered to an acceptable level (Eigenvalues with Z scores: .63, .93, 1.00).

In order to explore the pathways through which parenting impacts the risk for someone developing depressive symptoms, I focused on a simple path analysis from parenting through automatic thoughts to depressive symptoms. Because of the multiple forms of parenting and

depression that were being examined, there were six models that were used. The first three models featured overprotection as the independent variable, automatic negative thoughts and automatic positive thoughts as simultaneous mediating variables, and depression symptoms as the dependent variable. Model one used total scores for depression symptoms (Figure 1), model two used cognitive-affective symptom scores (Figure 2), and model three used somatic symptom scores (Figure 3) as the independent variables. The same models were used with parental care as the independent variable, automatic negative thoughts and automatic positive thoughts as simultaneous mediating variables, and total scores for depression symptoms (Figure 4), cognitive-affective symptom scores (Figure 5), and somatic symptom scores (Figure 6) as three separate independent variables.

Initial Analyses

Prior to exploring the models proposed, initial analyses examined the relationship between bonding and the distinct depression symptoms: cognitive-affective and somatic. These analyses were then examined in relation to overall symptom scores. Initial correlational analysis revealed a significant positive correlation between overprotection and somatic symptoms as well as overall symptom scores, however, the relationship between overprotection and cognitive-affective scores was not significant (see Table 2). On the other hand, care had a significant negative correlation with overall scores and both subtypes of symptoms (see Table 2). Simple regression analyses, regressing overall scores and the two subtype scores onto parental styles, revealed similar patterns. Overprotection was a significant predictor for an increase in both overall scores ($F(192)=7.41$, $r_2=.04$), and somatic scores ($F(192)=17.06$, $r_2=.08$), but was not a significant predictor of cognitive-affective scores ($F(192)=3.58$, $r_2=.02$). Care was a significant predictor for a decrease of overall scores ($F(192)=15.59$, $r_2=.07$), cognitive-affective scores

($F(192)=13.52$, $r^2=.07$), and somatic scores ($F(192)=6.74$, $r^2=.03$). These results reveal the relationships between the two variables, while also revealing the individual differences that appear when separating depressive symptoms into clusters.

The next analyses were performed to examine the relationship between the depressive symptoms and automatic thoughts. The correlational analysis revealed a positive correlation between negative thoughts and overall scores, as well as symptom cluster scores. Positive thoughts correlated negatively with overall scores and symptom cluster scores. When examining the relationship between thoughts and parental styles, negative thoughts positively correlated with overprotection and had no significant relationship with care, whereas positive thoughts correlated positively with care and had no significant relationship with overprotection. Exploring further, the regression analysis revealed a similar pattern. When regressing negative thoughts onto overprotection, overprotection significantly predicted an increase in negative thoughts ($F(192)=4.66$, $r^2=.02$), however overprotection was not a significant predictor of positive thoughts ($F(192)=2.16$, $r^2=.01$). When regressing negative and positive thoughts on care, care was not a significant predictor of negative thoughts ($F(192)=3.22$, $r^2=.02$), but care did predict an increase in positive thoughts ($F(192)=12.00$, $r^2=.06$). These initial analyses seemed to mirror results of prior research and seemed to support our initial hypotheses. These results warranted further exploration into our third analysis goal, the examination of the specific pathways between parental bonding, automatic thoughts, and depressive symptoms

Mediation Analysis

The third and final goal of the study was to examine the pathways through which bonding and automatic thoughts may predict different depressive symptoms. This examination was done through mediational analysis. I conducted mediation testing using SPSS and multiple statistical

macros to verify the results of the path analysis, including the Sobel test (Baron and Kenny, 1986) and the PROCESS macro (Preacher & Hayes, 2008). Multiple regression analyses were conducted to test each model with each of the three depressive scores regressed on them, for a total of 6 path models. The first model conducted was with automatic thoughts separately regressed on both parenting styles, then the three depression symptom scores regressed onto either overprotection and both automatic thoughts or care and both automatic thoughts as predictor variables. In addition to the multiple regression analysis the PROCESS analysis tool was implemented to measure the significance of the direct and indirect effects of the main independent variable, parenting style. The PROCESS macro tested whether the indirect path from the independent variable to the dependent variable through the mediators differs significantly from zero. The analysis used bootstrapping with 5000 samples to determine the confidence intervals. This tool did not allow for multiple independent variables, so a model with both overprotection and care as independent variables was not included in this analysis. Table 3 shows the results of the PROCESS analysis, Figures 1-6 show a visual representation of the paths analyzed.

