Doctoral thesis submitted to
the Faculty of Behavioural and Cultural Studies
Heidelberg University
in partial fulfillment of the requirements of the degree of
Doctor of Philosophy (Dr. phil.)
in psychology

Title of the publication-based thesis

Adolescent reinforcement sensitivity as a longitudinal predictor of psychopathology: Investigating inhibitory control and emotion regulation as underlying mechanisms

presented by
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year of submission
2017

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Table of Contents

Acknowledgments

First, I would like to thank my supervisor Prof. Dr. Barnow for giving me the opportunity to follow my interest in research on emotion regulation under his supervision and to extend my knowledge in his research group. I learned a lot in these past four years and had the possibility to experience new challenges and extend my scientific knowledge and skills. I found the opportunity to expand my horizon by getting to know a new culture that contributed tremendously to the development of my emotional and intellectual self. All of this became possible because of his decision to supervise my doctoral project and I am very grateful for that.

I also would like to thank Prof. Dr. Koch who agreed to review this thesis. Furthermore, I thank all colleagues and cooperation partners that were involved in the Greifswald Family Study. Without their efforts the respective studies could not have been carried out.

I would like to extend my gratitude to my great colleagues and friends Helene Hausner, Julia Holl, Veronika Lerche, Andreas Neubauer, Maren Schumacher, Cornelia Schwarze, and Sebastian Wolff for their intellectual and emotional support within past years.

Special thanks go to my parents, Sergej, Elmar, and Beate whose love and support made me stronger during this journey.

Publications

The present dissertation is based on the following publications, which are referred to by their Arabic numerals in the Appendix.

1. Izadpanah, S., Schumacher, M., Bähr, A., Stopsack, M., Grabe, H. J., & Barnow, S. (2016). A 5-year longitudinal study of the adolescent reinforcement sensitivity as a risk factor for anxiety symptoms in adulthood: Investigating the indirect effect of cognitive emotion regulation. *Personality and Individual Differences, 95*, 68-73.

2. Izadpanah, S., Schumacher, M., & Barnow, S. (2017). Anger rumination mediates the relationship between reinforcement sensitivity and psychopathology: Results of a 5-year longitudinal study. *Personality and Individual Differences, 110*, 49-54

3. Izadpanah, S., Aldinger, M., Arens, E., Stopsack, M., I., U., Hansenne, M., Grabe, H. J., & Barnow, S. (2016). Adolescent harm avoidance as a longitudinal predictor of maladaptive cognitive emotion regulation in adulthood: The mediating role of inhibitory control. *Journal of Adolescence, 52,* 49-59.

Summary

Prevention and treatment of mental health problems are considered as two major aims of clinical psychological research. Designing prevention programs requires empirical studies that shed light on the risk factors underlying the development of psychopathology that need to be integrated as intervention targets. The current dissertation focuses on personality traits and emotion regulation (ER) as two key elements of theoretical models explaining the development of anxiety and depression. In the first part of this dissertation, the concepts of ER and reinforcement sensitivity are explained and the aims of the dissertation are further clarified. The first two studies investigate adolescent reinforcement sensitivity as a longitudinal risk factor for psychopathology symptoms. The differentiating effects of punishment and reward sensitivity on various psychopathology symptoms are highlighted. In order to understand the underlying mechanism of this link, the indirect effects of ER (Study 1) and anger rumination (Study 2) on this link are examined. Results show that punishment sensitivity leads to an increased use of maladaptive ER which in turn increases the risk for development of psychopathology.

The Study 3 takes one step back to examine inhibitory control as an underlying mechanism of the punishment sensitivity and ER link. The findings provide some support for the hypothesis that punishment sensitivity contributes to the habitual use of maladaptive ER through inducing attentional control deficit. In general, results suggest that punishment sensitivity and maladaptive ER constitute vulnerability for development of mental health problems. Therefore measurement of individual differences in reinforcement sensitivity and targeting ER among those with high levels of punishment sensitivity seems to be a promising pathway for reducing the risk for development of psychopathology. Further, our results suggest that targeting attentional control might have protective effects against the development of maladaptive ER strategies.

Finally, the findings are discussed and summarized and the implications and directions for future research are highlighted.

1. Introduction

1.1 The problem

Mental disorders haven been considered as a core health challenge of Europe in the 21st century with an estimate of over 38% prevalence of 27 major mental health diagnoses (Wittchen et al., 2011). It is estimated that at least one fourth of the adult population in Western countries meet criteria of at least one mental disorder during a 12 month time period (de Graaf, ten Have, van Gool, & van Dorsselaer, 2012; Kessler, Petukhova, Sampson, Zaslavsky, & Wittchen, 2012). Among these, anxiety disorders are the most prevalent disorders at a 12 months period (14%), followed by affective disorders (7.8%) with major depression being the most prevalent disorder (6.9%) (Wittchen et al., 2011). Studies conducted in Germany have also shown similar findings on anxiety disorders as the most frequent group of psychological disorders followed by unipolar depression (Jacobi et al., 2015). It is of note that German young adults have shown the highest prevalence of mental disorders compared to older adults and elderly in Germany (Jacobi et al., 2015). The transition from adolescence to adulthood is marked by an increase in overall rates of mental disorders such as anxiety and depression (Costello, Copeland, & Angold, 2011). However, there is limited knowledge about the adolescent risk factors that predict psychopathology symptoms in adulthood. The period between adolescence and adulthood is accompanied by various life stressor that are associated with a more intense emotional impact on individual compared to other developmental phases within the life span (Rutter, 2007). Therefore, knowledge on adolescent risk factors for psychopathology is crucial for prevention and intervention programs because they facilitate early identification of vulnerable adolescents and provide the opportunity for strengthening protective factors against psychopathological development during this stressful developmental stage.

Furthermore, the high degree of disability and costs associated with mental disorders highlights the significance of studies that elucidate longitudinal risk factors of psychopathology

and help identifying new potentials for prevention and treatment programs. However, despite a huge amount of studies on psychopathological development, there are still only limited studies that go beyond the correlational relationship between risk factors and psychopathology symptoms. Furthermore, although significant advances have been made in unraveling origins of anxiety and depression in the last decade, the field still lacks a sufficient understanding into the relevant risk factors and mechanisms underlying the development of depression and anxiety. Investigating these underlying mechanisms provides knowledge about potential constructs that play a mediating role in the development of these symptoms and contributes to prevention programs by introducing new targets for early prevention and treatment efforts.

1.2 The current dissertation

This dissertation aims at promoting knowledge about risk factors and mechanisms underlying the development of depression and anxiety symptoms in adulthood. Among various risk factors that have been studied in previous research, personality traits and emotion regulation (ER), are two key elements of theoretical models explaining the development of anxiety and depressive disorders from the developmental and personality research perspective (Bijttebier, Beck, Claes, & Vandereycken, 2009; Yap, Allen, & Sheeber, 2007). Furthermore, among various frameworks in personality research, the concept of reinforcement sensitivity has provided a great platform for studying the development of psychopathology (Corr, 2008). Therefore, standing on this platform, this thesis aims at analyzing adolescent reinforcement sensitivity as a longitudinal risk factor for development of psychopathology in adulthood and also investigating emotional dysregulation as an underlying mechanism of this link. In a further step, the longitudinal relationship between adolescent reinforcement sensitivity and adulthood emotional dysregulation is investigated and the indirect effect of inhibitory control on this relationship is examined. In the following section, I define the aforementioned constructs and discuss the most important findings relevant to the aims of this dissertation.

2 Conceptualizing the constructs

2.1 Emotion regulation

Thousands of new publications each year have made the emotion regulation one of the fastest growing areas within the field of psychology (Gross, 2013; p.3). In the most prominent model of ER, Gross (1998) defined ER as "the processes by which individuals influence which emotions they have, when they have them and how they experience and express these emotions". Based on the process model, individuals apply various ER strategies to influence their emotional experience and expression. ER is also defined as "extrinsic and intrinsic processes responsible for monitoring, evaluating, and modifying emotional reactions, especially their intensive and temporal features, to accomplish ones' goals"(Thompson, 1994). This definition illustrates that ER is a very broad concept encompassing various extrinsic and intrinsic aspects such as behavioral, biological, social, conscious and unconscious cognitive processes. Cognitive ER is one of the ER aspects that have shown to be a significant correlate of individuals' wellbeing (Aldao & Nolen-Hoeksema, 2010). Garnefski, Kraaij, and Spinhoven (2001) defined cognitive ER as conscious cognitive ways of managing the intake of emotionally arousing information. They introduced various cognitive ER strategies of *self-blame* ("thoughts of blaming yourself for what you have experienced"), *blaming others* ("thoughts of putting the blame of what you have experienced on others"), *rumination* ("thinking about the feelings and thoughts associated with the negative event"), *catastrophizing* ("thoughts of explicitly emphasizing the terror of an experience"), *acceptance* (thoughts of accepting what you have experience and resigning yourself to what has happened"), *refocus on planning* ("thinking about what steps to take and how to handle the negative event"), *positive refocusing* ("thinking about joyful and pleasant issues instead of thinking about the actual event"), *positive reappraisal* ("thoughts of attaching a positive meaning to the event in terms of personal growth"), and *putting into perspective* ("thoughts of minimizing the seriousness of the event or emphasizing its relativity when compared to other events"). The current dissertation focuses on these ER strategies and applies the suggestion of

Garnefski et al. (2001) about the classification of these nine strategies in two categories of "adaptive" (acceptance, refocus on planning, positive refocusing, positive reappraisal, putting into perspective) and "maladaptive" strategies (self-blame, blaming others, rumination, catastrophizing). The phrase "maladaptive" indicates that an ER strategy is either unsuccessful in reducing the unwanted emotional response or is associated with costs that outweigh any benefits of short-time reduction of acute emotions. In contrast, adaptive strategies facilitate (1) the reduction of subjective distress, physiological arousal, or dysfunctional behavior; and (2) maintaining abilities to pursue individuals' short- and long-term goals (Campbell-Sills, Ellard, & Barlow, 2014). Although this categorization can be influenced by factors associated with the context in which ER strategies are applied (Aldao & Nolen-Hoeksema, 2012a), past findings provide significant support for the validity of this categorization by showing different health outcomes for ER strategies, with the first category resulting in better health outcomes and with the second category being associated with an increased level of psychopathology symptoms (e.g., Aldao & Nolen-Hoeksema, 2010; Garnefski & Kraaij, 2006).

2.2 Reinforcement sensitivity

There are considerable individual differences in the way we live and experience our lives. Personality traits have a great influence on our emotional lives as they determine the quality and intensity of emotions that we experience to a great extent. An example of these individual differences is illustrated in the following two scenarios that describe reactions of two different persons to the same events:

First scenario: Martin wakes up stressed brooding over the last night; the things he talked about and the way he behaved at the party that he was invited to; wondering if he will be invited again or if he will be ignored the next time because he has not been talkative enough. Hearing the neighbor playing music so loud again early in the morning makes him frustrated. He clenches his teeth while thinking how reckless this neighbor is. However, he is not willing to bother himself or

cause any potential for hostility by disclosing his discomfort. He thinks with himself that this is not a good start for such a stressful and important day. He has an important appointment with his superior today to discuss the possibility of his promotion. He is uncertain and stressed about how the superior thinks about his potentials for this promotion. This would mean more responsibility and less free time. He remembers the conflict they had in the past and gets tensed about the possibility of escalating this conflict now that he has to work more intensively with his superior after the potential promotion.

Second scenario: Philipp remembers the conversation from the last night and becomes excited about knowing a couple of new people and the possibility to meet them again. The neighbor is playing the music so loud again. He seems to have forgotten about the last conversation they had about the noise. He decides to call him after breakfast to remind him how disturbing this loud music is so early in the morning. He thinks about the important meeting at work today at which the possibility of his promotion will be discussed. It makes him feel proud to think about the possibility of proving his capabilities for the new job. This would mean earning more money and having a great vacation in summer. He is optimistic about this chance. Despite the conflict with his superior, he is still thinking about giving him a promotion and it might mean that they have solved the conflict successfully.

Reading these two different scenarios highlights fundamental differences between these two individuals. In the first scenario, Martin experiences more negative emotions in response to the described situations. He shows brooding over the past, fearfulness of being punished or about having a bad performance, a passive reaction to the disturbance in the present moment, and fearing of the uncertain and negative consequences in the future, together with his pessimism about the chance he has been given. His behavior is more avoidance oriented rather than approach oriented. In the second scenario, Philipp experiences more positive than negative emotions. He shows an active reaction to discomfort rather than passively ruminating about it. Instead of focusing on the uncertainty and worries, he shows optimism and is excited about the chance of

improving his life after a promotion. This behavior represents a more approach oriented behavior. These two scenarios describe an essential concept in personality research which is called reinforcement sensitivity. Reinforcement sensitivity was proposed by Gray (1982) and refers to individuals' variations in the sensitivity of basic brain and behavioral systems that respond to punishing and rewarding stimuli. Reinforcement sensitivity has become a common mechanism among a family of personality theories that deal with approach and avoidance processes. It is probably due to the centrality of the reinforcement sensitivity in personality research that the theory put forward by Gray, which has had the most prominent influence on this area, has been named the reinforcement sensitivity theory (for a detailed review of this theory and its impact on personality psychology see Corr, 2008). In the current dissertation, I approach the concepts of punishment and reward sensitivity using the theoretical framework of the reinforcement sensitivity theory (in Studies 1 and 2) and the psychobiological model of temperament and character (in Study 3) (Cloninger, Svrakic, & Przybeck, 1993). In the following section, I provide an introduction to both these theories and their relevant concepts for the current dissertation.

2.2.1 The psychobiological model of temperament and character

Cloninger (1987) developed a psychobiological model of personality and proposed four temperament dimensions (harm avoidance, novelty seeking, reward dependence, persistence) and three character dimensions (self-directedness, cooperativeness, self-transcendence). Cloninger et al. (1993) hypothesized temperament systems in the brain as functionally organized and independently varying systems for the activation, maintenance, and inhibition of behavior in response to certain categories of stimuli. Based on his model, *"behavioral activation*" was involved in the activation of behavior in response to those stimuli related to novelty, signals of reward, or removal of punishment, while "*behavioral inhibition*" occurred in response to signals of punishment or non-reward. In his model, individual differences in behavioral activation and behavioral inhibition were called novelty seeking and harm avoidance, respectively. The concept of harm avoidance in this model has been the focus of Study 3. It is viewed as a heritable bias

towards the inhibition of behavior, such as pessimistic worry in anticipation of future problems, fear of uncertainty and shyness of strangers, and rapid fatigability. Harm avoidance is also related to avoidance processes and is characterized by individuals' tendency to respond intensely to aversive stimuli and to avoid punishment, novelty, and non-reward passively (Cloninger et al., 1993).

2.2.2 The reinforcement sensitivity theory

The reinforcement sensitivity theory (Gray & McNaughton, 2003) is a biologically based model consisting of three major neuropsychological systems that underlie personality, namely, the Behavioral Approach System (BAS), Behavioral Inhibition System (BIS) and Fight-Flight-Freeze System (FFFS). The BAS is activated by all appetitive or reward related stimuli and the termination of signals of punishment. It is related to anticipatory pleasure, optimism, rewards-orientation and impulsiveness and underlies externalizing symptoms such as impulsive and high-risk behavior. The FFFS is activated by unconditioned (e.g., pain, innate fear) and conditioned aversive stimuli (e.g., environmental cues that signal pain). FFFS is related to personality factors of fear-proneness and avoidance, which clinically underlies disorders such as phobia and panic (Corr & McNaughton, 2008).

The BIS underlies the "watch out for danger" emotion of anxiety and following activation produces outputs of behavioral inhibition, increased arousal, heightened attention and information processing, and the emotion of anxiety. BIS is hypothesized to facilitate the resolution of goal conflict which means that BIS is activated by simultaneous activation of BAS and FFFS (approach-avoidance conflict) (Corr, 2008). BIS becomes increasingly activated as resolving the decision between approach-avoidance becomes more difficult (i.e., as the relative strength of approach and avoidance becomes more equal). In order to resolve the conflict, BIS amplifies the activity of aversive system but not appetitive one by increasing the valence of negative stimuli, risk aversion and facilitating avoidance. This process leads to the experience of worry and

rumination. Based on this theory, high level of BIS activation leads to risk aversion and underlies clinical conditions such as generalized anxiety disorder. The recent reconceptualization of the reinforcement sensitivity theory has made a clearer distinction between FFFS and BIS suggesting that they are responsible for emotions of fear and anxiety, respectively (for a detailed review see Corr, 2008). Given that many questionnaires for measurement of reinforcement sensitivity theory are still based on the original version of this theory, they assess combined BIS–FFFS sensitivity within the revised version of the reinforcement sensitivity theory. In the same vein, in the current dissertation, Action Regulation Emotion Systems (Hartig & Moosbrugger, 2003) has been used that measures combined BIS/FFFS. However, the construct of interest in this dissertation is punishment sensitivity, which based on the reinforcement sensitivity theory is increased following the activation of both BIS and FFFS (see Figure 1). Therefore, throughout this dissertation, when using BIS we refer to the combined BIS–FFFS sensitivity.