The bootstrap analyses revealed, first of all, that for all BDI scores both models predicted total scores significantly. Examining the mediation models individually, results indicated that negative thought scores (ATQ) were the only mediator for overprotective parenting, and further, mediated the relationship between overprotection and all three depression scores. Within the depression total score regression on overprotection ($F(190) = 90.73$, r_2=.59) the analysis revealed a significant mediation through negative thoughts (CI: Lower Limit .19 to Upper Limit 1.84, $p < .05$). Within the cognitive-affective depression score regression on overprotection ($F(190) = 101.32$, r_2=.62) the mediation analysis also revealed a significant mediation through

negative thoughts (CI: Lower Limit .08 to Upper Limit 1.18, $p < .05$). Finally, within the somatic depression score regression on overprotection ($F(190) = 21.69$, r_2=.26) the mediation analysis again revealed a significant mediation through negative thoughts CI: Lower Limit .02 to Upper Limit .34, $p < .05$, however overprotection still had a significant direct effect, indicating only a partial mediation of overprotection when predicting somatic symptoms. There was no evidence of mediation of overprotection through automatic positive thoughts (ATQ_P) (see Table 3 for all results and effects).

Furthermore, the models showed the pathways through which overprotection and automatic thoughts predict all three types of depressive symptoms. Figure one reveals the decrease in significance between direct ($\beta 1$) and indirect ($\beta 3$) effects of overprotection scores, due to the addition of automatic thoughts. It also reveals the mediation of overprotection through negative thoughts and the large influence that negative thoughts play in increasing symptoms, while positive thoughts have a smaller impact in decreasing symptoms. Figure 2 shows a similar pattern with cognitive-affective symptoms wherein the direct effect of overprotection scores decreases when automatic thoughts are added to the equation. Figure 2 also shows the relatively larger role of negative thoughts in increasing symptoms and the smaller role positive thoughts have in decreasing them. However, overprotection is not a significant predictor of cognitive-affective symptoms in any of the equations. Figure 3 reveals a unique pattern when predicting somatic symptoms through overprotection and automatic thoughts. Overprotection did have a slight decrease from direct to indirect effects, however the direct effect was still significant even after adding automatic thoughts to the equation. This indicates that overprotection is only partially mediated by negative thoughts. It is also clear that positive thoughts played little to no

role in predicting somatic symptoms, while negative thoughts and overprotection were larger predictors of somatic symptoms.

Examining the mediation models for the care variable it was revealed that positive thoughts (ATQ-P) were the only mediator for care scores, and it only mediated the relationship to the total and cognitive-affective depression scores. However, the direct effects of care on both total scores and cognitive-affective scores were also significant, indicating only a partial mediation. Within the depression total score regression on care ($F(190) = 95.38$, r_2=.60) the mediation of care through positive thoughts was significant (CI: Lower Limit -.74 to Upper Limit -.14, $p < .05$). Within the cognitive-affective depression score regression on care ($F(190) = 106.53$, r_2=.63) the mediation analysis also revealed a significant mediation through positive thoughts (CI: Lower Limit -.71 to Upper Limit -.16, $p < .05$). Within the somatic depression score regression on care ($F(190) = 18.19$, r_2=.22) the mediation analysis revealed that care's direct effect was significant (–.46, $p < .05$), but the indirect effect was not found to be different than zero. Thus, there was no evidence of mediation of care through automatic thoughts for somatic symptoms (see Table 3 for all results and effects).

Exploring further, Figure 4 shows the decrease in effect between direct ($\beta 1$) and indirect ($\beta 3$) effects of care scores because of the addition of automatic thoughts. This shift suggests that care is partially mediated through automatic thoughts, though there was still a significant direct effect of care. This model also reveals the larger effect that negative thoughts have on increasing depressive symptoms and the smaller effects of positive thoughts and care that decreases symptoms. Figure 5 shows a similar pattern for predicting cognitive-affective symptoms, although care had a smaller, but still significant effect. Figure 6 illustrates a unique pattern of prediction in that negative thoughts were the only significant predictor of somatic symptoms.

Suggesting care and positive thoughts had insignificant effects when predicting this subset of symptoms.

Discussion

This study aimed to explore the relationships between parental bonding, dysfunctional cognitive patterns, and depressive symptoms. The first goal was to examine whether there is an association between different parenting styles and the two depressive symptom profiles. The second goal was to better understand the relationship between these specific symptom profiles and current negative cognitive style as measured by both positive and negative automatic thoughts. The final goal was to examine the pathway through which parenting style may predict current depressive symptoms, specifically through mediation by automatic thoughts.

Preliminary analysis revealed that care and overprotection correlated with depressive symptoms in the predicted direction, supporting the initial hypothesis. Care was negatively correlated with overall depression scores and both subtypes of depressive scores, indicating that as care scores increase, depressive symptoms decrease. Overprotection was positively correlated with only BDI total scores and somatic scores, indicating that as overprotection scores increased, overall BDI and somatic depressive symptoms increased. These patterns are consistent with findings from other studies that have examined these relationships (Ingram & Ritter, 2000; McLeod, Weisz, & Wood, 2007), and as such, further support a relationship between dysfunctional parental bonding and depressive symptoms.

Although results for parental care were in the predicted direction, overprotection was not significantly correlated with the cognitive-affective scores. Though this was not expected, this result may explain some of the insignificant results involving overprotective parenting when compared to care (Patton, Coffey, Posterino, Carlin, & Wolfe, 2001; McLeod, Weisz, & Wood,

2007). That is, previous studies have found only care to be a significant predictor of overall depressive symptoms. However, it is possible that if these studies examined both subtypes of depression, overprotection may have predicted depressive symptoms, but only the somatic subtype. Such examinations are important from the standpoint of understanding.

As with parental care, the relationship between automatic thoughts and depressive symptoms aligned with the hypothesis. Negative thoughts were positively associated with depressive symptoms, (i.e., as thoughts increased, symptoms also increased) and positive thoughts were negatively correlated with depressive symptoms (i.e., as thoughts increased, symptoms decreased). These findings were consistent with other studies that examined automatic thoughts and depression (e.g., Ingram, Overbey, & Fortier, 2001; Ingram et al., 1995; Hollon & Kendall, 1980). The present study extends these findings by not only exploring the relationships between automatic thoughts and depression, but also exploring the unique relationship within the two subtypes of depression. Regression analysis revealed that negative thoughts predicted both subtypes of depression, whereas positive thoughts were only predictive of cognitive-affective symptoms. This unique finding may indicate that positive thoughts might reduce cognitive affective symptoms but have no effect on the somatic symptoms. Though purely speculation, this finding could suggest that increasing positive thoughts in therapy may be less important than decreasing negative thinking, especially when a client is experiencing more somatic symptoms.

After completion of initial analyses, I explored the specific pathways through which parental style, cognitive style, and depressive symptoms may be related. Regression analysis indicated that the relationship between depressive symptoms and parental bonding was completely or partially dependent on the relationship between bonding and automatic thoughts, hence the change in automatic thoughts that bonding predicts better explains the shift in

depressive symptoms than bonding alone. Moreover, the pathways through which overprotective and caring parenting predicted symptoms of depression were either fully or partially mediated by automatic thoughts, dependent on the types of symptoms being predicted. The relationship between overprotection and cognitive-affective symptoms as well as overall symptom scores was fully explained by the increase in negative thinking. This result suggests that overprotective parenting may encourage negative thinking that could increase risk for developing depressive symptoms. However, I found a different pattern when examining somatic symptoms separately. Overprotection not only predicted somatic symptoms through an increase in negative thoughts, it was also directly associated with somatic symptoms outside of its relationship with negative thoughts. This pattern suggests that outside of the increase in negative thinking, there may be other factors that explain the effect of overprotection predicting the increase in somatic symptoms. These patterns indicate that there may be other factors that explain the unique relationship between overprotection and somatic symptoms.