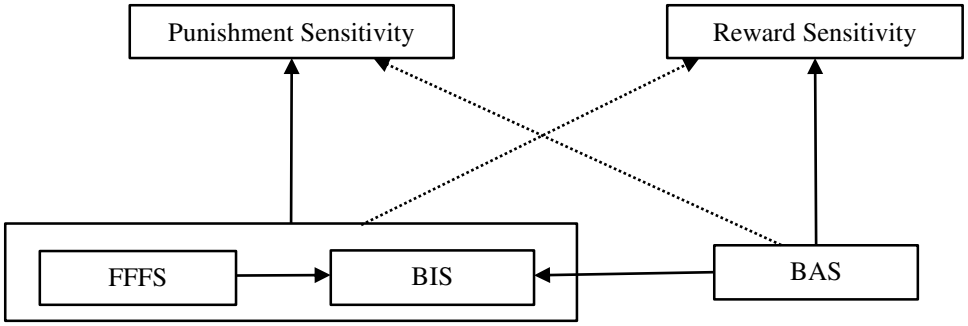

Figure 1. Schematic representation of the hypothesized relationship between FFFS/BIS (punishment sensitivity) and BAS (reward sensitivity) illustrating the facilitatory (unbroken line) and inhibitory effects (broken line) (Corr, 2008).
Fight-Flight-Freeze System (FFFS), Behavioral Inhibition System (BIS), Behavioral Activation System (BAS).

The BIS/BAS as independent or joint subsystems*:* There are two different hypotheses about the interplay between BIS and BAS. *The separable subsystems hypothesis* assumes that BIS and BAS are orthogonal, which means responses to reward should be the same at all levels of BIS and responses to punishment should be the same at all levels of BAS (Corr, 2002). *The joint subsystems hypothesis* postulates that BIS and BAS have the potential to influence both reward-mediated and punishment-mediated behavior (Corr, 2002). It means that in the case of

punishment-mediated behavior, high BIS facilitates and high BAS antagonizes; and in the case of reward-mediated behavior, high BAS facilitates and high BIS antagonizes. The output behavior is determined by the interplay between BIS and BAS. For example, in the context of psychopathological development, the joint subsystems hypothesis assumes that those individuals with high BIS and low BAS should be at a higher risk for development of internalizing psychopathology because based on this hypothesis low reward sensitivity cannot implement a strong antagonistic effect or suppress the effect of punishment sensitivity. In other words, BIS increases the risk for anxiety without being hampered by the antagonistic effect of BAS. Figure 1 illustrates the hypothesized antagonistic and facilitatory effects of BIS/BAS on reward and punishment mediated behavior.

3 Clarifying the questions

3.1 Emotion regulation and psychopathology

Difficulties in selection and implementation of functional ER strategies have become a central concept in explaining the etiology of psychopathology symptoms (Sheppes, Suri, & Gross, 2015). Emotion dysregulation appears to be so central in anxiety and depression (Barnow, 2012; Barnow, Aldinger, Ulrich, & Stopsack, 2013; J. Cisler, Olatunji, Feldner, & Forsyth, 2010) that the dysregulated emotional state is considered as one of the characteristics in the definition of these disorders (American Psychiatric Association, 2013). Emotional dysregulation has been related to depression and anxiety among groups of children, adolescents and adults (Garnefski, Kraaij, & van Etten, 2005; Maack, Tull, & Gratz, 2012; Suveg, Hoffman, Zeman, & Thomassin, 2009). Difficulties in down regulation of negative emotions seem to be particularly important in the context of depression and anxiety disorders. This is partly due to typical presentations of anxiety disorders and depression, which are largely characterized by excessive negative emotions such as sadness, fear and anxiety. Applying maladaptive ER strategies can lead to an unsuccessful down-regulation of negative emotions or might even contribute to the escalation or maintenance

of these emotions which increase the risk for development of psychopathology (Suveg, Morelen, Brewer, & Thomassin, 2010).

A current area of particular interest concerns investigating various ER strategies in relation to development of anxiety and depression symptoms (Campbell-Sills et al., 2014). Recent studies have shown that the ability to terminate an ineffective regulation strategy and generate and implement an alternative and effective strategy is related to various forms of psychopathology including depression, anxiety, and general distress (Kato, 2012). A useful example is rumination, which is an effort to make sense of negative events that individuals have experienced. However, an excessive use of this strategy and failure in stopping rumination, results in persistent rumination, which is a core feature in depression and anxiety disorders (Grafton & MacLeod, 2013; McLaughlin & Nolen-Hoeksema, 2011). Similar to rumination, other cognitive ER strategies have shown to be risk factors for psychopathology. For example, Garnefski, Boon, and Kraaij (2003) showed that among adolescents who experienced a stressful life event, strategies of self-blame, catastrophizing, and rumination were associated with higher depression scores, regardless of the type of the life events involved. In another study, self-blame, catastrophizing, and positive reappraisal distinguished the clinical sample with emotional disorders from the non-clinical (Garnefski et al., 2002). Although overreliance on maladaptive ER strategies conveys a higher risk of psychopathological development, the habitual use of adaptive strategies does not seem to impart resilience to psychopathology. The only longitudinal study on this link was conducted by Aldao and Nolen-Hoeksema (2012b) and showed that self-report use of adaptive strategies (cognitive reappraisal and acceptance) did not significantly predict psychopathology symptoms one year later. This finding converges with the finding from cross-sectional studies showing that maladaptive strategies have stronger associations with depression and anxiety than adaptive strategies (Aldao & Nolen-Hoeksema, 2010; Aldao, Nolen-Hoeksema, & Schweizer, 2010). It has been argued that the detrimental effects of maladaptive ER are less context-dependent than the beneficial effects of adaptive strategies (Aldao & Nolen-Hoeksema, 2012a).

In line with previous findings that support a stronger link between maladaptive ER and anxiety and depression compared to adaptive ER, in the current dissertation we have put a larger focus on the first category of ER, namely maladaptive ER. In Study 1, we investigate the differentiating effect of both adaptive and maladaptive ER on anxiety symptoms. Furthermore, in line with previous studies that emphasize taking an emotion-specific approach into studying the relationship between ER and psychopathology (Brenning & Braet, 2013), an emotion-specific and symptom-specific approach has been taken in Study 2 to investigate the effect of anger rumination on symptoms of depression, anxiety and aggression (path b in Figure 2). While past work on ER has focused on regulation across emotions, investigating specific emotions (e.g., anger) is necessary to determine if the association between ER and psychopathology is the same across various emotions and various symptoms (Folk, Zeman, Poon, & Dallaire, 2014). The importance of anger regulation becomes more salient at late adolescence and young adulthood given that individuals experience higher levels of anger during this developmental phase (Schieman, 1999)

As I mentioned above, previous findings provide valuable evidence supporting the link between ER and depression and anxiety. However, these findings portray an incomplete picture as they do not explain how emotional dysregulation develops in the first place. To provide a more comprehensive picture of psychopathological development, we take one step back to study development of cognitive ER in adulthood from individual differences in adolescent reinforcement sensitivity (Path a in Figure 2).

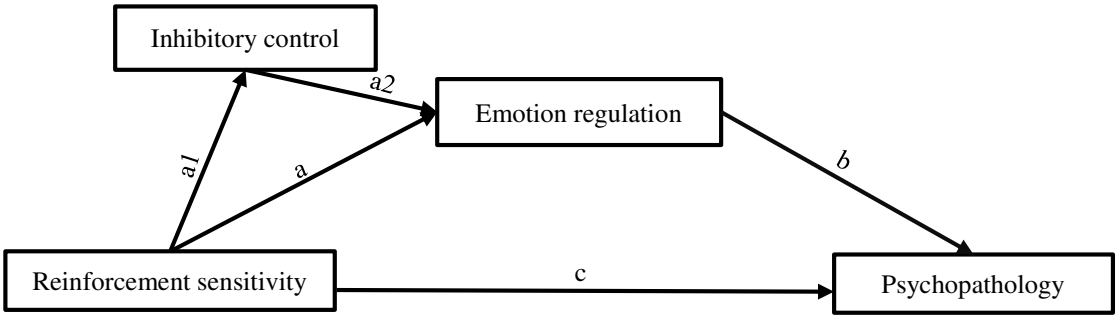

Fig. 2. Schematic representation of hypothesized pathways across three studies. Paths a, b, and c are investigated in Studies 1 and 2. Paths a, a1, and a2 are investigated in Study 3.

3.2 Reinforcement sensitivity and emotion regulation

The two scenarios that were described earlier portrayed how individual differences in reinforcement sensitivity affect the levels of experienced positive and negative emotions in daily life. Punishment sensitivity has been associated with higher levels of negative emotions such as irritability, sadness, and uncertainty and magnified reactions towards negative events, while reward sensitivity has shown to be related to higher levels of positive emotions such as happiness, confidence, excitement, and enthusiasm (Gable, Reis, & Elliot, 2000; Hundt, Brown, et al., 2013). The experience of negative emotions related to punishment sensitivity and higher levels of emotional arousal requires more regulatory effort and can lead to emotional dysregulation (Fox, Henderson, Marshall, Nichols, & Ghera, 2005). Similarly, it has been argued that high levels of child temperamental reactivity contributes to emotional dysregulation because it impedes the use of helpful ER strategies (Suveg et al., 2009). This assumption is supported by the studies that show punishment sensitivity is related to more difficulties in ER (e.g., Schreiber, Grant, & Odlaug, 2012; Suveg et al., 2010). On the other hand, previous studies have also found a significant yet small relationship between reward sensitivity and less ER difficulties (Hannan & Orcutt, 2013; Tull, Gratz, Latzman, Kimbrel, & Lejuez, 2010). For example, Markarian, Pickett, Deveson, and Kanona (2013) in a study on 459 undergraduate students found that BIS is related to more difficulties in ER, while there was a smaller negative link between BAS and ER difficulties. Similarly, Schreiber et al. (2012) used Difficulties in Emotion Regulation Scale to categorize their participants in two groups of high and low emotional dysregulation. They found that harm avoidance was related to more ER difficulties, while reward dependence showed a smaller but negative association with ER difficulties. In another study, Tortella-Feliu, Balle, and Sesé (2010) investigated a large sample of adolescents and provided support for punishment sensitivity as a risk factor for applying maladaptive cognitive ER strategies, as measured by cognitive Emotion Regulation Questionnaire (CERQ). These findings support punishment sensitivity and reward

sensitivity as possible risk and protective factors for ER, respectively, with punishment sensitivity seemingly having more predictive strength than reward sensitivity.

However, previous studies have several limitations that need to be acknowledged. First, to our knowledge, the existing literature on the link between reinforcement sensitivity and ER has mainly focused on ER difficulties, while particular cognitive ER strategies are associated with psychopathology (Garnefski et al., 2005). When emotional problems do arise from emotion dysregulation, it is essential to specify precisely what type of emotion dysregulation might be in operation. Given that individuals apply a repertoire of ER strategies that have shown distinct influence on the individual's mental health (Aldao & Nolen-Hoeksema, 2010), providing findings on the specific ER strategies is essential as it allows researchers to identify what ER strategies are most strongly related to punishment and reward sensitivity. Only two studies, to our knowledge, investigated the link between punishment sensitivity and specific ER (Leen-Feldner, Zvolensky, Feldner, & Lejuez, 2004; Manfredi et al., 2011). However, these studies have been limited to the cognitive ER strategy rumination. In a study on a sample of adults, Manfredi et al. (2011) showed that punishment sensitivity (i.e., harm avoidance) is related to a tendency to ruminate. Authors argue that punishment sensitivity seems to be correlated with a passive form of mental problem solving rather than active problem solving that leads to a ruminative response to emotional experiences (Manfredi et al., 2011). Similarly, in another study, Leen-Feldner et al. (2004) showed that BIS is associated with a ruminative ER style. Although these two studies provide evidence for the link between BIS and dysfunctional ER strategy rumination, the link between punishment/reward sensitivity and other well-known cognitive ER strategies remains unknown.

Second, past studies have used cross-sectional data and cannot answer the question whether reinforcement sensitivity measured in adolescence will be still significantly correlated with ER measured after a long interval. The third limitation is that previous studies have not investigated how the interaction between punishment and reward sensitivity is related to ER. The joint subsystems hypothesis proposed by Corr (2002) assumes that BIS and BAS have the

potential to influence both reward-mediated and punishment-mediated responses, while based on the separable subsystems hypothesis "individual differences in the functional capacity of one system are independent of the individual differences in the functional capacity of the other system" (Pickering, 1997). It still remains a question which hypothesis is applicable to the link between BIS/BAS and ER. Significant main effects for both BIS and BAS or statistically significant BIS/BAS interaction will support the assumption of joint subsystems (Corr, 2002).

In search of the underlying mechanism: Inhibitory control

As we discussed in previous section, punishment sensitivity seems to be associated with difficulties in ER and a maladaptive style of regulating emotions such as rumination. Looking into previous literature does not answer the question how punishment sensitivity leads to emotional dysregulation. This is an interesting question because knowing the underlying mechanism of this link helps us to recognize the indirect pathways that can be targeted in preventive programs. One possible mechanism might rely on the implications of inhibitory control. Inhibitory control is defined as the ability to suppress inappropriate responses or attention tendencies, in order to act properly in the task at hand (Dempster, 1992; Nigg, 2000). Punishment sensitivity has been associated with an automated pattern of attending to neutral and emotional stimuli (Hansenne et al., 2003) and a strong attentional bias towards emotional negative stimuli (Cloninger, 1994b; Zhang et al., 2013), both of which might impair suppressing irrelevant information and facilitate the interference of negative emotional information leading to inhibitory control deficits (Matthews & Deary, 2000a; Weierich, Treat, & Hollingworth, 2008). This interference of irrelevant negative information that is accompanied by inhibitory control deficit can fuel maladaptive cognitive ER strategies by facilitating the increased access to intrusive cognitions. Previous studies provide support for this assumption showing that deficits in inhibiting neutral and emotionally negative information convey a risk for increased rumination (De Lissnyder et al., 2012; Joormann & Gotlib, 2010; Zetsche, D'Avanzato, & Joormann, 2012). Although inhibitory control has been mainly investigated in relation to rumination, all cognitive maladaptive strategies share a

cognitive nature that consists of recurrent dysfunctional thoughts (Garnefski et al., 2001) that can be influenced by the degree of inhibitory control. Therefore, it seems necessary to investigate how inhibitory control might be related to other maladaptive cognitive ER strategies. In Study 3, we answer the question whether individuals with punishment sensitivity might be prone to development of maladaptive cognitive ER through impaired inhibitory control.

3.3 Reinforcement sensitivity and psychopathology

We discussed earlier how reinforcement sensitivity is important in the context of ER. Another line of research supports the significance of reinforcement sensitivity for psychopathology research. The reinforcement sensitivity theory assumes that BIS underlies internalizing disorder, whereas elevated BAS has been assumed to make individuals more prone to externalizing problems (Gray, 1994). This assumption that depression and anxiety are associated with high BIS is supported by previous studies, while findings on BAS are less consistent (see Bijttebier et al., 2009). Some studies show that low BAS is associated with depression (e.g., Hundt, Williams, Mendelson, & Nelson-Gray, 2013; Kimbrel, Nelson-Gray, & Mitchell, 2007), while other studies did not find such an association (S. L. Johnson, Turner, & Iwata, 2003; Jorm et al., 1998; Muris, Meesters, de Kanter, & Timmerman, 2005). For example, in the study of Hundt, Williams, et al. (2013) on young adults, BIS predicted depression, anxiety, and worry symptoms, while BAS predicted alcohol and drug use directly and depression only indirectly through less problem-focused coping. On the other hand, S. L. Johnson et al. (2003) in an epidemiological study did not find a significant relation between BAS and depression. Based on previous literature, it seems that BIS but not BAS predicts anxiety and depression symptoms (S. L. Johnson et al., 2003), while externalizing symptoms such as aggression are related to a dominance of BAS over BIS (Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008; Quay, 1993). Nevertheless, this line of research still needs more evidence to clarify the link between BIS/BAS and internalizing/externalizing symptoms. It is particularly important because no study, to our knowledge, has answered the question whether BIS/BAS predict psychopathology symptoms

longitudinally. It is possible that the negative correlation between BAS and depression in some studies (e.g., Kimbrel et al., 2007) merely represent the shared characteristics between low BAS and depression such as lower levels of positive experiences and expectancies, rather than suggesting BAS as a risk factor (Beevers & Meyer, 2002). Therefore, in the Studies 1 and 2, we go beyond this cross-sectional view and investigate BIS/BAS as longitudinal predictors of depression, anxiety and aggression and explore the differentiating effect of BIS/BAS in predicting these symptoms. Furthermore, we investigate the joint and separated Subsystems hypotheses in prediction of these various symptoms. According to Corr (2002), the joint subsystems hypothesis does not necessarily require the BIS/BAS interaction to be significant, but rather two main effects would be sufficient to support this hypothesis. Very few studies have investigated these two hypotheses in relation to psychopathology symptoms. For example, Kimbrel et al. (2007) found no significant BIS/BAS interaction effect in predicting anxiety and depression. However, they provided limited support for the joint subsystems hypothesis showing that both, high BIS and low BAS predict anhedonic depression, but only BIS predicted mixed depression/anxiety symptoms. Similarly, Hundt, Nelson-Gray, Kimbrel, Mitchell, and Kwapil (2007) found that both low BAS and high BIS predicts anhedonic depression, but only BIS predict mixed depression/anxiety symptoms. Furthermore, they found that the interaction between high BIS and high BAS also predicts mixed depression/anxiety symptoms. On the other hand, S. L. Johnson et al. (2003) in an epidemiological study with a large community sample found support for the BIS model of depression/anxiety and showed that BAS was significantly related to drug abuse, but unrelated to depression. However, they did not investigate the effect of BIS/BAS interaction. Therefore, previous findings do not provide sufficient and consistent information on these two hypotheses, which emphasizes the necessity of further investigations.