Analyses of parental care also revealed a nuanced relationship between care, automatic thoughts, and depressive symptoms. Parental care predicted both an increase in positive thoughts and a decrease in depressive symptoms partially linked to the increase in positive thinking. The partial pathway through positive thoughts indicates that positive thoughts were not the only pathway that decreased depressive symptoms. Within overall depression and cognitive-affective scores, care was associated with lower symptom levels through its relationship with positive thoughts and through its own relationship with depressive symptoms. Thus, although care predicted higher levels of positive thinking, which decreased overall and cognitive-affective symptoms, there are other factors responsible for the predicted increase in these symptoms as well. The relationship between care and somatic symptoms, on the other hand, was not evident in

theses analyses. These patterns indicate that levels of care do seem to influence the likelihood of experiencing depressive symptoms, but only cognitive-affective symptoms.

After examining these pathways through the symptomal lens of depression, the results reveal the unique impact of care on cognitive-affective symptoms and overprotection on somatic symptoms. Clearly, these distinct pathways suggest a more complicated model of risk than has been previously presumed. Bonding does indeed predict risk through a shift in thinking patterns, however the influence of bonding and these thinking patterns depends on the symptoms of depression that are predicted. Indeed, these findings suggest a need to shift our view of risk from a singular pathway to a more complex model and warrants further study. Moreover, because the impacts of both care and overprotection were not fully explained by automatic thoughts, this may indicate that the relationships between the variables may act through other mechanisms to influence vulnerability for depression. Also important is the unique way that overprotection was related to only somatic symptoms, and care was related to only cognitive-affective symptoms. Hence, these results could point to different aspects of parental bonding that impact how a person expresses or experiences negative affect.

Limitations

As with all research, possible limitations need to be considered. First, the study used an online platform to recruit participants, which may have skewed the sample. Although MTurk has been validated by some researchers (e.g., Berinsky & Huber, 2012; Landers, & Behrend, 2015) it is difficult to determine how generalizable this sample is to the population. Also, the average age of participants was around 40 years old, with some participants as old as 70. Although this sample may be more generalizable than a college sample, this age variance may have had an impact on the retrospective measurements such as the PBI. Although Wilhelm, Niven, Parker

and Hadzi-Pavlovic (2005) found the measure to be valid in longitudinal studies after 20 years, there may still be some differences in how a person remembers styles of parenting after a certain age. Finally, the cross-sectional style of this study limits the conclusions about causality and generalizations that can be made from this data.

Conclusions and Future Directions

The present study extended research into the pathways through which parental bonding may impact the risk for developing symptoms of depression. Through mediational analysis, I found an indirect relationship between bonding, automatic thoughts, and depressive symptoms. Caring parenting predicted higher levels of positive thinking and less depressive symptoms, whereas overprotective parenting predicted higher levels of negative thinking and more depressive symptoms. These relationships imply that parenting style may contribute to how a person thinks about themselves and may contribute to their risk for depression. The present study not only measured how both care and overprotective bonding styles distinctly predicted depressive symptoms, but also distinguished the unique prediction of cognitive-affective and somatic depressive symptoms. Previous research conceptualizing depression as unidimensional has yielded mixed results (e.g., Achara & Relojo, 2017; Williams et al., 2015; Ingram & Ritter, 2000). Achara & Relojo (2017), among others, were only able to identify care as a contributing factor to predicting depression and did not find a significant effect of overprotection. This categorization of symptoms reveals the unique role that overprotection and care play in predicting different symptoms of depression and reveals new pathways through which bonding can impact risk for depression.

In sum, this study considered how parenting style predicts both overall and sub-categories of depressive symptoms through an automatic-thought mediated pathway. Previous