In search of the underlying mechanism: Emotion regulation

It takes two to tango! As we discussed in the previous section, punishment sensitivity increases the risk for internalizing psychopathologies. An interesting question to ask is that how

individual differences in punishment sensitivity lead to the development of psychopathology. In line with the above mentioned proverb, development of psychopathology is also influenced by the interplay of multiple factors. Although studies show that punishment sensitivity leads to the development of depression and anxiety, it has been suggested that this link might be assisted by the mediating effect of emotional dysregulation (Suveg et al., 2010). In other words, punishment sensitivity might contribute to development of depression/anxiety through increasing the habitual use of maladaptive ER. Investigating this question is important, given that a critical issue for further research is the investigation of the mechanisms and the processes through which reinforcement sensitivity differences translate into vulnerability to psychopathology (Bijttebier et al., 2009). Current theories of vulnerability highlight the importance of self-regulatory processes enabling individuals to modulate their reactions and reduce the risk associated with their temperamental reactivity (Lonigan, Vasey, Phillips, & Hazen, 2004; Nigg, 2006). In line with these theories, two previous studies have shown that ER difficulties mediate the link between punishment sensitivity and anxiety (Markarian et al., 2013; Suveg et al., 2010). Furthermore, only one study so far provided evidence for an indirect effect of maladaptive cognitive ER on punishment sensitivity and anxiety (Tortella-Feliu et al., 2010). However, this study did not include adaptive cognitive ER and reward sensitivity in their model. In Study 1, we include BIS/BAS and investigate the indirect effects of both adaptive and maladaptive cognitive ER in prediction of anxiety symptoms in order to test the relative strength of each category of ER strategies and to examine the differentiating effects of BIS and BAS on ER and anxiety symptoms. Furthermore, to the extent of our knowledge, no study answers the question whether reinforcement sensitivity leads to psychopathology symptoms through increasing the vulnerability for dysregulation of specific emotions such as anger. Anger is important in this context, given that BIS/BAS have shown to be associated with elevated levels of anger (Harmon-Jones, 2003; Smits & Kuppens, 2005). It seems necessary to investigate whether dysregulation of this emotion might underlie development of psychopathology among those with BIS and BAS sensitivity. In Study 3,

we narrow our focus to answer this question and to investigate the indirect effect of anger rumination on the link between BIS/BAS and depression, anxiety, and aggression symptoms.

3.4 Procedure and aims

Samples that were included in this dissertation were drawn from the population-based Greifswald family study (Aldinger et al., 2014; Barnow, Schuckit, Lucht, John, & Freyberger, 2002; Barnow, Stopsack, & Ulrich, 2010), a subpopulation of the Study of Health in Pomerania (SHIP; John et al., 2001). In SHIP, 4308 people aged 20 to 79 were randomly selected between March 1997 and May 2000, proportional to the population size of each community. From this sample, 527 families with at least one offspring between the ages of 11 and 18 years were invited to participate in the family study. 141 families could not be accessed and 71 families refused to participate, resulting in 315 families with 381 offspring (mean age = 15.1, *SD* = 2.3) who participated in the baseline assessment (T_0). The first follow up (T_1), took place five years later between 2005 and 2008 (mean interval$_{T1\text{-}T0}$ = 53.18 months, *SD*$_{T1\text{-}T0}$ = 12.97) and included 87.7% of the offspring from the first assessment (*n* = 334, mean age = 19.6, *SD* = 2.4). From May 2011 to April 2014 (T_2) they were investigated a third time (mean interval$_{T2\text{-}T1}$ = 65.63 months, *SD*$_{T2\text{-}T1}$ = 8.14) and, from this assessment, data for 85% of the T_1 offspring participants are available (*n* = 284, mean age = 25, *SD* = 2.41). Those who participated in all three assessments did not differ from individuals who dropped out after T_0 concerning gender (χ^2 = 2.37, *p* = .146), and age (*F* = 2.05, *p* = .153). The studies in the current dissertation concern the data from the offspring.

The Greifswald family study targeted the life span between adolescence and adulthood. There are various reasons why adolescence is such an important time of risk (and therefore important for prevention) for the development of psychopathology. First, during adolescence there is a fast increase in emotional arousability, novelty seeking, and motivation for peer acceptance, while self-regulation capacities are still immature and their development is slower and more gradual (Steinberg, 2005). This developmentally normative mismatch between strong affective

and behavioral impulses and the adolescents' still limited capacity to regulate them is accompanied by an increase in the amount of unsupervised time (Yap et al., 2007). These features of adolescence suggest that this period is associated with a heightened vulnerability to problems associated with poor regulation of affect and behavior. The longitudinal nature of the Greifswald family study provided the possibility to examine our research questions with a developmental perspective. Therefore, with the central aim of studying the risk factors and underlying mechanisms of development of psychopathology symptoms, the current dissertation pursues the following aims:

a) Investigating the differentiating effect of adolescent punishment/reward sensitivity on the development of cognitive ER (path a in Figure 2) and depression, anxiety, and aggression symptoms (path b in Figure 2) in young adulthood.
b) Examining cognitive ER as an underlying mechanism through which reinforcement sensitivity leads to the development of depression and anxiety symptoms (Path a, b, c in Figure 2)
c) Understanding the underlying mechanism of the link between punishment sensitivity and maladaptive cognitive ER by investigating the indirect effect of inhibitory control (path a, a1, a2 in Figure 2).

In the Greifswald family study, reinforcement sensitivity is measured at T_0 using Temperament and Character Inventory (Cloninger, 1994a) and at T_1 using Action Regulating Emotion System for measurement of BIS/BAS (Hartig & Moosbrugger, 2003). We also applied data for psychopathology symptoms measured at T_1 and about 5 years later at T_2 using the Symptom Checklist SCL-90-R (Franke, 1995) and its short version, the Brief Symptom Inventory (Franke, 2000). Cognitive ER was measured at T_2 using the Cognitive Emotion Regulation Questionnaire (Garnefski & Kraaij, 2007). Furthermore, a multimethod assessment of inhibitory control was conducted at T_1 using two well-known experimental tasks of emotional Stroop and stop-signal task (Khng & Lee, 2014) as measures for state-dependent inhibitory control, together

with Barrat Impulsiveness Scale (Patton, Stanford, & Barratt, 1995) as a measure for trait-dependent inhibitory control. In the first two studies, the longitudinal effect of punishment/reward sensitivity on psychopathology symptoms and the indirect effect of cognitive ER on this link are examined. The first study focuses on anxiety symptoms and maladaptive cognitive ER, while the second study complements the first study by providing symptom-specific results through examining the differential effects of punishment/reward sensitivity (T1) on various symptoms of depression, aggression, and anxiety. We also narrowed the focus to provide emotion-specific results by examining the indirect effect of anger rumination (as a specific regulation strategy towards the specific emotion of anger) on the link between reinforcement sensitivity and psychopathology symptoms. In Study 3 we took one step back to investigate adolescent punishment sensitivity (T_0) as a longitudinal risk factor for maladaptive ER in young adulthood (T_2). We further explored the underlying mechanism of this link by examining the role of inhibitory control as a possible mediator.

4 Reinforcement sensitivity and development of psychopathology: Emotion regulation as an underlying mechanism

4.1 Reinforcement sensitivity and anxiety symptoms: The indirect effect of cognitive emotion regulation: Study 1

As mentioned above, the main aim of Study 1 was to examine the longitudinal direct effect of punishment and reward sensitivity (BIS/BAS) on development of anxiety symptoms and to test the indirect effect of cognitive ER on this link. Reinforcement sensitivity theory assumes that BIS and not BAS underlies the development of anxiety disorders (Corr, 2008; Gray, 1982). In line with this assumption, studies with community samples and clinical samples have shown that anxiety symptoms are positively associated with BIS and insignificantly associated with BAS (Campbell-Sills, Liverant, & Brown, 2004; S. L. Johnson et al., 2003; Jorm et al., 1998; Kimbrel et al., 2007; Muris, Merckelbach, Schmidt, Gadet, & Bogie, 2001). It seems that the influence of

punishment sensitivity outweighs the effect of reward sensitivity on development of anxiety disorders. Although theoretical background and previous findings suggest that BIS (not BAS) is related to anxiety symptoms, there are several limitations to previous findings that need to be taken into account. First, past studies have been mainly based on cross-sectional data on adults and cannot answer the question whether punishment sensitivity in younger ages is a longitudinal risk factor for development of anxiety in adulthood. Second, a critical issue concerns investigating the processes or mechanisms through which individual differences in BIS/BAS sensitivity lead to specific disorders (Bijttebier et al., 2009). Not all individuals with high level of punishment sensitivity develop anxiety disorders and this suggests the existence of potential variables with moderating or mediating effect (White, McDermott, Degnan, Henderson, & Fox, 2011). Past studies suggest ER as one possible mediator of the link between punishment sensitivity and anxiety symptoms (Markarian et al., 2013). However, very limited studies have investigated the indirect effect of ER on the relationship between reinforcement sensitivity and anxiety (Tortella-Feliu et al., 2010). Third, limited studies have investigated the effect of BIS/BAS interaction on anxiety symptoms (Hundt et al., 2007). Based on the separate subsystem hypothesis, no BIS/BAS interaction is expected as it assumes that individual differences in the functional capacity of one system are independent of the individual differences in the functional capacity of the other system. On the other hand, based on the joint subsystems hypothesis, BIS/BAS have the potential to influence both reward-mediated and punishment-mediated response (Corr, 2002). It states that BIS/BAS are functionally interdependent and each has an antagonistic effect on the other. Thus, low approach is assumed to exacerbate the effect of high BIS on anxiety symptoms. This hypothesis does not necessarily need interaction, but two significant main effects of BIS/BAS would be sufficient to support it. Very limited studies have investigated these two hypotheses in relation to anxiety symptoms and no previous study, to our knowledge, has investigated this interaction effect on the use of habituated ER strategies. Finally, no study to our knowledge has investigated how reinforcement sensitivity is related to various cognitive ER strategies.

Considering these shortcomings, we tested our hypotheses using data from the second (T_1) and the third (T_2) assessment levels of the Greifswald family study. Our sample included 274 participants (154 women) who had completed the Brief Symptom Inventory and Cognitive Emotion Regulation Questionnaire at T_2, and SCL-90 and Action Regulation Emotion Systems at T_1. We examined how BIS/BAS at late adolescence (T_1, mean age = 19.56) predicts anxiety symptoms five years later in early adulthood (T_2). Using structural equation modelling, we designed a model with BIS/BAS and their interaction as predictor variables and anxiety symptoms as outcome variable. In order to examine the longitudinal effect of BIS/BAS on T_2-anxiety, we controlled for anxiety symptoms at T_1 in our model. Furthermore, total scores of both adaptive and maladaptive cognitive ER were included in the model as two mediators. As we expected, the model perfectly fitted the data. Our results showed that higher levels of BIS, but not BAS, predicts anxiety symptoms after a 5-year interval even after controlling for T_1-anxiety. This replicates and also extends previous findings on BIS-anxiety (e.g., S. L. Johnson et al., 2003; Sportel, Nauta, Hullu, Jong, & Hartman, 2011) by providing the first longitudinal evidence for this link. Further, we found a significant indirect effect of maladaptive ER on BIS-anxiety link that supports the hypothesis that BIS contributes to development of anxiety by increasing the tendency to use maladaptive cognitive ER strategies. Tortella-Feliu et al. (2010) found similar results in a large sample of adolescents (N = 1441, mean age = 14.04). Their results supported a mediating role of maladaptive cognitive ER on the link between punishment sensitivity and anxiety. However, they did not include adaptive ER and also did not report the relation between BIS/BAS and specific ER strategies. Our study showed that BIS is positively related to an increased use of all maladaptive cognitive ER strategies such as blaming self, rumination, and catastrophizing. On the other hand, BAS predicted applying three adaptive cognitive ER strategies of planning, positive reappraisal, and putting into perspective. In line with our expectation regarding the stronger effect of maladaptive ER on psychopathology symptoms, our results showed that maladaptive strategies but not adaptive ER strategies were significantly associated

with anxiety symptoms and there was also no significant indirect effect for adaptive ER. BIS sensitivity might increase maladaptive cognitive ER by provoking concerns regarding potential threats or uncertainties, and might also facilitates catastrophizing due to oversensitivity to situations of punishment and non-reward. On the contrary, reward sensitivity is related to more positive emotions and less difficulties in ER (Markarian et al., 2013). Past studies suggest that responsiveness to reward can increase individuals' resilience to negative experiences and may help buffer against daily stresses (Tugade & Fredrickson, 2004). However, as we discussed earlier, the effect of adaptive ER on psychopathology seems to be more dependent to the context in which ER strategy is deployed (Aldao & Nolen-Hoeksema, 2010).

Findings of this study illustrate how BIS in late adolescence, which is characterized by risk aversion, emotional reactivity, intolerance of uncertainty, increased negative affect, and inhibition of behavior conveys a longitudinal risk for anxiety symptoms in young adulthood. A tendency to engage in counterproductive styles of managing emotions might represent a form of reactive control in individuals with increased emotional reactivity which makes them prone to maladaptive cognitive ER (Tortella-Feliu et al., 2010). The period between late adolescence and young adulthood is an important developmental phase where adolescents face novel situations that trigger both approach (e.g., attractions of new opportunities) and avoidance motivation (e.g., risk and uncertainty associated with novel situations) (Roisman, Masten, Coatsworth, & Tellegen, 2004). This feature contributes to the more frequent experience of conflictual approach-avoidance situations in this period which results in activation of BIS with the purpose of resolving the conflict. BIS resolves the conflict by scanning the risk associated with the situation and increasing the negative affect in favor of avoidance and behavioral inhibition (Corr, 2008). Therefore, during this developmental phase, an overactivation of BIS can lead to increased negative affect that requires extra regulatory effort and can lead to development of anxiety symptoms. In addition, while there are various internal and external stressors in adolescence, BIS seems to be related to

difficulties coping with distress which means more vulnerability for adolescents with high BIS (Hundt et al., 2007).

Regarding the joint and separable effects of BIS and BAS, results showed that in contrast to the assumption of joint subsystems hypothesis, only BIS (but not BAS) predicted anxiety. Further, BIS and BAS also showed effects in favor of separable subsystems by predicting higher maladaptive and adaptive ER strategies, respectively. Furthermore, BIS/BAS interaction significantly predicts anxiety symptoms but not ER. Based on the joint subsystems hypothesis, we would expect that low levels of BAS exacerbates the effect of high levels of BIS due to the reduced level of antagonistic effect from BAS on punishment-mediated behavior. However, our results showed that the combination of high BIS and high BAS (not low BAS) predicted anxiety symptoms. These results replicated the findings of Hundt et al. (2007) that supported the main effect of BIS (not BAS) and the interaction of high BIS and high BAS in prediction of mixed depression-anxiety symptoms. This finding is highly important as it shows those adolescents with both high approach and avoidance motivations are at increased risk for development of anxiety. High levels of both approach and avoidance motivation leads to the frequent experience of conflict situations and delays the procedure of decision making, which results in the persistent activation of BIS and an increased level of negative affect, all of which contribute to the vulnerability for development of anxiety. Our results also suggest that when both approach and avoidance are high, the antagonistic effects of these systems on one another might be impaired, following which both systems might remain activated resulting in the maintenance of the conflict state and increased distress.

Finally, although the results of testing our initial model provided support that ER mediates the relationship between BIS and anxiety symptoms, given that our data for ER and anxiety were cross-sectional (both were measured at T_2), we could not make firm conclusions about the causal direction of their relationship. Therefore, in order to further examine the causal relationship between ER and anxiety, we tested an alternative model. In this model, we changed the position

of ER and anxiety to test if BIS can also lead to maladaptive ER through increasing anxiety symptoms (anxiety symptoms as mediator). This alternative model also showed a good fit. This result suggests that although the habitual use of maladaptive ER strategies such as rumination is a risk factor for development of psychopathology, it is also plausible that maladaptive ER strategies are an epiphenomenon or a by-product of psychopathology. Despite the fact that causal conclusion about the relationship between ER and psychopathology is limited since many findings so far have used cross-sectional data, previous findings suggest that the use of maladaptive ER conveys risk for psychopathology (Aldao & Nolen-Hoeksema, 2012b; Campbell-Sills et al., 2014). However, given that previous studies on the mediating effect of ER have been cross-sectional and have not tested this second alternative model (Markarian et al., 2013; Tortella-Feliu et al., 2010), more longitudinal research is necessary to provide further evidence for the direction of this relationship.