research has shown clear links between parental bonding and the development of depression (Alloy et al., 2001; Haugh, Miceli, & DeLorme, 2017; Ingram, Miranda, & Segal, 1998; Lima, Mello, & de Jesus Mari, 2010), but little attention has be paid to how bonding may impact risk. The data reported here suggest that the pathway between bonding and depressive symptoms is more complex than originally expected. This owes in part to the fact that depression is not a unitary phenomenon, and that different subsets of depressive symptoms may yield different relations with parental bonding. Likewise, positive and negative cognition can function independently, and may exert a differential influence depending on whether thinking patterns are positive or negative. Clearly there are other potentially important pathways between bonding and depressive symptoms, and correspondingly, many pathways leading to depressive symptoms. An abundance of data have shown the pernicious effect that problematic parenting behaviors have on depression, and data such as those reported here may provide some clues about the nature of caregiver-child interactions and the development of depression through cognitive patterns. Indeed, dysfunctional bonding styles may lead to a propensity to think about the world and the self in a negative fashion, and thus provide fertile ground for the stressful events to be interpreted as indicative of personal deficits. As these results suggest, the root of such dysfunctional interpretation patterns may lie in part in problematic caregiver-offspring interactions and the negative cognitive apparatus that these interactions build.

Tables

Table 1

Descriptive statistics for model variables

Variables	Mean	Range	SD
BDI Total	15.65	0-51	8.20
BDI Cognitive-Affective	11.28	0-40	6.36
BDI Somatic	4.06	0-13	2.47
PBI Overprotection	28.36	0-67	13.80
PBI Care	42.19	0-72	16.87
ATQ Positive	81.61	30-139	23.15
ATQ Negative	70.63	30-142	24.88

Table 2

Correlation table for model variables.

Variables	BDI Total	BDI Cognitive-Affective	BDI Somatic	PBI Overprotection	PBI Care	ATQ Positive	ATQ Negative
BDI Total	1						
BDI Cognitive-Affective	.97**	1					
BDI Somatic	.74**	.55**	1				
PBI Overprotection	.19**	.14	.29**	1			
PBI Care	-.27**	-.26**	-.18*	-.37**	1		
ATQ Positive	-.47**	-.52**	-.20**	-.11	.24**	1	
ATQ Negative	.73**	.74**	.46**	.15*	-.13	-.37**	1

Note. *p< .05. ** p<.01.

Table 3

Multiple mediational analyses of the relationship between parenting style and depression symptoms with bootstrapped confidence intervals.

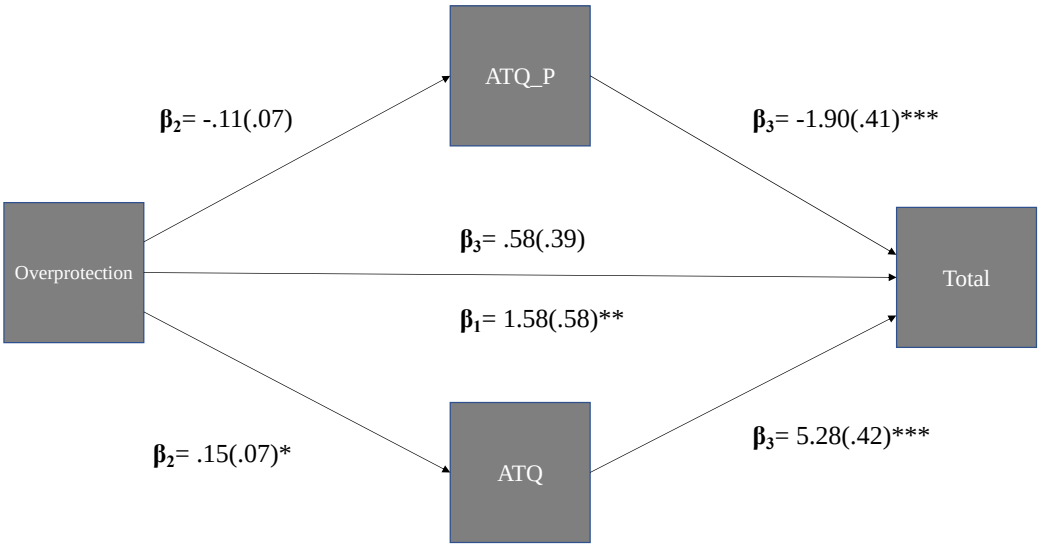
Outcome and Predictors	**Effect (SE)**		**Total Indirect Effect**		**Indirect Effect Through ATQ Positive**		**Indirect Effect Through ATQ Negative**	
BDI Total	**Total**	**Direct**	**LL**	**UL**	**LL**	**UL**	**LL**	**UL**
Overprotection	1.58(.58)*	.58(.39)	.19*	1.84*	-.05	.47	.11*	1.55*
Care	-2.18(.57)*	-1.10(.39)*	-2.06*	-.19*	-.74*	-.14*	-1.54	.10
BDI Cognitive-Affective	**Total**	**Direct**	**LL**	**UL**	**LL**	**UL**	**LL**	**UL**
Overprotection	.86(.45)*	.05(.29)	.15*	1.46*	-.05	.45	.08*	1.18*
Care	-1.64(.44)*	-.72(.29)*	-1.68*	-.22*	-.71*	-.16*	-1.12	.08
BDI Somatic	**Total**	**Direct**	**LL**	**UL**	**LL**	**UL**	**LL**	**UL**
Overprotection	.70(17)*	.54(.16)*	.02*	.34*	-.03	.06	.02*	.34*
Care	-.46(18)*	-.31(.16)	-.34	.02	-.09	.10	-.35	.02

Note. Tests were conducted with 95% confidence intervals (CI). CIs containing zero are not significant. LL: Lower Limit; UL: Upper Limit. *$p < .05$.

Figures

Figure 1

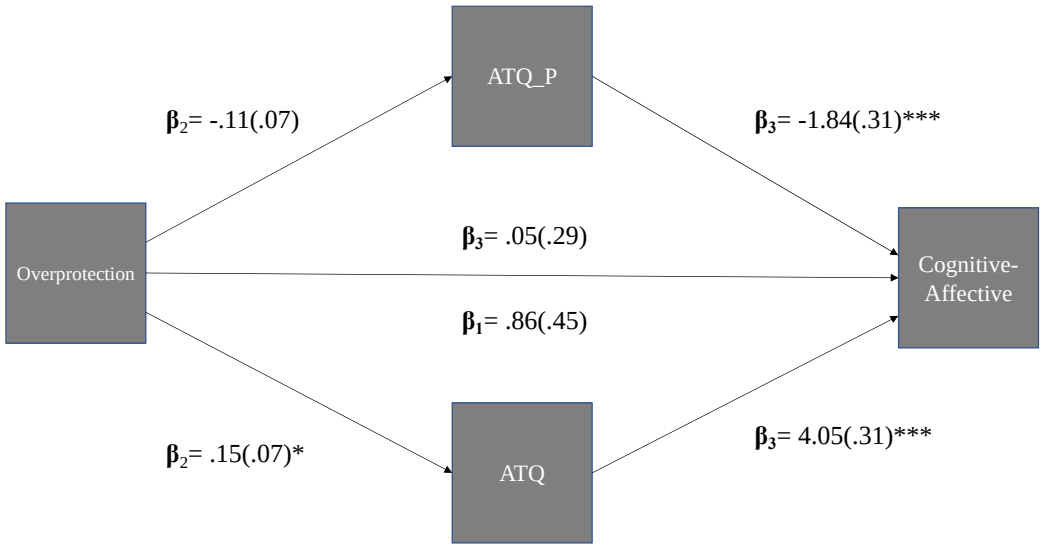
Multiple mediation model of total depression score regressed on automatic thoughts and overprotection parenting scores.



Note. ATQ_P = Positive Automatic thoughts, ATQ= Negative Automatic thoughts. Standard errors are presented in parentheses. *p< .05. ** p<.01. *** p<.001.