Although the Study 1 provides findings for the indirect effect of maladaptive cognitive ER on the link between BIS and anxiety, it cannot answer the question whether maladaptive ER also plays a mediating role on the link between BIS and other psychopathology symptoms. Furthermore, we measured ER independent from the emotion that needs to be regulated, while it would be interesting to narrow the focus and investigate if reinforcement sensitivity is also related to the regulation of specific emotions. Finally, although this study investigated the current and less studied hypothesis of joint and separable subsystems in relation to anxiety symptoms, the application of these two hypotheses for other psychopathology symptoms needs to be further investigated. Therefore, we designed the second study and included symptoms of depression, anxiety and aggression simultaneously, and tested the indirect effect of anger rumination as a specific strategy for regulation of the specific emotion of anger on the link between reinforcement sensitivity and psychopathology.

4.2 Reinforcement sensitivity and depression, anxiety, and aggression symptoms: The indirect effect of anger rumination: Study 2

As mentioned above, this study was conducted to 1) test the specificity of the findings from Study 1 in relation to symptoms of depression, anxiety, and aggression, and 2) to narrow the focus of Study 1 by examining the indirect effect of anger rumination on BIS/BAS and psychopathology. To examine the hypotheses of this study, we used data from the second and the third assessment phases of the Greifswald family study. Further, using structural equation modelling, we designed a model with BIS/BAS (measured at T_1) and their interaction as predictors, depression, anxiety, and aggression symptoms as outcome (measured at T_2), and anger rumination (T_2) as mediator. We also controlled for baseline psychopathology symptoms at (measured at T_1). Sample included 273 participants (154 women) with a mean age of 19.51 years (14-27) at T_1 and 24.99 years (19-34) at T_2.

In line with our findings from Study 1, there is convincing evidence that suggests that punishment sensitivity is a risk factor for internalizing symptoms such as depression and anxiety, while findings on reward sensitivity are less consistent (see Bijttebier et al., 2009). Some studies have found that low reward sensitivity is related to depression (Kimbrel et al., 2007), while other studies have found no significant relationship (Campbell-Sills et al., 2004; S. L. Johnson et al., 2003). It seems that reward sensitivity is more strongly related to externalizing symptoms rather than internalizing symptoms (Hundt et al., 2008). In addition, given that previous studies suggesting a relationship between low reward sensitivity and depression have been cross-sectional, their findings might be simply a result of shared features of low reward sensitivity and depression such as lack of positive experience (Beevers & Meyer, 2002). Our findings supported this assumption by showing a significant direct effect of BIS on depression and anxiety but not aggression, and a significant direct effect of BAS on aggression but not on depression and anxiety. Findings strengthen previous evidence on BIS as a risk factor for depression and anxiety and provide no support for the role of low reward sensitivity as vulnerability for depression.

Furthermore, given that we controlled for depression, anxiety, and aggression symptoms at T_1, our findings provide evidence for the longitudinal effect of BIS on depression/anxiety and the longitudinal effect of BAS on aggression.

The second aim of the Study 2 was to narrow the focus of Study 1 by investigating the indirect effect of anger rumination on the link between punishment/reward sensitivity and depression, anxiety, and aggression symptoms. The emotion of anger offers considerable importance considering the positive relation between punishment/reward sensitivity and elevated anger (Harmon-Jones, 2003; Smits & Kuppens, 2005). Although both punishment and reward sensitivity are related to more experience of anger, they are related to different anger responses. Termination of reward or approach obstruction results in frustration and anger among individuals with high reward sensitivity, which leads to an outward anger response and less anger control (Cooper, Gomez, & Buck, 2008). On the other hand, although punishment sensitivity is also related to greater anger arousal, it predicts an inward anger response and the inhibition of an outward anger response such as physical/verbal aggression (Cooper et al., 2008). This means that the experience of anger among individuals with punishment sensitivity demands more self-regulatory effort and might result in a prolonged cognitive processing of the emotional experience and leads to a vulnerability for anger rumination. Anger rumination is an inward response towards anger that has not been studied in relation to reinforcement sensitivity in previous research. Sukhodolsky, Golub, and Cromwell (2001) suggested that ruminative tendencies toward angry moods and experiences fall under four categories of "angry afterthoughts" (rethinking about a recent episode of anger), "angry memories" (recalling and getting angry about a distant episode of anger) "thoughts of revenge" (fantasies of taking revenge), and "understanding of causes" (trying to achieve a meaningful understanding of an anger episode). We included all these sub-traits into our model to investigate the relative importance of each of them on the relation between BIS and the aforementioned psychopathology symptoms. Our results showed that punishment sensitivity indirectly leads to depression and anxiety through recalling angry memories from previous

episodes of anger (i.e., angry memories). This finding is quite interesting given that it implies that anger experiences may remain unresolved among individuals with elevated BIS or it is possible that BIS is related to difficulties to forget and forgive following anger situations. This might also be explained by a more passive style of handling anger situations among individuals with punishment sensitivity that hampers a functional expression and communication of anger, resulting in prolonged and unresolved anger (J. L. Johnson, Kim, Giovannelli, & Cagle, 2010). Another interesting finding was that although BIS did not show a direct effect on aggression, there was an indirect effect through inducing angry memories and thoughts of revenge. In other words, punishment sensitivity might also lead to aggression through facilitating the frequent recall of angry memories or reviewing thoughts of taking revenge. It suggests that while the effect of BIS on internalizing symptoms of depression and anxiety seems to be more direct, BIS can also convey risk for externalizing symptoms indirectly through pathways such as anger rumination.

Finally, examining the BIS/BAS interaction did not provide further support for the joint subsystems hypothesis. In this study, the effects of BIS/BAS on depression/anxiety and aggression were consistent with separable subsystems hypothesis. However, we found limited support for the joint subsystems hypothesis by results showing that BIS also predicts aggression but only indirectly through increased anger rumination. It is possible that the joint effects of BIS/BAS are more dependent to moderating and mediating factors, as it has been shown in previous studies (Hundt et al., 2007). In the same line, as Corr (2002) suggests, findings on separable or joint effects of BIS/BAS might be influenced by other factors such as the level of aversive experience, life stress, and also the study sample. For example, studying individuals that are confronted with stronger or more frequent aversive stimuli might result in the dominance of BIS and independent rather than interdependent effects (Corr, 2002). Therefore, focusing on the period between late adolescence and young adulthood which is accompanied by various life stressors and a tendency for stronger response to these stressors might be a possible explaining factor in findings separable effects in this study (Rutter, 2007). Similarly, Hundt et al. (2007)

found that the effect of high BIS on anhedonic depression was increased at low BAS among young adults (mean age = 20.30) only at low levels of life stressors. It seems that at high levels of life stress, BIS increases the vulnerability for depression independent from the effect of BAS. Furthermore, Corr (2002) argues that in case of very strong BIS/BAS, the facilitatory effect is stronger than the antagonistic effect resulting in separable main effects. However, as the results of our study and the study of Hundt et al. (2007) showed the combination of high BIS and BAS predicted anxiety symptoms, which means that BAS did not antagonize the effect of BIS on anxiety. It can be argued that at both high levels of BIS and BAS, the antagonistic of one system on another system might be impaired resulting in the maintenance of the conflict state and increased distress.

Similar to the Study 1 that supported a positive relationship between punishment sensitivity and maladaptive cognitive ER, results of this study supported a positive link between Punishment sensitivity and anger rumination. This finding brought us to the next research question about the mechanism of the effect that makes individuals with high punishment sensitivity prone to maladaptive cognitive ER. As Tortella-Feliu et al. (2010) suggest, negative ER could be a form of automatic/unconscious reactive control in subjects with high emotional reactivity. They further suggest that negative ER might be a consequence of an inability to automatically inhibit the processing of threatening cues or problems with mechanisms of executive and cognitive control. Therefore, having this question in mind, we took one step back to investigate inhibitory control deficits as an underling mechanism of the link between punishment sensitivity and maladaptive cognitive ER.

5 One step back: Adolescent punishment sensitivity and dysfunctional emotion regulation in adulthood: Inhibitory control as a mechanism of effect: Study 3

Building upon the first two studies that showed individuals characterized by punishment sensitivity are prone to use maladaptive cognitive ER, this third study aims at investigating the

question how punishment sensitivity contributes to this emotional dysregulation. For this study, we applied Cloninger's (1994b) psychobiological model of personality, in which he defined the construct of punishment sensitivity under the concept of harm avoidance. Harm avoidance is regarded as a heritable bias in the inhibition or cessation of behaviors, such as fear of uncertainty, anticipatory worries, passive avoidant behaviors, shyness with the strangers, and rapid fatigability. Individuals high in harm avoidance tend to be fearful, tense, negativistic, nervous, timid, cautious, and pessimistic even in situations that do not usually worry people, and they show strong reactions towards aversive stimuli (Cloninger et al., 1993; Most, Chun, Johnson, & Kiehl, 2006). Some evidence suggests that experience of these strong negative emotions in individuals with increased harm avoidance might lead to difficulties in the procedure of ER (Schreiber et al., 2012). Similarly, harm avoidance is higher among psychopathologies characterized by emotional dysregulation (e.g., Barnow et al., 2007). It has been argued that those individuals who experience intense emotional responses may not believe that they can efficiently regulate their emotions, and therefore may be unwilling to try to regulate their emotions (Flett, Blankstein, & Obertynski, 1996). Only one study, to our knowledge, investigated the relation between harm avoidance and ER difficulties (Schreiber et al., 2012). Results of this study showed that young adults with higher levels of ER difficulties show higher levels of harm avoidance. Furthermore, in line with the findings from Studies 1 and 2 in the current dissertation, previous evidence suggests that punishment sensitivity contributes to the application of maladaptive cognitive ER strategy rumination (Manfredi et al., 2011; Tortella-Feliu et al., 2010). However, to our knowledge, no study has investigated if harm avoidance can also lead to other maladaptive cognitive ER such as self-blame, and catastrophizing. Adolescents' harm avoidance might lead to self-blame and catastrophizing in response to worries, uncertainty, or shyness that are characteristics of harm avoidance (Gilbert & Miles, 2000; Gunthert, Cohen, & Armeli, 1999). Considering previous findings that support a positive relationship between punishment sensitivity and ER difficulties

and rumination, together with our results from Studies 1 and 2, we expected harm avoidance to be also positively associated with other maladaptive cognitive ER.

As we mentioned earlier, we were interested to understand the mechanism by which higher levels of punishment sensitivity is translated to maladaptive cognitive ER. We hypothesized that one possible mechanism for this association might rely on the implications of inhibitory control. It is referred to a cognitive process that enables individuals to suppress the habitual, dominant, and inappropriate responses or attention tendencies in order to act appropriately on the task at hand (Dempster, 1992; Nigg, 2000). Although no study so far has investigated how harm avoidance is related to increased inhibitory control deficits, previous evidence suggests such an association (Hansenne, 1999; Most et al., 2006). Harm avoidance is associated with an automated pattern of attention to neutral and emotional stimuli (Hansenne et al., 2003; Mardaga & Hansenne, 2009), and an attentional bias towards emotional stimuli with negative valence (Cloninger, 1994b; Zhang et al., 2013), both of which can hamper the procedure of suppressing the irrelevant information and facilitate the interference of negative emotional information, which results in inhibitory control deficits (Matthews & Deary, 2000a; Weierich et al., 2008). In line with this, Schreiber et al. (2012) found that harm avoidance is related to attentional impulsiveness that is characterized by intrusive/racing thoughts and an inability to focus attention. Past findings have shown that individuals with high harm avoidance have difficulties inhibiting irrelevant information when searching for targets during an attentional task (Most et al., 2006; Most, Chun, Widders, & Zald, 2005). Regarding the performance in inhibitory tasks, Matthews, Joyner, Gilliland, Huggins, and Falconer (1999) showed that neuroticism (a close concept to harm avoidance) might lead to higher levels of distraction and interfering cognitions during an emotional Stroop Task, which might interfere with inhibitory control processes. Further, negative attentional bias associated with harm avoidance, might slow down the process of naming the color of emotional words that represents lower inhibitory control of the emotional stimuli (Matthews & Deary, 2000a).

On the other hand, inhibitory control deficits contribute to rumination (e.g., Joormann, 2006; Zetsche et al., 2012). For example, inhibition deficit in negative priming task has been positively associated with applying rumination (Joormann, 2006). Deficits in inhibiting neutral (Whitmer & Banich, 2007) and emotionally negative information (De Lissnyder et al., 2012; Donaldson, Lam, & Mathews, 2007; Joormann, 2010; Zetsche et al., 2012) has shown to be associated with increased rumination. Thus, previous evidence suggesting a link between harm avoidance and inhibitory control deficit coupled with findings that support inhibitory control deficits as an underlying factor for maladaptive cognitive ER point to the possibility that harm avoidance might lead to maladaptive cognitive ER through inducing inhibitory control deficits. In line with this hypothesis, in the extended process model presented by Sheppes et al. (2015), authors argue that an engagement bias (a rapid process of orientating attention toward threat), and disengagement bias (a delayed withdrawal of attention from threat following initial engagement), affect the process of emotion generation and emotion regulation, respectively. These both biases result in an overrepresentation of the current emotional state and leads to an increased regulatory effort that might be unnecessary and maladaptive. More specific, the disengagement bias has been related to insufficient attentional control, resulting in sustained engagement with threat. This sustained engagement involves, among other things, overly representing threatening information associated with the current emotional state (for a review, see J. M. Cisler & Koster, 2010). Accordingly, in the context of harm avoidance, characteristics such as attentional bias towards threat and anticipatory worries can lead to increased engagement and disengagement bias towards threatening information which demands extra regulatory effort and can facilitate maladaptive ER. Although previous studies had only focused on the link between inhibitory control and rumination, given that maladaptive cognitive ER strategies have a cognitive nature that consists of recurrent dysfunctional thoughts, it seems promising that inhibitory control deficits among individuals with punishment sensitivity might also exacerbate the application of these strategies through increasing the accessibility of dysfunctional cognitions.

To test our hypotheses, we used data from three assessment levels of the Greifswald family study. Our sample consisted of 147 female and 114 male participants (mean age at T_0 = 15.03, *SD* = 2.28) who had participated in all three assessments. Harm avoidance was measured at T_0 using the Temperament and Character Inventory (Cloninger, 1994a). At the second phase (T_1), we conducted a multimethod assessment of inhibitory control as it seems that different measures test different underlying components of inhibitory control deficits (Khng & Lee, 2014; Reynolds, Ortengren, Richards, & de Wit, 2006). We applied two well-known computer-based inhibitory control tasks of *emotional Stroop* and *stop-signal task*, together with a self-report instrument, the *Barrat Impulsiveness Scale* (Patton et al., 1995). Barrat Impulsiveness Scale measures three subscales of motor impulsivity (the tendency to act without thinking), non-planning impulsivity (lack of futuring or forethought), and attentional impulsivity (rapid shifts in attentional focus, intrusive thoughts). In emotional Stroop task participants should name the ink color of the emotional and neutral word stimuli as fast and accurately as possible, while at the same time ignoring the word meaning. Slowing of naming the ink color of emotional as compared to neutral words represents the emotional interference effect. The difference between reaction time to emotional and neutral stimuli is called emotional interference and represents the extent to which participants could inhibit the interference of emotional word on the task at hand (naming the color). The stop-signal paradigm requires a rapid and practiced response to visual stimulus on go trials, and the withholding of that response on a minority of trials when this visual stimuli is followed by an audio stop-signal (Khng & Lee, 2014). The interval between go-signal and stop-signal is called stop-signal delay. We calculated Stop-Signal Reaction Time (SSRT) by subtracting the mean stop-signal delay required by subjects to correctly inhibit responses, from mean go reaction time on no-stop trials. Higher SSRT represents lower inhibitory control. Furthermore, maladaptive cognitive ER was measured at the third phase (T_2) using Cognitive Emotion Regulation Questionnaire (Garnefski & Kraaij, 2007). To ensure the longitudinal effect of harm avoidance on ER, we included participants' scores on harm avoidance measured at T_2 to

control for cross-sectional effect of harm avoidance on ER in adulthood. We designed a mediation model with harm avoidance as predictor of maladaptive cognitive ER and included all inhibitory control indexes as mediator variables (SSRT, emotional interference, attentional impulsiveness, motor impulsiveness, and non-planning impulsiveness). Given that previous studies show significantly higher levels of harm avoidance among women compared to men (Al-Halabí et al., 2011; Cloninger, Svrakic, & Przybeck, 2006), we also investigated the moderating effect of gender in the paths that included harm avoidance (harm avoidance to ER; harm avoidance to inhibitory control), which resulted in a moderated mediation model.

Results of correlational analysis for female participants showed a positive correlation between female adolescents' harm avoidance (at both T_0 and T_2), and higher levels of attentional impulsivity, catastrophizing, rumination, self-blame, and blaming others. Among inhibitory control measures, only attentional impulsivity was related to higher levels of all the above-mentioned ER strategies, while other inhibitory control indexes appeared to be unrelated.

Among male participants, harm avoidance only predicted more catastrophizing and less emotional interference. However, the cross-sectional positive correlation between harm avoidance and maladaptive ER at T_2 was significant for all ER strategies. Among inhibitory control measures, only attentional impulsivity was correlated with higher levels of the ER strategy rumination. In summary, harm avoidance was correlated with more attentional impulsivity only among women but attentional impulsivity was related to more rumination among both women and men.

In order to conduct the moderated mediation model, we applied the bootstrapping method introduced by Preacher and Hayes (2008) and used the SPSS macro PROCESS (Hayes, 2013). We entered harm avoidance at T_0 as predictor, all inhibitory control indexes as mediator, and total scores of maladaptive ER as outcome. We also included harm avoidance at T_2 in order to control for the cross-sectional effect of harm avoidance on ER. Conducting the analysis resulted in

gender-specific and measure-specific findings. First, we found that adolescent harm avoidance among female but not male participants predicted higher levels of maladaptive ER 10 years later in adulthood, even after controlling for the harm avoidance at T_2. Our results suggest that harm avoidance among female adolescents can be a risk factor for development of maladaptive ER in young adulthood. This is a novel finding since previous studies on the relationship between harm avoidance and ER did not control for the moderating effect of gender. This gender-specific finding might be related to women reporting higher levels of harm avoidance (e.g., Al-Halabí et al., 2011) and maladaptive cognitive ER strategies compared to men (Zlomke & Hahn, 2010). Another explanation might rely on men and women's different approaches toward stressors. In a meta-analysis by Tamres, Janicki, and Helgeson (2002) women used more rumination and self-blame, appraised stressors as being more severe than men and also engaged more often in uncontrollable stressors, while men tended to withdraw or avoid such situations (Tamres et al., 2002). This approach can put female adolescents at higher risk of increased emotional distress particularly during the period between adolescence and young adulthood when they face important developmental stressors and are required to make important decisions regarding educational, occupational, and emotional aspects of life (Roisman et al., 2004)

Our analysis also demonstrated a conditional indirect effect for inhibitory control, as measured with attentional impulsivity, on the relationship between females' adolescent harm avoidance and maladaptive cognitive ER. In other words harm avoidance among women leaded to higher attentional impulsivity which in turn contributed to higher scores on maladaptive ER. This finding is consistent with Schreiber et al. (2012) who found a significant positive correlation between harm avoidance and both ER difficulties and attentional impulsivity. Furthermore, in line with our findings, attentional control or the ability to voluntarily focus or shift attention when needed, has been suggested as a possible mechanism that provides a path by which personality traits such as punishment sensitivity might lead to emotional dysregulation (Bijttebier et al., 2009; Lonigan et al., 2004). Findings support the assumption of different underling mechanisms for

different inhibitory control measures (Reynolds et al., 2006). While behavioral or performance-based tasks of inhibitory control are influenced by temporal fluctuations (state-dependent), self-report measures represent a more stable aspect of inhibition that cover broad periods of time (trait-dependent) (Dougherty, Mathias, Marsh, & Jagar, 2005). Therefore, low stability and state-dependency of performance-based measures might explain the insignificant relation between harm avoidance, stop-signal task, and emotional Stroop, particularly in longitudinal studies. According to past theories, it is possible that performance in behavioral tasks is more strongly influenced by negative affective state rather than the trait (Matthews & Deary, 2000b). Future studies could answer the question whether the effect of harm avoidance on inhibitory control tasks is mediated by negative affective state before and after completing the task. It is also important to mention that stop-signal task and emotional Stroop measure deliberate and controlled suppression of prepotent behavior, while harm avoidance might be related to an automatic disinhibition which is unintentional and might be better measured through other measures such as negative priming or saccadic interference task (Roberts, Fillmore, & Milich, 2011). In saccadic interference task, an automatic response to a visual distractor should be inhibited in favor of responding to a visual target. Considering that harm avoidance is associated with an automatic pattern of attending to stimuli, it seems plausible that harm avoidance is associated with a less intentional kind of disinhibition, which is not measured through stop-signal task and emotional Stroop. However, it is possible that this disinhibition can be recognized by the person and therefore reflected in self-report measure of attentional impulsivity that represents deficits in inhibiting intrusive thoughts and impulsive attentional shifts (Patton et al., 1995).

Study 3 provided the first evidence for a longitudinal effect of adolescent harm avoidance on maladaptive cognitive ER among adult females. We further found evidence for the indirect effect of attentional impulsivity on the link between harm avoidance and maladaptive cognitive ER. However, the lack of multiple assessments of inhibitory control and ER at all three measurement phases restricts our ability to make firm cause-effect conclusions about the

relationship between inhibitory control and ER. However, findings of the current study contribute to prevention programs by offering new insight into an underlying mechanism through which female adolescents with high harm avoidance might become prone to development of maladaptive ER. Harm avoidance is a trait with considerable stability across the life span (Josefsson et al., 2013). Therefore, the maladaptive influence of harm avoidance might be better controlled through targeting indirect pathways such as attentional control that has been shown to be improved through training programs such as mindfulness (Chambers, Lo, & Allen, 2008). Considering the aforementioned limitations of this study, further research can complement these findings by including measures for less intentional inhibitory control such as saccadic interference, designing longitudinal studies with multiple assessments of both ER and inhibitory control, and testing the hypotheses using data from clinical samples.

6 Implications for clinical practice and future research

Findings from the current dissertation demonstrate that punishment sensitivity in adolescence is a longitudinal risk factor for symptoms of depression, anxiety, and also aggression in adulthood, while reward sensitivity contributes to development of aggressive symptoms. It was shown that punishment sensitivity contributes to a habitual use of maladaptive ER, which in turn increases the risk for development of the aforementioned psychopathology symptoms. These findings have particular theoretical and practical implications. Given that current models of ER do not provide clear prediction about the relation between ER strategies and specific psychological strengths and vulnerabilities, empirical research that investigates the development of ER from important psychological constructs such as personality, have both empirical and theoretical value. Furthermore, the results of the present studies can inform etiological models of mood and anxiety disorders and also contributes to the development of prevention and intervention programs. Our finding on the indirect effect of maladaptive ER implies that improvement of ER skills among those adolescents with elevated levels of punishment sensitivity might convey a protective effect

against the development of psychopathology symptoms. This requires early assessment of reinforcement sensitivity to identify adolescents prone to development of dysfunctional ER and psychopathology. Adolescence offers a significant importance for conducting such preventive interventions as in this period there is a mismatch between adolescents' self-regulation capacities that are not mature and the fast increase in their emotional arousability. This mismatch makes this developmental stage a high risk period for development of emotional dysregulation and psychopathology (Steinberg, 2005). It is notable that while stressful life events seems to have a stronger emotional impact on adolescents compared to other developmental stages (Rutter, 2007), there is a growing desire for independence in adolescence, which means an increased reliance on personal self-regulatory resources (Yap et al., 2007). These features suggest that adolescence is associated with a heightened vulnerability to problems associated with poor regulation of affect and behavior. Therefore it is essential for clinicians to identify vulnerable adolescents and to include components of ER skills in their preventive plan in order to facilitate the development of a functional repertoire of ER strategies.

Our results also highlighted the importance of studies that investigate the regulation of specific emotions in relation to psychopathology. Our findings showed that those with high punishment sensitivity who ruminated anger more often by recalling anger memories, were more prone towards depressive and anxiety symptoms. One particularly interesting finding was that although aggression is an externalizing symptom and is assumed to be related to reward sensitivity (Hundt et al., 2008), punishment sensitivity did also indirectly lead to aggression through increased levels of angry memories and thoughts of revenge. It is possible that a passive approach in dealing with anger situations lead to unresolved anger experiences and therefore anger rumination. This is up to further research to investigate whether enhancing forgiveness or instructing an assertive approach towards anger situations is associated with lower anger rumination among individuals with punishment sensitivity. Furthermore, future research might complement these findings by investigating various types of aggression (e.g., verbal, physical,

hostility) in relation to punishment sensitivity and anger rumination (Anestis, Anestis, Selby, & Joiner, 2009). Given that showing active aggression might be accompanied by negative social consequences that individuals with punishment sensitivity usually try to avoid, it is possible that anger rumination mediates the relationship between punishment sensitivity and less active types of aggression such as hostility or passive aggression (Smits & Kuppens, 2005). Additionally, studies have shown that those individuals who ruminate anger also tend to ruminate sadness. Therefore, including both anger rumination and sadness rumination in future research can shed light on their specific roles on the relationship between reinforcement sensitivity and development of various symptoms. Furthermore, an interesting question for further research is that how regulation of other specific emotions such as shame, or using other strategies for regulating anger is relevant to psychopathology symptoms among individuals with higher levels of punishment sensitivity (Orth, Berking, & Burkhardt, 2006; Sheikh & Janoff-Bulman, 2009).

Building upon the first two studies, in order to understand the mechanism by which punishment sensitivity leads to a vulnerability to maladaptive style of ER, in Study 3 we investigated the indirect effect of inhibitory control and found initial evidence that supported an indirect effect of attentional impulsivity on the relationship between punishment sensitivity and maladaptive cognitive ER. This finding suggests that focusing on enhancement of attentional control might have a buffering effect against development of maladaptive ER. It has been shown that clinical interventions such as mindfulness facilitate the application of higher level executive attention for regulating automatic emotional responses (Jha, Krompinger, & Baime, 2007) and improve both attentional control (Lattimore, Fisher, & Malinowski, 2011) and ER ability (A. M. Hayes & Feldman, 2004). The significance of attentional bias for ER and emotional disorders have found more support in novel attentional bias modification treatment (Grafton & MacLeod, 2014; MacLeod & Clarke, 2015) that has been proven to be effective in treatment of emotional disorders by targeting the identification stage, with the goal of reducing the overrepresentation of threatening information related to the current emotional state. This seems to be particularly

important among those with higher levels of punishment sensitivity as this treatment might reduce their attentional bias towards emotionally negative stimuli and has a protective effect against development of maladaptive ER strategies and emotional disorders (Joormann & Gotlib, 2010). Although these results provide valuable insight into the role of attentional control in development of maladaptive ER, future research should replicate this study to further clarify the degree to which the indirect effect of attentional control on the link between harm avoidance and maladaptive ER is gender specific. The significance of this indirect effect among women might be explained by their higher levels of punishment sensitivity (e.g., Al-Halabí et al., 2011; Heym, Ferguson, & Lawrence, 2008). Replicating this study with clinical samples or individuals at the high pole of punishment sensitivity might be able to answer the question whether the relation between punishment sensitivity and attentional control is independent from gender in samples with elevated punishment sensitivity. Furthermore, we did not find a significant relationship between punishment sensitivity and the computer-based task of inhibitory control, namely emotional Stroop and stop-signal task. Considering that these two tasks measure a more controlled, and deliberate inhibitory control (Miyake et al., 2000), while punishment sensitivity is related to an automatic pattern of attending to stimuli, future research might provide valuable complementary evidence by investigating the relationship between punishment sensitivity and less intentional measures of inhibitory control such as negative priming or saccadic interference (Roberts et al., 2011).

In addition, our findings were in favor of separable subsystems hypothesis and provided only limited support for the joint subsystems hypothesis. Findings suggested that depression and anxiety and emotional dysregulation are related to a dominance of BIS, while adaptive ER and aggression are related to BAS. However, there was also an indirect effect of BIS on aggression through anger rumination. These findings have important empirical and theoretical implications. First, they suggest that joint effects of BIS/BAS might operate through indirect pathways that can be best investigated by unpacking studies that consider the influence of potential mediators or

moderators. Second, it seems that we cannot completely reject one of these two hypotheses in favor of the other because as it appears, depending on the psychological construct (e.g., depression, anxiety, ER), the developmental stage of the participants, and the levels of BIS and BAS in sample, joint or separable subsystems hypothesis might be in operation (Corr, 2002). Finally, although Corr (2002) hypothesized that at high levels of BIS or BAS facilitatory effects are in operation that result in separable effects, our findings suggest that in case of both high BIS and BAS, the two systems seem to interact and their interaction exacerbates the risk of anxiety symptoms.

Although our findings on ER showed that measurement of ER through questionnaires can be an economical method that represents the relative functionality and dysfunctionality of applied strategies, measurement of ER through daily-based methods such as ecological momentary assessment (whereby ER is assessed at multiple times and across a number of different contexts) provides important context-related information that facilitates understanding the functionality or dysfunctionality of a particular strategy. This is particularly important given that the degree of the functionality of ER strategies seems to be dependent on contextual demands. These methods also assess other important ER indexes such as ER flexibility, which is defined as the degree to which an ER strategy is synchronized with contextual demands and facilitates achieving personally meaningful goals (Aldao, Sheppes, & Gross, 2015). In addition to that, the inclusion of non-conscious and less intentional ER processes is also important pathway for further research, which is still limited by measurement concerns. Furthermore, including multiple ER assessments at baseline and follow up measurements in future research enables investigators to make firm conclusions about the cause-effect relationship between ER and psychopathology. In Greifswald Family Study, the first assessment level started in the late 1990s that research of ER was still at the very beginning and the currently well-known questionnaires were not developed yet. Cognitive emotion regulation questionnaire was measured at T_2 and enabled us to include various cognitive ER strategies in our models. Nevertheless, other important strategies such as

suppression and avoidance were not included in this questionnaire. In addition, there have been theoretical and empirical concerns regarding its criterion validity (Izadpanah, Barnow, Neubauer, & Julia, Under review). Further implication of our findings is the importance of examining mediation models as they provide insight into hidden mechanisms that provide valuable information for treatment and prevention programs. Findings also suggested that further research investigating mediation models should acknowledge the new methodological changes in mediation analysis that reject the necessity of a significant direct effect of predictors on outcomes (Hayes, 2013). For example, in our results from Study 2, punishment sensitivity leaded to aggression only when participants had experienced thoughts of revenge and recalled angry memories (despite insignificant direct effect of punishment sensitivity on aggression). Therefore, neglecting these current methodological developments might lead to misunderstanding the data and missing valuable information.

7 Conclusion

The major aim of this dissertation was to examine the role of reinforcement sensitivity in development of psychopathology symptoms and to provide insight into the role of ER and inhibitory control as possible mechanisms that underlie this development. Our results provided evidence for punishment sensitivity as a longitudinal risk factor for development of depression, anxiety, and aggression symptoms. Findings support the importance of including personality in theoretical models on development of ER and psychopathology. Understanding the degree to which personality traits predispose individuals to later development of psychopathology is essential for development of effective preventive programs. The early identification of personality profiles that put adolescents at risk for development of psychopathology leads to interventions that concentrate on modification of personality characteristics or on promoting the resilience of adolescents by enhancing skills that can implement protective effects (Tackett, 2006). This approach provides the opportunity to influence psychopathological trajectories before

development of a rigid and rigorous to change psychopathological state. For designing these prevention programs, empirical studies that unpack the underlying mechanisms of the personality-psychopathology link are highly important. Given that personality traits show considerable stability across the life span (Josefsson et al., 2013), the chance of preventing development of psychopathology among adolescents with predisposing personality profiles will increase by enhancing the knowledge on the possible mediating and moderating constructs that can be included in programs with the aim of prevention and early intervention. The current dissertation introduced emotional dysregulation and attentional control as two underlying constructs that can be targeted in such programs. Findings suggest that an enhanced attentional control might protect those with punishment sensitivity against development of maladaptive ER. Furthermore, our results support that training effective regulation of emotions that elaborates adolescents' ER repertoire might help adolescents with punishment sensitivity to deal with negative affect in an adaptive way and prevent maladaptive ER from turning into long established and resistant to change regulation style.

References

Al-Halabí, S., Herrero, R., Sáiz, P. A., García-Portilla, M. P., Errasti, J. M., Corcoran, P., . . . Bobes, J. (2011). A cross-cultural comparison between Spain and the USA: Temperament and character distribution by sex and age. *Psychiatry Research, 186*(2–3), 397-401. doi:10.1016/j.psychres.2010.07.021

Aldao, A., & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: A transdiagnostic examination. *Behaviour Research and Therapy, 48*(10), 974-983. doi:10.1016/j.brat.2010.06.002

Aldao, A., & Nolen-Hoeksema, S. (2012a). The influence of context on the implementation of adaptive emotion regulation strategies. *Behaviour Research and Therapy, 50*(7), 493-501. doi:10.1016/j.brat.2012.04.004. Epub 2012 May 7

Aldao, A., & Nolen-Hoeksema, S. (2012b). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology, 121*(1), 276-281. doi:10.1037/a0023598

Aldao, A., Nolen-Hoeksema, S., & Schweizer, S. (2010). Emotion-regulation strategies across psychopathology: A meta-analytic review. *Clinical Psychology Review, 30*(2), 217-237. doi:10.1016/j.cpr.2009.11.004

Aldao, A., Sheppes, G., & Gross, J. J. (2015). Emotion regulation flexibility. *Cognitive Therapy and Research, 39*(3), 263-278. doi:10.1007/s10608-014-9662-4

Aldinger, M., Stopsack, M., Ulrich, I., Appel, K., Reinelt, E., Wolff, S., . . . Barnow, S. (2014). Neuroticism developmental courses - implications for depression, anxiety and everyday emotional experience: A prospective study from adolescence to young adulthood. *BMC Psychiatry, 14*, 210. doi:10.1186/s12888-014-0210-2

American Psychiatric Association (Ed.) (2013). *Diagnostic and statistical manual of mental disorders : DSM-5* (5. ed. ed.). Washington, DC [u.a.]: American Psychiatric Assoc.