Figure 2

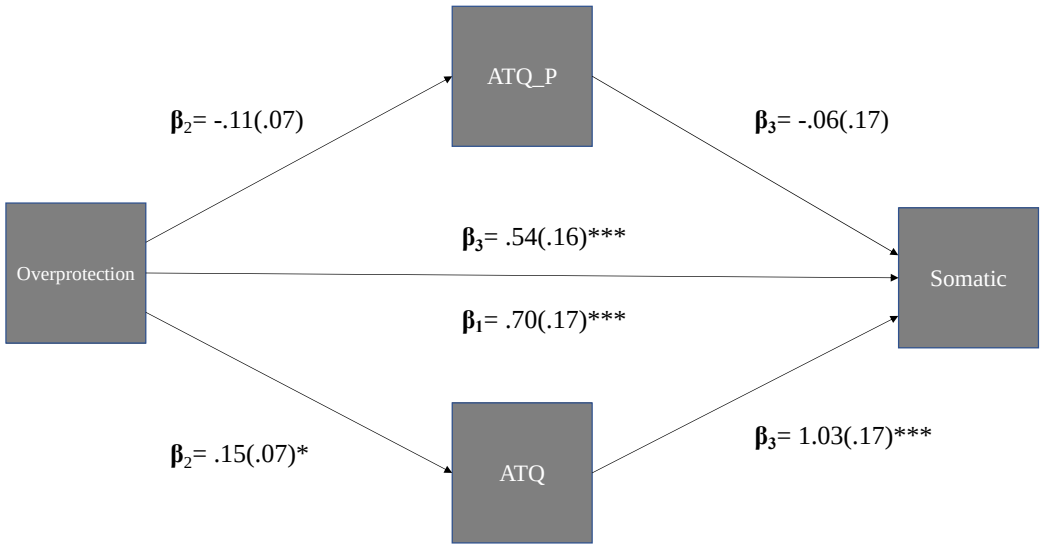
Multiple mediation model of cognitive-affective depression score regressed on automatic thoughts and overprotection parenting scores.



Note. ATQ_P = Positive Automatic thoughts, ATQ= Negative Automatic thoughts. Standard errors are presented in parentheses. *p< .05. ** p<.01. *** p<.001.

Figure 3

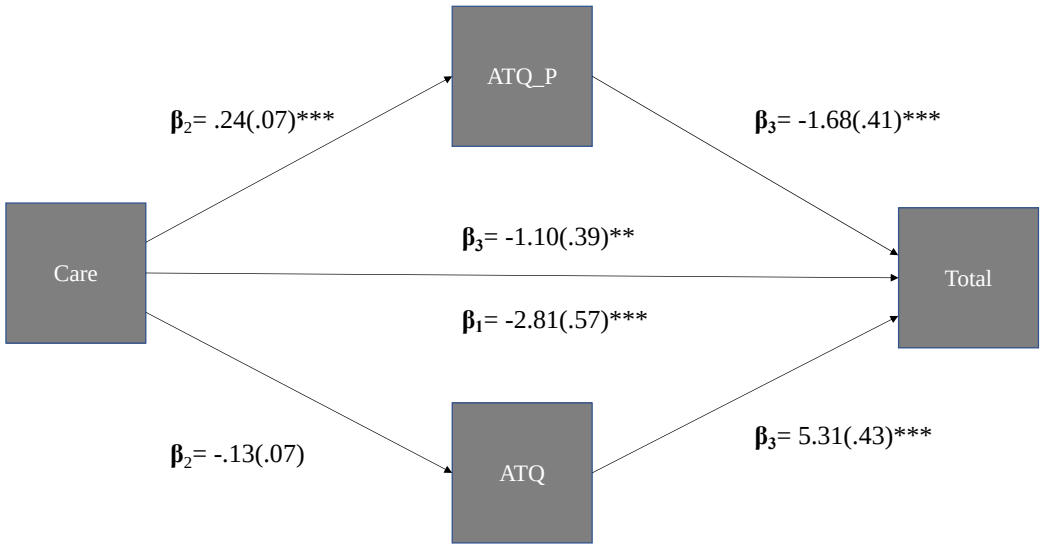
Multiple mediation model of somatic depression score regressed on automatic thoughts and overprotection parenting scores.



Note. ATQ_P = Positive Automatic thoughts, ATQ= Negative Automatic thoughts. Standard errors are presented in parentheses. *p< .05. ** p<.01. *** p<.001.