Anestis, M. D., Anestis, J. C., Selby, E. A., & Joiner, T. E. (2009). Anger rumination across forms of aggression. *Personality and Individual Differences, 46*(2), 192-196. doi:10.1016/j.paid.2008.09.026

Barnow, S. (2012). Emotion regulation and psychopathology [Emotionsregulation und Psychopathologie]. *Psychologische Rundschau, 63*(2), 111-124. doi:doi:10.1026/0033-3042/a000119

Barnow, S., Aldinger, M., Ulrich, I., & Stopsack, M. (2013). Emotion regulation in depression: An overview of results using various methods [Emotionsregulation bei Depression: Ein multimethodaler Überblick]. *Psychologische Rundschau, 64*(4), 235-243. doi:10.1026/0033-3042/a000172

Barnow, S., Herpertz, S. C., Spitzer, C., Stopsack, M., Preuss, U. W., Grabe, H. J., . . . Akiskal, H. S. (2007). Temperament and character in patients with borderline personality disorder taking gender and comorbidity into account. *Psychopathology, 40*(6), 369-378. doi:10.1159/000106467

Barnow, S., Schuckit, M. A., Lucht, M., John, U., & Freyberger, H. J. (2002). The importance of a positive family history of alcoholism, parental rejection and emotional warmth, behavioral problems and peer substance use for alcohol problems in teenagers: a path analysis. *Journal of Studies on Alcohol, 63*(3), 305-315. doi:10.15288/jsa.2002.63.305

Barnow, S., Stopsack, M., & Ulrich, I. (2010). Prävalenz und Familiarität von Persönlichkeitsstörungen in Deutschland : Ergebnisse der Greifswalder Familienstudie [Prevalence and familiarity of personality disorders in Germany: Results of the Greifswald family study]. *Psychotherapie, Psychosomatik, Medizinische Psychologie, 60*(9/10), 334-341.

Beevers, C. G., & Meyer, B. (2002). Lack of positive experiences and positive expectancies mediate the relationship between BAS responsiveness and depression. *Cognition & Emotion, 16*(4), 549-564. doi:10.1080/02699930143000365

Bijttebier, P., Beck, I., Claes, L., & Vandereycken, W. (2009). Gray's reinforcement sensitivity theory as a framework for research on personality–psychopathology associations. *Clinical Psychology Review, 29*(5), 421-430. doi:10.1016/j.cpr.2009.04.002

Brenning, K. M., & Braet, C. (2013). The emotion regulation model of attachment: An emotion-specific approach. *Personal Relationships, 20*(1), 107-123.

Campbell-Sills, L., Ellard, K. K., & Barlow, D. H. (2014). Emotion regulation in anxiety disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (2nd ed., pp. 393-412). New York, NY: Guilford Press.

Campbell-Sills, L., Liverant, G. I., & Brown, T. A. (2004). Psychometric evaluation of the behavioral Inhibition/behavioral activation scales in a large sample of outpatients with anxiety and mood disorders. *Psychological Assessment, 16*(3), 244-254. doi:10.1037/1040-3590.16.3.244

Chambers, R., Lo, B. C. Y., & Allen, N. B. (2008). The impact of intensive mindfulness training on attentional control, cognitive style, and affect. *Cognitive Therapy and Research, 32*(3), 303-322. doi:10.1007/s10608-007-9119-0

Cisler, J., Olatunji, B., Feldner, M., & Forsyth, J. (2010). Emotion regulation and the anxiety disorders: An integrative review. *Journal of Psychopathology and Behavioral Assessment, 32*(1), 68-82. doi:10.1007/s10862-009-9161-1

Cisler, J. M., & Koster, E. H. (2010). Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review. *Clinical Psychology Review, 30*(2), 203-216. doi:10.1016/j.cpr.2009.11.003. Epub 2009 Dec 14

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants: A proposal. *Archives of General Psychiatry, 44*(6), 573-588. doi:10.1001/archpsyc.1987.01800180093014

Cloninger, C. R. (1994a). *The temperament and character inventory (TCI): A guide to its development and use*. St. Louis, MO: Center for Psychobiology of Personality, Washington University.

Cloninger, C. R. (1994b). Temperament and personality. *Current Opinion in Neurobiology, 4*(2), 266-273. doi:10.1016/0959-4388(94)90083-3

Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry, 50*(12), 975-990. doi:10.1001/archpsyc.1993.01820240059008

Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (2006). Can personality assessment predict future depression? A twelve-month follow-up of 631 subjects. *Journal of Affective Disorders, 92*(1), 35-44. doi:10.1016/j.jad.2005.12.034

Cooper, A., Gomez, R., & Buck, E. (2008). The relationships between the BIS and BAS, anger and responses to anger. *Personality and Individual Differences, 44*(2), 403-413. doi:10.1016/j.paid.2007.09.005

Corr, P. J. (2002). J. A. Gray's reinforcement sensitivity theory: tests of the joint subsystems hypothesis of anxiety and impulsivity. *Personality and Individual Differences, 33*(4), 511-532. doi:10.1016/S0191-8869(01)00170-2

Corr, P. J. (Ed.) (2008). *The reinforcement sensitivity theory of personality* (1. publ. ed.). Cambridge [u.a.]: Cambridge Univ. Press.

Corr, P. J., & McNaughton, N. (2008). Reinforcement sensitivity theory and personality. In P. J. Corr (Ed.), *The reinforcement theory of personality* (pp. 155-187). Cambridge: Cambridge University Press.

Costello, E. J., Copeland, W., & Angold, A. (2011). Trends in psychopathology across the adolescent years: what changes when children become adolescents, and when adolescents become adults? *Journal of Child Psychology and Psychiatry, 52*(10), 1015-1025.

de Graaf, R., ten Have, M., van Gool, C., & van Dorsselaer, S. (2012). Prevalence of mental disorders and trends from 1996 to 2009. Results from the Netherlands mental health survey and Iicidence study-2. *Social Psychiatry and Psychiatric Epidemiology, 47*(2), 203-213. doi:10.1007/s00127-010-0334-8

De Lissnyder, E., Koster, E. H. W., Goubert, L., Onraedt, T., Vanderhasselt, M.-A., & De Raedt, R. (2012). Cognitive control moderates the association between stress and rumination. *Journal of Behavior Therapy and Experimental Psychiatry, 43*(1), 519-525. doi:10.1016/j.jbtep.2011.07.004

Dempster, F. N. (1992). The rise and fall of the inhibitory mechanism: Toward a unified theory of cognitive development and aging. *Developmental Review, 12*(1), 45-75. doi:10.1016/0273-2297(92)90003-K

Donaldson, C., Lam, D., & Mathews, A. (2007). Rumination and attention in major depression. *Behaviour Research and Therapy, 45*(11), 2664-2678. doi:10.1016/j.brat.2007.07.002

Dougherty, D. M., Mathias, C. W., Marsh, D. M., & Jagar, A. A. (2005). Laboratory behavioral measures of impulsivity. *Behavior Research Methods, 37*(1), 82-90. doi:10.3758/bf03206401

Flett, G. L., Blankstein, K. R., & Obertynski, M. (1996). Affect intensity, coping styles, mood regulation expectancies, and depressive symptoms. *Personality and Individual Differences, 20*(2), 221-228.

Folk, J. B., Zeman, J. L., Poon, J. A., & Dallaire, D. H. (2014). A longitudinal examination of emotion regulation: Pathways to anxiety and depressive symptoms in urban minority youth. *Child and Adolescent Mental Health, 19*(4), 243-250.

Fox, N. A., Henderson, H. A., Marshall, P. J., Nichols, K. E., & Ghera, M. M. (2005). Behavioral inhibition: Linking biology and behavior within a developmental framework. *Annual Review of Psychology, 56*(1), 235-262. doi:10.1146/annurev.psych.55.090902.141532

Franke, G. H. (1995). *SCL-90-R: The symptom-checklist from Derogatis: German version*. Göttingen: Beltz Test.

Franke, G. H. (2000). *BSI: Brief symptom inventory from Derogatis (short form of SCL-90-R); German version; manual*: Beltz Test.

Gable, S. L., Reis, H. T., & Elliot, A. J. (2000). Behavioral activation and inhibition in everyday life. *Journal of Personality and Social Psychology, 78*(6), 1135-1149. doi:10.1037//0022-3514.78.6.1135

Garnefski, N., Boon, S., & Kraaij, V. (2003). Relationships between cognitive strategies of adolescents and depressive symptomatology across different types of life event. *Journal of youth and adolescence, 32*(6), 401-408. doi:10.1023/A:1025994200559

Garnefski, N., & Kraaij, V. (2006). Relationships between cognitive emotion regulation strategies and depressive symptoms: A comparative study of five specific samples. *Personality and Individual Differences, 40*(8), 1659-1669. doi:10.1016/j.paid.2005.12.009

Garnefski, N., & Kraaij, V. (2007). The cognitive emotion regulation questionnaire. *European Journal of Psychological Assessment, 23*(3), 141-149. doi:10.1027/1015-5759.23.3.141

Garnefski, N., Kraaij, V., & Spinhoven, P. (2001). Negative life events, cognitive emotion regulation and emotional problems. *Personality and Individual Differences, 30*(8), 1311-1327. doi:10.1016/S0191-8869(00)00113-6

Garnefski, N., Kraaij, V., & van Etten, M. (2005). Specificity of relations between adolescents' cognitive emotion regulation strategies and Internalizing and Externalizing psychopathology. *Journal of Adolescence, 28*(5), 619-631. doi:10.1016/j.adolescence.2004.12.009

Garnefski, N., Van den Kommer, T., Kraaij, V., Teerds, J., Legerstee, J., & Onstein, E. (2002). The relationship between cognitive emotion regulation strategies and emotional problems: Comparison between a clinical and a non-clinical sample. *European Journal of Personality, 16*(5), 403-420. doi:10.1002/Per.458

Gilbert, P., & Miles, J. N. V. (2000). Sensitivity to social put-down: It's relationship to perceptions of social rank, shame, social anxiety, depression, anger, and self-other blame. *Personality and Individual Differences, 29*(4), 757-774. doi:10.1016/S0191-8869(99)00230-5

Grafton, B., & MacLeod, C. (2013). Don't start what you can't stop: Differentiating individual differences in ruminative onset and ruminative persistence, and their contributions to dysphoria. *Emotion, 13*(6), 1080. doi:10.1037/a0033876

Grafton, B., & MacLeod, C. (2014). Enhanced probing of attentional bias: The independence of anxiety-linked selectivity in attentional engagement with and disengagement from negative information. *Cognition and Emotion, 28*(7), 1287-1302.

Gray, J. A. (1982). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system.* . Oxford, England: Oxford University Press.

Gray, J. A. (1994). Framework for a taxonomy of psychiatric disorder In S. H. M. Van Goozen, N. E. Van de Poll, & J. A. Seregeant (Eds.), *Emotions: essays on emotion theory* (pp. 29-59). Hillsdale, N. J: Lawrence Erlbaum Associates.

Gray, J. A., & McNaughton, N. (2003). *The neuropsychology of anxiety: An enquiry into the function of the septo-hippocampal system* (2nd ed.). Oxford: Oxford University Press.

Gross, J. J. (1998). Antecedent-and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *Journal of Personality and Social Psychology, 74*(1), 224. doi:10.1037/0022-3514.74.1.224

Gross, J. J. (2013). *Handbook of emotion regulation* (2. ed.). New York, NY [u.a.]: Guilford publications.

Gunthert, K. C., Cohen, L. H., & Armeli, S. (1999). The role of neuroticism in daily stress and coping. *Journal of Personality and Social Psychology, 77*(5), 1087-1100. doi:10.1037/0022-3514.77.5.1087

Hannan, S. M., & Orcutt, H. K. (2013). Emotion dysregulation as a partial mediator between reinforcement sensitivity and posttraumatic stress symptoms. *Personality and Individual Differences, 55*(5), 574-578. doi:10.1016/j.paid.2013.04.028

Hansenne, M. (1999). P300 and personality: An investigation with the Cloninger's model. *Biological Psychology, 50*(2), 143-155. doi:10.1016/S0301-0511(99)00008-3

Hansenne, M., Pinto, E., Scantamburlo, G., Renard, B., Reggers, J., Fuchs, S., . . . Ansseau, M. (2003). Harm avoidance is related to mismatch negativity (MMN) amplitude in healthy subjects. *Personality and Individual Differences, 34*(6), 1039-1048. doi:10.1016/s0191-8869(02)00088-0

Harmon-Jones, E. (2003). Anger and the behavioral approach system. *Personality and Individual Differences, 35*(5), 995-1005. doi:10.1016/S0191-8869(02)00313-6

Hartig, J., & Moosbrugger, H. (2003). The ARES-Scales for measurment of BIS and BAS sensitivity. *Zeitschrift für Differentielle und Diagnostische Psychologie, 24*(4), 293-310. doi:10.1024/0170-1789.24.4.293

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*: Guilford Press.

Heym, N., Ferguson, E., & Lawrence, C. (2008). An evaluation of the relationship between Gray's revised RST and Eysenck's PEN: Distinguishing BIS and FFFS in Carver and White's BIS/BAS scales. *Personality and Individual Differences, 45*(8), 709-715. doi:10.1016/j.paid.2008.07.013

Hundt, N. E., Brown, L. H., Kimbrel, N. A., Walsh, M. A., Nelson-Gray, R., & Kwapil, T. R. (2013). Reinforcement sensitivity theory predicts positive and negative affect in daily life. *Personality and Individual Differences, 54*(3), 350-354. doi:10.1016/j.paid.2012.09.021

Hundt, N. E., Kimbrel, N. A., Mitchell, J. T., & Nelson-Gray, R. O. (2008). High BAS, but not low BIS, predicts externalizing symptoms in adults. *Personality and Individual Differences, 44*(3), 565-575. doi:10.1016/j.paid.2007.09.018

Hundt, N. E., Nelson-Gray, R. O., Kimbrel, N. A., Mitchell, J. T., & Kwapil, T. R. (2007). The interaction of reinforcement sensitivity and life events in the prediction of anhedonic depression and mixed anxiety-depression symptoms. *Personality and Individual Differences, 43*(5), 1001-1012. doi:10.1016/j.paid.2007.02.021

Hundt, N. E., Williams, A. M., Mendelson, J., & Nelson-Gray, R. (2013). Coping mediates relationships between reinforcement sensitivity and symptoms of psychopathology. *Personality and Individual Differences, 54*(6), 726-731. doi:10.1016/j.paid.2012.11.028

Izadpanah, S., Barnow, S., Neubauer, A., & Julia, H. (Under review). Development and validation of the Heidelberg Form for Emotion Regulation Strategies (HFERST): Factor structure, reliability, and validity.

Jacobi, F., Höfler, M., Strehle, J., Mack, S., Gerschler, A., Scholl, L., . . . Wittchen, H.-U. (2015). Twelve-months prevalence of mental disorders in the German Health Interview and Examination Survey for Adults – Mental Health Module (DEGS1-MH): A methodological addendum and correction. *International Journal of Methods in Psychiatric Research, 24*(4), 305-313. doi:10.1002/mpr.1479

Jha, A. P., Krompinger, J., & Baime, M. J. (2007). Mindfulness training modifies subsystems of attention. *Cognitive Affective and Behavioral Neuroscience, 7*(2), 109-119. doi:Doi 10.3758/Cabn.7.2.109

John, U., Greiner, B., Hensel, E., Ludemann, J., Piek, M., Sauer, S., . . . Kessler, C. (2001). Study of health in Pomerania (SHIP): A health examination survey in an east German region: Objectives and design. *Sozial- und Praventivmedizin, 46*(3), 186-194. doi:10.1007/BF01324255

Johnson, J. L., Kim, L. M., Giovannelli, T. S., & Cagle, T. (2010). Reinforcement sensitivity theory, vengeance, and forgiveness. *Personality and Individual Differences, 48*(5), 612-616. doi:10.1016/j.paid.2009.12.018

Johnson, S. L., Turner, R. J., & Iwata, N. (2003). BIS/BAS Levels and Psychiatric Disorder: An epidemiological study. *Journal of Psychopathology and Behavioral Assessment, 25*(1), 25-36. doi:10.1023/A:1022247919288

Joormann, J. (2006). Differential effects of rumination and dysphoria on the inhibition of irrelevant emotional material: Evidence from a negative priming task. *Cognitive Therapy and Research, 30*(2), 149-160. doi:10.1007/s10608-006-9035-8

Joormann, J. (2010). Cognitive inhibition and emotion regulation in depression. *Current Directions in Psychological Science, 19*(3), 161-166. doi:10.1177/0963721410370293

Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion, 24*(2), 281-298. doi:10.1080/02699930903407948

Jorm, A. F., Christensen, H., Henderson, A. S., Jacomb, P. A., Korten, A. E., & Rodgers, B. (1998). Using the BIS/BAS scales to measure behavioural inhibition and behavioural activation: Factor structure, validity and norms in a large community sample. *Personality and Individual Differences, 26*(1), 49-58. doi:10.1016/S0191-8869(98)00143-3

Josefsson, K., Jokela, M., Cloninger, C. R., Hintsanen, M., Salo, J., Hintsa, T., . . . Keltikangas-Järvinen, L. (2013). Maturity and change in personality: Developmental trends of temperament and character in adulthood. *Development and Psychopathology, 25*(03), 713-727. doi:10.1017/S0954579413000126

Kato, T. (2012). Development of the coping flexibility scale: Evidence for the coping flexibility hypothesis. *Journal of Counseling Psychology, 59*(2), 262. doi:10.1037/a0027770

Kessler, R. C., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Wittchen, H.-U. (2012). Twelve-month and lifetime prevalence and lifetime morbid risk of anxiety and mood disorders in the United States. *International Journal of Methods in Psychiatric Research, 21*(3), 169-184. doi:10.1002/mpr.1359

Khng, K. H., & Lee, K. (2014). The relationship between Stroop and stop-signal measures of inhibition in adolescents: Influences from variations in context and measure estimation. *PloS One, 9*(7), e101356. doi:10.1371/journal.pone.0101356

Kimbrel, N. A., Nelson-Gray, R. O., & Mitchell, J. T. (2007). Reinforcement sensitivity and maternal style as predictors of psychopathology. *Personality and Individual Differences, 42*(6), 1139-1149. doi:10.1016/j.paid.2006.06.028

Leen-Feldner, E. W., Zvolensky, M. J., Feldner, M. T., & Lejuez, C. W. (2004). Behavioral inhibition: Relation to negative emotion regulation and reactivity. *Personality and Individual Differences, 36*(6), 1235-1247. doi:10.1016/S0191-8869(02)00113-7

Lonigan, C. J., Vasey, M. W., Phillips, B. M., & Hazen, R. A. (2004). Temperament, anxiety, and the processing of threat-relevant stimuli. *Journal of Clinical Child and Adolescent Psychology, 33*(1), 8-20. doi:10.1207/S15374424JCCP3301_2

Maack, D. J., Tull, M. T., & Gratz, K. L. (2012). Examining the incremental contribution of behavioral inhibition to generalized anxiety disorder relative to other Axis I disorders and cognitive-emotional vulnerabilities. *Journal of Anxiety Disorders, 26*(6), 689-695. doi:10.1016/j.janxdis.2012.05.005

MacLeod, C., & Clarke, P. J. (2015). The attentional bias modification approach to anxiety intervention. *Clinical Psychological Science, 3*(1), 58-78. doi:10.1177/2167702614560749

Manfredi, C., Caselli, G., Rovetto, F., Rebecchi, D., Ruggiero, G. M., Sassaroli, S., & Spada, M. M. (2011). Temperament and parental styles as predictors of ruminative brooding and worry. *Personality and Individual Differences, 50*(2), 186-191. doi:10.1016/j.paid.2010.09.023

Mardaga, S., & Hansenne, M. (2009). Personality modulation of P300 wave recorded within an emotional oddball protocol. *Clinical Neurophysiology, 39*(1), 41-48. doi:10.1016/j.neucli.2008.12.005

Markarian, S. A., Pickett, S. M., Deveson, D. F., & Kanona, B. B. (2013). A model of BIS/BAS sensitivity, emotion regulation difficulties, and depression, anxiety, and stress symptoms in relation to sleep quality. *Psychiatry Research, 210*(1), 281-286. doi:10.1016/j.psychres.2013.06.004

Matthews, G., & Deary, I. J. (2000a). Personality: Performnce and information processing *Personality traits* (Repr. ed., pp. 220-242). Cambridge: Cambridge University Press.

Matthews, G., & Deary, I. J. (2000b). Stable traits and transient states *Personality traits* (Repr. ed., pp. 70-90). Cambridge: Cambridge University Press.

Matthews, G., Joyner, L., Gilliland, K., Huggins, J., & Falconer, S. (1999). Validation of a comprehensive stress state questionnaire: Towards a state big three? . In I. Merville, I. J. Deary, a. F. DeFruyt, & F. Ostendorf (Eds.), *Personality psychology in Europe* (pp. 335-350). Tilburg: Tilburg University

McLaughlin, K. A., & Nolen-Hoeksema, S. (2011). Rumination as a transdiagnostic factor in depression and anxiety. *Behaviour Research and Therapy, 49*(3), 186-193. doi:10.1016/j.brat.2010.12.006

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*(1), 49-100. doi:10.1006/cogp.1999.0734

Most, S. B., Chun, M. M., Johnson, M. R., & Kiehl, K. A. (2006). Attentional modulation of the amygdala varies with personality. *Neuroimage, 31*(2), 934-944. doi:10.1016/j.neuroimage.2005.12.031

Most, S. B., Chun, M. M., Widders, D. M., & Zald, D. H. (2005). Attentional rubbernecking: Cognitive control and personality in emotion-induced blindness. *Psychonomic Bulletin and Review, 12*(4), 654-661. doi:10.3758/bf03196754

Muris, P., Meesters, C., de Kanter, E., & Timmerman, P. E. (2005). Behavioural inhibition and behavioural activation system scales for children: relationships with Eysenck's personality traits and psychopathological symptoms. *Personality and Individual Differences, 38*(4), 831-841. doi:10.1016/j.paid.2004.06.007

Muris, P., Merckelbach, H., Schmidt, H., Gadet, B., & Bogie, N. (2001). Anxiety and depression as correlates of self-reported behavioural inhibition in normal adolescents. *Behaviour Research and Therapy, 39*(9), 1051-1061. doi:10.1016/S0005-7967(00)00081-4

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology. *Psychological Bulletin, 126*(2), 220-246. doi:10.1037/0033-2909.126.2.220

Nigg, J. T. (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 47*(3-4), 395-422. doi:10.1111/j.1469-7610.2006.01612.x

Orth, U., Berking, M., & Burkhardt, S. (2006). Self-conscious emotions and depression: Rumination explains why shame but not guilt is maladaptive. *Personality and Social Psychology Bulletin, 32*(12), 1608-1619. doi:10.1177/0146167206292958

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. *Journal of Clinical Psychology, 51*(6), 768-774. doi:10.1002/1097-4679(199511)51:6<768::Aid-Jclp2270510607>3.0.Co;2-1

Pickering, A. D. (1997). The conceptual nervous system and personality: From Pavlov to neural networks. *European Psychologist, 2*(2), 139. doi:10.1027/1016-9040.2.2.139

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*(3), 879-891. doi:10.3758/BRM.40.3.879

Quay, H. C. (1993). The psychobiology of undersocialized aggressive conduct disorder: A theoretical perspective. *Development and Psychopathology, 5*(1-2), 165-180. doi:10.1017/S0954579400004326

Reynolds, B., Ortengren, A., Richards, J. B., & de Wit, H. (2006). Dimensions of impulsive behavior: Personality and behavioral measures. *Personality and Individual Differences, 40*(2), 305-315. doi:10.1016/j.paid.2005.03.024

Roberts, W., Fillmore, M. T., & Milich, R. (2011). Separating automatic and intentional inhibitory mechanisms of attention in adults with attention-deficit/hyperactivity disorder. *Journal of Abnormal Psychology, 120*(1), 223-233. doi:10.1037/a0021408

Roisman, G. I., Masten, A. S., Coatsworth, J. D., & Tellegen, A. (2004). Salient and emerging developmental tasks in the transition to adulthood. *Child Development, 75*(1), 123-133. doi:10.1111/j.1467-8624.2004.00658.x

Rutter, M. (2007). Psychopathological development across adolescence. *Journal of youth and adolescence, 36*(1), 101-110.

Schieman, S. (1999). Age and anger. *Journal of Health and Social Behavior*, 273-289.

Schreiber, L. R., Grant, J. E., & Odlaug, B. L. (2012). Emotion regulation and impulsivity in young adults. *Journal of Psychiatry Research, 46*(5), 651-658. doi:10.1016/j.jpsychires.2012.02.005

Sheikh, S., & Janoff-Bulman, R. (2009). The "shoulds" and "should nots" of moral emotions: A self-regulatory perspective on shame and guilt. *Personality and Social Psychology Bulletin, 36*(2), 213-224. doi:10.1177/0146167209356788

Sheppes, G., Suri, G., & Gross, J. J. (2015). Emotion regulation and psychopathology. *Annual Review of Clinical Psychology, 11*(1), 379-405. doi:10.1146/annurev-clinpsy-032814-112739

Smits, D. J. M., & Kuppens, P. (2005). The relations between anger, coping with anger, and aggression, and the BIS/BAS system. *Personality and Individual Differences, 39*(4), 783-793. doi:10.1016/j.paid.2005.02.023

Sportel, B., Nauta, M., Hullu, E., Jong, P., & Hartman, C. (2011). Behavioral inhibition and attentional control in adolescents: Robust relationships with anxiety and depression. *Journal of Child and Family Studies, 20*(2), 149-156. doi:10.1007/s10826-010-9435-y

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in cognitive sciences, 9*(2), 69-74. doi:10.1016/j.tics.2004.12.005

Sukhodolsky, D. G., Golub, A., & Cromwell, E. N. (2001). Development and validation of the anger rumination scale. *Personality and Individual Differences, 31*(5), 689-700. doi:10.1016/S0191-8869(00)00171-9

Suveg, C., Hoffman, B., Zeman, J. L., & Thomassin, K. (2009). Common and specific emotion-related predictors of anxious and depressive symptoms in youth. *Child Psychiatry and Human Development, 40*(2), 223-239. doi:10.1007/s10578-008-0121-x. Epub 2008 Nov 28

Suveg, C., Morelen, D., Brewer, G. A., & Thomassin, K. (2010). The emotion dysregulation model of anxiety: A preliminary path analytic examination. *Journal of Anxiety Disorders, 24*(8), 924-930. doi:10.1016/j.janxdis.2010.06.018

Tackett, J. L. (2006). Evaluating models of the personality–psychopathology relationship in children and adolescents. *Clinical Psychology Review, 26*(5), 584-599. doi:10.1016/j.cpr.2006.04.003

Tamres, L. K., Janicki, D., & Helgeson, V. S. (2002). Sex differences in coping behavior: A meta-analytic review and an examination of relative coping. *Personality and Social Psychology Review, 6*(1), 2-30. doi:10.1207/s15327957pspr0601_1

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development, 59*(2/3), 25-52. doi:10.1111/j.1540-5834.1994.tb01276.x

Tortella-Feliu, M., Balle, M., & Sesé, A. (2010). Relationships between negative affectivity, emotion regulation, anxiety, and depressive symptoms in adolescents as examined through structural equation modeling. *Journal of Anxiety Disorders, 24*(7), 686-693. doi:10.1016/j.janxdis.2010.04.012

Tugade, M. M., & Fredrickson, B. L. (2004). Resilient individuals use positive emotions to bounce back from negative emotional experiences. *Journal of Personality and Social Psychology, 86*, 320-333.

Tull, M. T., Gratz, K. L., Latzman, R. D., Kimbrel, N. A., & Lejuez, C. W. (2010). Reinforcement sensitivity theory and emotion regulation difficulties: A multimodal investigation. *Personality and Individual Differences, 49*(8), 989-994. doi:10.1016/j.paid.2010.08.010

Weierich, M. R., Treat, T. A., & Hollingworth, A. (2008). Theories and measurement of visual attentional processing in anxiety. *Cognition and Emotion, 22*(6), 985-1018. doi:10.1080/02699930701597601

White, L. K., McDermott, J. M., Degnan, K. A., Henderson, H. A., & Fox, N. A. (2011). Behavioral inhibition and anxiety: The moderating roles of inhibitory control and attention shifting. *Journal of Abnormal Child Psychology, 39*(5), 735-747. doi:10.1007/s10802-011-9490-x

Whitmer, A. J., & Banich, M. T. (2007). Inhibition versus switching deficits in different forms of rumination. *Psychological Science, 18*(6), 546-553. doi:10.1111/j.1467-9280.2007.01936.x

Wittchen, H. U., Jacobi, F., Rehm, J., Gustavsson, A., Svensson, M., Jönsson, B., . . . Steinhausen, H. C. (2011). The size and burden of mental disorders and other disorders of the brain in Europe

2010. *European Neuropsychopharmacology, 21*(9), 655-679. doi:10.1016/j.euroneuro.2011.07.018

Yap, M. B., Allen, N. B., & Sheeber, L. (2007). Using an emotion regulation framework to understand the role of temperament and family processes in risk for adolescent depressive disorders. *Clinical Child and Family Psychology Review, 10*(2), 180-196.

Zetsche, U., D'Avanzato, C., & Joormann, J. (2012). Depression and rumination: Relation to components of inhibition. *Cognition and Emotion, 26*(4), 758-767. doi:10.1080/02699931.2011.613919

Zhang, W., Lu, J., Ni, Z., Liu, X., Wang, D., & Shen, J. (2013). Harm avoidance in adolescents modulates late positive potentials during affective picture processing. *International Journal of Developmental Neuroscience, 31*(5), 297-302. doi:10.1016/j.ijdevneu.2013.03.009

Zlomke, K. R., & Hahn, K. S. (2010). Cognitive emotion regulation strategies: Gender differences and associations to worry. *Personality and Individual Differences, 48*(4), 408-413. doi:10.1016/j.paid.2009.11.007

Appendix 1

1. Izadpanah, S., Schumacher, M., Bähr, A., Stopsack, M., Grabe, H. J., & Barnow, S. (2016). A 5-year longitudinal study of the adolescent reinforcement sensitivity as a risk factor for anxiety symptoms in adulthood: Investigating the indirect effect of cognitive emotion regulation. *Personality and Individual Differences, 95*, 68-73. doi: 10.1016/j.paid.2016.02.021

Personality and Individual Differences 95 (2016) 68–73

Contents lists available at ScienceDirect

Personality and Individual Differences

journal homepage: www.elsevier.com/locate/paid

A 5-year longitudinal study of the adolescent reinforcement sensitivity as a risk factor for anxiety symptoms in adulthood: Investigating the indirect effect of cognitive emotion regulation

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ARTICLE INFO

Article history:
Received 5 August 2015
Received in revised form 18 January 2016
Accepted 8 February 2016
Available online 18 February 2016

Keywords:
Behavioral Inhibition System
Behavioral Activation System
Emotion regulation
Anxiety
Reinforcement Sensitivity Theory

ABSTRACT

This study investigated the longitudinal effects of the Behavioral Inhibition System (BIS) and the Behavioral Activation System (BAS) on anxiety symptomatology and tested the indirect effect of cognitive emotion regulation as a possible mechanism underlying this link. In this study, 274 individuals were assessed two times (T_1 and T_2), at a 5-year interval. We found an excellent fit for the hypothesized model, with BIS (T_1) predicting both maladaptive cognitive emotion regulation (mCER) and T_2-anxiety even after controlling for T_1-anxiety. Further, mCER significantly mediated the relationship between BIS and T_2-anxiety, and between T_1-anxiety and T_2-anxiety. However, an alternative model, supposing that BIS and T_1-anxiety indirectly affect mCER through T_2-anxiety, showed a similar fit. While BAS predicted higher levels of adaptive cognitive emotion regulation (aCER), it was unrelated to mCER and showed a small positive association with anxiety only at higher levels of BIS. These findings provide longitudinal support for BIS as a risk for anxiety symptoms and support the importance of targeting mCER in the prevention and treatment of anxiety, especially among individuals with BIS sensitivity. Finally, the results suggest a possible overlap between anxiety and mCER that requires further longitudinal research to clarify the direction of their relationship.

1. Introduction

The Reinforcement Sensitivity Theory (RST), proposed by Gray (1982), is considered a milestone in personality research and significantly contributed to a consensus on the association between personality factors and emotional systems (Pickering & Corr, 2008). RST postulates that three major brain subsystems, the Behavioral Approach System (BAS), the Behavioral Inhibition System (BIS) and the Fight–Flight System (FFS), are responsible for individual differences in personality and psychopathology (Gray, 1982). In this model, BAS is defined as a sensitivity to reward signals, whereas BIS is characterized by sensitivity to aversive stimuli (signals of punishment, non-reward and novelty), is activated by potential threats and underlies anxiety (Corr & McNaughton, 2008; Gray, 1982). Anxiety symptoms have been positively associated with BIS but unrelated to BAS (e.g., Hundt, Williams, Mendelson, & Nelson-Gray, 2013). BIS resolves approach-avoidance conflicts by increasing the valence of negative stimuli. This leads to a subjective state of worry and constant checking of the environment for potential signs of danger, which in turn contributes to anxiety (Pickering & Corr, 2008), as supported by previous empirical evidence (e.g., Maack, Tull, & Gratz, 2012).

1.1. BIS/BAS, emotion dysregulation, and anxiety

The underlying mechanism through which BIS leads to anxiety is largely unknown. Research suggests that emotion dysregulation is a possible explanation for this link (Bijttebier, Beck, Claes, & Vandereycken, 2009). Accordingly, previous cross-sectional studies have shown that BIS is associated with more emotion dysregulation among young adults (Leen-Feldner, Zvolensky, Feldner, & Lejuez, 2004; Tull, Gratz, Latzman, Kimbrel, & Lejuez, 2010). Markarian, Pickett, Deveson, and Kanona (2013) showed that emotion dysregulation mediates the relationship between BIS and anxiety. These findings are consistent with current theories on BIS, which link this construct with a variety of emotionally negative outcomes (Gray, 1982). Higher levels of negative emotions associated with BIS (Hundt, Brown, Kimbrel, Walsh, Nelson-Gray and Kwapil, 2013) might facilitate emotion dysregulation (Fox, Henderson, Marshall,

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http://dx.doi.org/10.1016/j.paid.2016.02.021
0191-8869/

Nichols, & Ghera, 2005). In contrast, BAS has been related to higher levels of positive affect (Hundt, Brownet al., 2013), but it has shown an insignificant or small negative association with emotion dysregulation (Markarian et al., 2013).