Figure 4

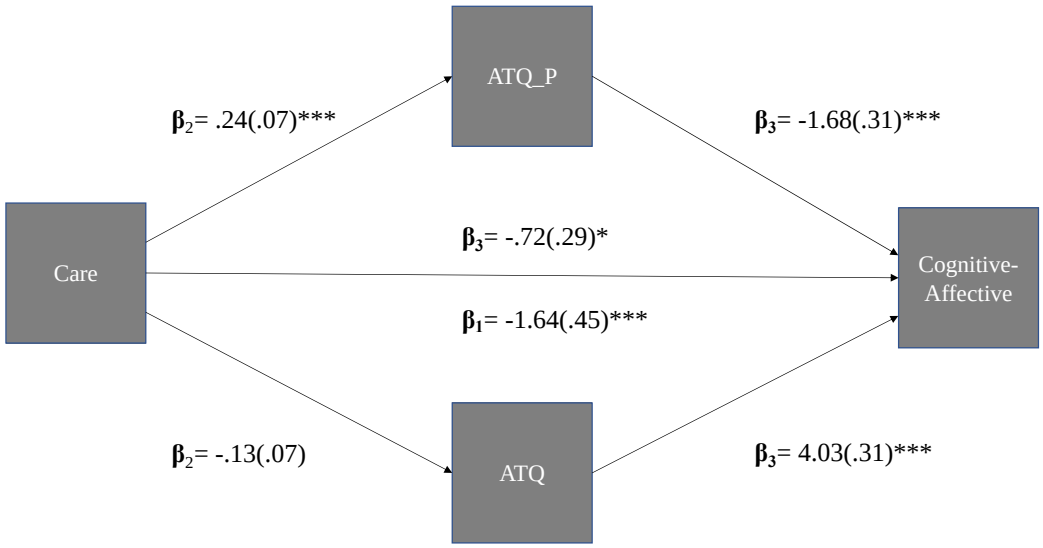
Multiple mediation model of total depression score regressed on automatic thoughts and care parenting scores.



Note. ATQ_P = Positive Automatic thoughts, ATQ= Negative Automatic thoughts. Standard errors are presented in parentheses. *p< .05. ** p<.01. *** p<.001.

Figure 5

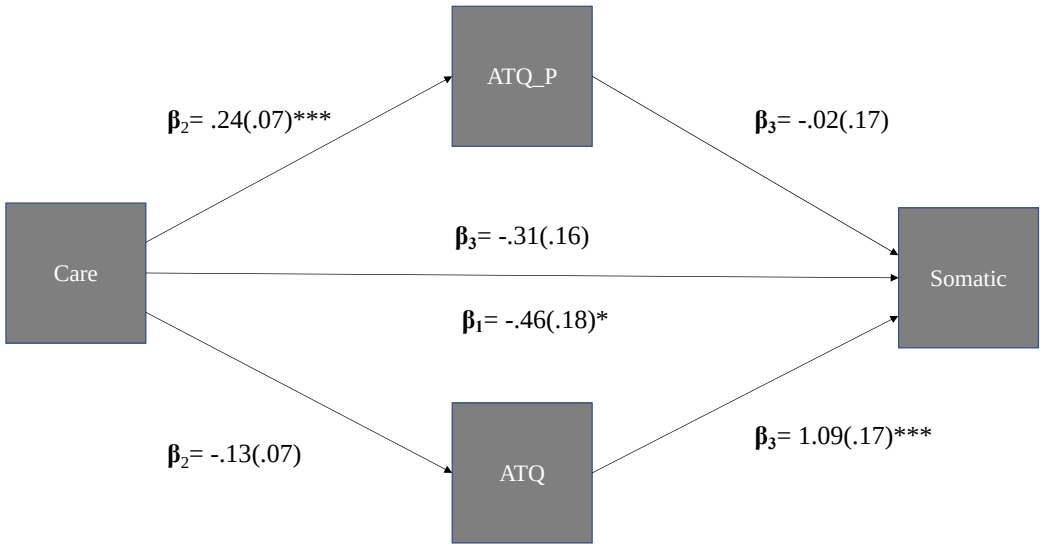
Multiple mediation model of cognitive-affective depression score regressed on automatic thoughts and care parenting scores.



Note. ATQ_P = Positive Automatic thoughts, ATQ= Negative Automatic thoughts. Standard errors are presented in parentheses. *p< .05. ** p<.01. *** p<.001.

Figure 6

Multiple mediation model of somatic depression score regressed on automatic thoughts and overprotection parenting scores.



Note. ATQ_P = Positive Automatic thoughts, ATQ= Negative Automatic thoughts. Standard errors are presented in parentheses. *p< .05. ** p<.01. *** p<.001.

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