The existing literature on the link between BIS/BAS and emotion dysregulation has mostly focused on emotion regulation difficulties such as awareness as measured with the Difficulties in Emotion Regulation Scale (DERS, Gratz & Roemer, 2004), while particular emotion regulation strategies are strongly associated with psychopathology (Garnefski, Kraaij, & van Etten, 2005). In this study, we focus on a definition of emotion regulation as cognitive strategies for handling the intake of emotionally arousing information and ways of responding to stressful events (Garnefski, Kraaij, & Spinhoven, 2001). Maladaptive cognitive emotion regulation strategies (mCER) such as self-blame, rumination, catastrophizing, and suppression have been shown to positively predict anxiety (Garnefski et al., 2005), while adaptive cognitive emotion regulation (aCER) such as acceptance and positive refocusing, have a marginal or non-significant association with anxiety symptomatology (Garnefski & Kraaij, 2007). The association between aCER and psychopathology seems to be weaker, less constant and more dependent on the context, compared to mCER (Aldao & Nolen-Hoeksema, 2012).

While we did not identify a study investigating the association between BIS/BAS and aCER, some studies have shown that BIS predicts higher levels of mCER. These, however, are cross-sectional and focus only on rumination (Leen-Feldner et al., 2004; Randles, Flett, Nash, McGregor, & Hewitt, 2010). The negative affect associated with BIS (Hundt, Brown et al., 2013) might contribute to negative cognitions such as mCER (Mausbach, Roepke, Depp, Patterson, & Grant, 2009). Further, BIS may lead to cognitive intrusions due to the increased sensitivity to punishment signals and constant checking of the environment for potential threats (Nigg, 2000), which in turn facilitate mCER such as rumination. Accordingly, Viana and Gratz (2012) demonstrated that catastrophizing explains the BIS-anxiety link among adolescents.

Although different lines of research relate both BIS and emotion dysregulation to anxiety, we know very little about their concomitant relations to anxiety symptoms. Such studies are of special importance considering findings on emotion dysregulation as a risk and maintaining factor, as well as a treatment target for anxiety disorders (Cisler, Olatunji, Feldner, & Forsyth, 2010).

1.2. The present study

This study addresses the aforementioned gap using structural equation modeling (SEM) to test the direct and indirect effects of BIS/BAS on anxiety symptoms. We hypothesized that BIS predicts higher levels of anxiety and mCER, when measured after a 5-year interval, and that BAS is less strongly related to mCER and anxiety. Further, we assumed that mCER mediates the relationship between BIS and anxiety, while aCER is only insignificantly or weakly related to both BIS and anxiety. Additionally, given the evidence for higher levels of BIS (Markarian et al., 2013), mCER (Garnefski & Kraaij, 2006), and anxiety (Viana & Gratz, 2012) among women, we controlled for the gender effect.

2. Materials and methods

2.1. Participants and procedure

The sample was drawn from the population-based Greifswald family study (Aldinger et al., 2014; Barnow, Lucht, & Freyberger, 2002; Barnow, Rüge, Spitzer, & Freyberger, 2005), a subpopulation from the Study of Health in Pomerania, Germany (SHIP; John et al., 2001). Longitudinal data were collected three times, at 5-year intervals, the second and the third of which (T_1 and T_2) were used in this study.

Between 1997 and 2000 (T_0), 381 offspring from 315 families participated in the study. Between 2005 and 2008, the first follow-up (T_1) was conducted with 334 participants (mean age = 19.56). From 2011 to 2013 (T_2), the participants were investigated again. Data for 85% of T_1 participants were available from this assessment ($N = 284$). Individuals who participated in T_2 did not differ in age from those who dropped out after T_1 ($F = 0.07$, $p = .79$). There was an insignificant tendency to more dropouts among men ($\chi = 3.50$, $p = .061$) and individuals who did not follow the T_2 assessment had significantly lower BIS ($F = 4.77$, $p = .03$) and depression ($F = 8.43$, $p = .004$), and higher BAS ($F = 4.43$, $p = .04$) at T_1. Further, 10 individuals with missing values for at least one relevant variable, were excluded from the analysis, resulting in a final sample of 274 participants (154 women and 120 men) with a mean age of 19.50 years (14–27) at T_1 and 24.99 years (19–34) at T_2 (see Table 1). All participants provided written informed consent and the study was approved by the local ethics committee.

2.2. Measures

2.2.1. BIS/BAS sensitivity

At T_1, BIS/BAS sensitivity was measured with the short version of Action Regulating Emotion Systems (ARES; Hartig & Moosbrugger, 2003). The ARES is a German alternative to the Behavioral Inhibition/Activation System scales (Carver & White, 1994) and consists of a 10-item BIS and a 10-item BAS, ranging from 1 (*strong disagreement*) to 4 (*strong agreement*). Both BIS and BAS subscales show good internal consistency ($\alpha = .89$ and $\alpha = .80$, respectively; Hartig & Moosbrugger, 2003).

2.2.2. Symptom checklist-revised (SCL-90-R) and brief symptom inventory (BSI)

T_1-anxiety and T_2-anxiety were measured with the German version of the SCL-90-R (Franke, 1995) and its short form, the BSI (Franke, 2000), respectively. Items are rated on a five-point Likert scale, ranging from *not at all* (0) to *extremely* (4). Both versions are comparable and measure psychopathology with nine scales assessing symptoms over the last seven days (Franke, 1995, 2000). SCL-90-R and BSI have shown excellent reliability and validity ($\alpha = .965$ and $\alpha = .963$, respectively) (Franke, 2000; Hessel, Schumacher, Geyer, & Brähler, 2001) and their anxiety subscales show good stability over one week ($r = 0.85$ and $r = 0.88$, respectively; Franke, 1995, 2000). In order to facilitate comparability of T_1-anxiety and T_2-anxiety, we extracted BSI items from SCL-90-R and summed them to produce the T_1-anxiety score.

2.2.3. The Cognitive Emotion Regulation Questionnaire (CERQ)

The CERQ (Garnefski et al., 2001), which consists of 36 Likert-type items ranging from *sometimes* (1) to *always* (5), was applied at T_2. The CERQ measures cognitive strategies of self-blame, rumination, catastrophizing, other-blame, acceptance, positive reappraisal, positive refocusing, planning, and putting into perspective. It has shown adequate internal consistency ($.60 < \alpha < .86$) and an acceptable to good test–retest reliability ($.65 < r < .83$), except for the "blaming others" and "positive refocusing" ($r = .51$ and $r = .48$, respectively; Loch, Hiller, & Witthöft, 2011).

2.3. Statistical analysis

We analyzed data using IBM SPSS version 20 and analysis of movement structure (AMOS) version 22. We analyzed descriptive statistics for each variable and calculated Pearson correlation coefficients between the variables. Using SEM, we designed and tested the hypothesized model in AMOS with a 95% confidence interval and using the following fit indices: an insignificant chi-square, chi-square/*df* ratio < 2.0, Comparative Fit Index (CFI) > .90, Goodness of Fit Index (GFI) > .90 and Root Mean Square Error of Approximation (RMSEA) < .08 (Tabachnick & Fidell, 2007). We conducted a curve estimation for all the relationships in our model and determined that all were sufficiently linear to be tested using covariance-based SEM. In keeping

Table 1
Means and standard deviations for study variables.

Variables	Total ($N = 274$)	Female ($n = 154$)	Male ($n = 120$)	F	η^2
	M (SD)	M (SD)	M (SD)		
Age (T_1)	19.50 (2.35)	19.70 (2.39)	19.23 (2.28)	2.69	.01
Age (T_2)	24.99 (2.42)	25.16 (2.46)	24.77 (2.36)	1.81	.01
ARES-BIS (T_1)	1.36 (.52)	1.46 (.59)	1.22 (.39)	14.84***	.05
ARES-BAS (T_1)	2.25 (.38)	2.33 (.35)	2.15 (.40)	16.73***	.06
Anxiety					
SCL-90-Anxiety (T_1)[a]	2.27 (2.51)	2.72 (2.75)	1.68 (2.03)	11.97***	.04
BSI-Anxiety (T_2)	2.00 (2.71)	2.27 (3.19)	1.67 (1.89)	3.33	.01
Δ Anxiety[b]	−0.26 (2.77)	−0.45 (3.14)	−0.02 (2.21)	1.68	.01
CERQ-maladaptive (T_2)					
Self-blame	3.02 (2.15)	3.20 (2.39)	2.80 (1.79)	2.36	.01
Rumination	4.21 (2.71)	4.617(2.80)	3.63 (2.47)	10.39***	.04
Catastrophizing	2.30 (2.19)	2.56 (2.27)	1.96 (2.03)	5.26*	.02
Blaming others	2.05 (2.05)	2.10 (2.21)	1.98 (1.84)	0.23	.001
Total maladaptive	11.59 (6.58)	12.54 (7.06)	10.37 (5.73)	7.51**	.03
CERQ-adaptive (T_2)					
Acceptance	6.34 (2.75)	6.21 (2.75)	6.50 (2.76)	.76	.003
Positive refocusing	3.58 (2.36)	3.90 (2.39)	3.17 (2.26)	6.69**	.02
Planning	7.05 (2.81)	6.97 (2.87)	7.14 (2.76)	.24	.001
Positive reappraisal	5.54 (2.76)	5.47 (2.98)	5.63 (2.46)	.20	.001
Putting into perspective	5.91 (2.73)	5.92 (2.78)	5.91 (2.68)	.00	.00
Total adaptive	28.42 (9.34)	28.47 (9.71)	28.34 (8.88)	.01	.91

The first assessment (T_1); the second assessment (T_2); Eta-squared (η^2); Action Regulating Emotion Systems (ARES); Behavioral Inhibition System (BIS); Behavioral Activation System (BAS); Symptom Checklist-90 (SCL-90); Brief Symptom Inventory (BSI); Cognitive Emotion Regulation Questionnaire (CERQ).
[a] BSI items were extracted and summed.
[b] Difference between T_2-anxiety and T_1-anxiety.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

with Preacher and Hayes (2008), we determined the significance of the indirect effect using bootstrapping with 2000 resamples.

3. Results

3.1. Descriptive statistics and gender comparisons

90.1% of our participants were single, 9.1% were married, and 0.8% were divorced. Table 1 shows the descriptive statistics of all study variables. Women had significantly higher scores on BIS, BAS, rumination, catastrophizing, positive refocusing and T_2-anxiety. In addition, 12 participants had anxiety scores within the clinical range at both T_1 and T_2 ($t \geq 63$, see Franke, 2000).

3.2. Correlation coefficients

Consistent with our hypothesis, BIS showed a strong positive association with all mCER strategies and both T_1 and T_2-anxiety. Further, in line with our expectations, mCER strategies were positively associated with T_1 and T_2-anxiety (Table 2) while aCER strategies were unrelated. In other words, individuals with a greater tendency to use mCER had higher cross-sectional and longitudinal anxiety scores. BAS was

Table 2
Pearson correlation coefficients between study variables.

Variables	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
1 ARES-BIS (T_1)	−.21**	.35**	.33**	−.01	.19*	.18*	.23**	.20**	.27**	−.00	.03	−.07	−.15*	−.08	−.08
2 ARES-BAS (T_1)		−.05	.01	.06	−.02	.04	−.02	−.02	−.01	−.03	.06	.13*	.17**	.17**	.15*
3 SCL-90-anxiety (T_1)[a]			.44***	−.52***	.29***	.24***	.27***	.23***	.35***	−.04	.06	−.00	−.11	−.08	−.05
4 BSI-anxiety (T_2)				.54***	.41***	.41***	.39***	.31***	.53***	.03	.01	.07	−.02	−.06	.01
5 Δ anxiety[b]					.12*	.16**	.11	.07	.17**	.06	−.05	.07	.08	.02	.05
6 CERQ-self-blame						.39***	.42***	.08	.65***	.20***	.04	.14*	.00	−.05	.10
7 CERQ-rumination							.53***	.29***	.80***	.36***	.07	.29***	.21***	.11	.31***
8 CERQ-catastrophizing								.43**	.82**	.13*	.01	.17**	−.05	−.21***	.02
9 CERQ-blaming others									.60***	.22***	.12*	.20***	.09	−.01	.18**
10 CERQ-total maladaptive (T_2)										.32***	.08	.29***	.10	−.04	.22***
11 CERQ-acceptance											.23***	.43***	.41***	.32***	.70***
12 CERQ-positive refocusing												.10	.25***	.32***	.52***
13 CERQ-planning													.51***	.33***	.70***
14 CERQ-positive reappraisal														.58***	.80***
15 CERQ-putting into perspective															.74***
16 CERQ-total adaptive (T_2)															

The first assessment (T_1); the second assessment (T_2); Action Regulating Emotion Systems (ARES); Behavioral Inhibition System (BIS); Behavioral Activation System (BAS); Symptom Checklist-90 (SCL-90); Brief Symptom Inventory (BSI); Cognitive Emotion Regulation Questionnaire (CERQ).
[a] BSI items were extracted and summed.
[b] Difference between T2-anxiety and T1-anxiety.
* $p < .05$.
** $p < .01$.
*** $p < .001$.

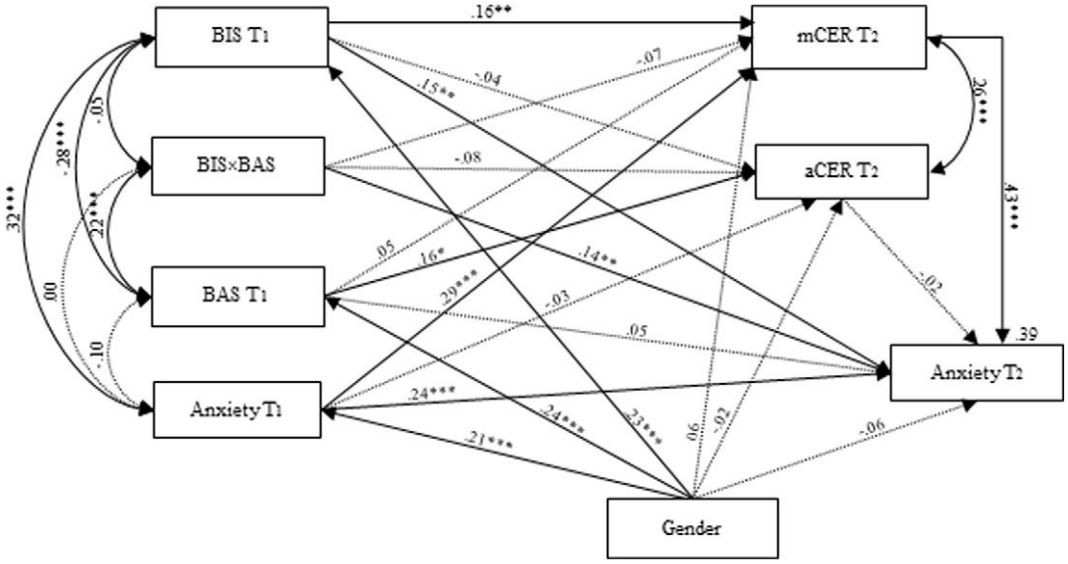

Fig. 1. Standardized coefficients for the hypothesized model linking BIS/BAS to anxiety, mCER, and aCER. The first assessment (T_1); the second assessment (T_2); Behavioral Inhibition System (BIS); Behavioral Activation System (BAS); maladaptive cognitive emotion regulation (mCER); adaptive cognitive emotion regulation (aCER). Dashed arrows represent insignificant paths. $^{*}p < .05$.$^{**}p < .01$.$^{***}p < .001$.

positively correlated with planning, positive reappraisal, and putting into perspective but unrelated to anxiety and mCER. Age was not significantly associated with other variables.

3.3. Testing the model

Fig. 1 illustrates the hypothesized model with the standardized regression weights. In general, we tested the model with the assumption that BIS would directly affect both mCER and T_2-anxiety, and indirectly affects T_2-anxiety through mCER, when controlling for gender. We were also interested in how aCER, BAS, and BAS × BIS interaction were related to other variables in the model. The model perfectly fitted the data: $\chi^2(1, N = 174) = 0.09$, $p = 0.77$, $\chi^2/df = .09$, RMSE = 0.00, CFI = 1.00, GFI = 1.00). As we expected, there was a significant path between BIS and both mCER (B = 2.07, bootstrap $SE = .78$, $p = .008$) and T_2-anxiety (B = 0.79, bootstrap $SE = 0.28$, $p = .004$). In addition, both mCER (B = 0.18, bootstrap $SE = .02$, $p < .001$) and T_1-anxiety (B = 0.26, bootstrap $SE = .06$, $p < .001$) significantly predicted T_2-anxiety. Further, T_1-anxiety significantly predicted mCER (B = 0.76, bootstrap $SE = .16$, $p < .001$). There was a significant path between gender and BIS (B = 0.24, bootstrap $SE = .06$, $p < .001$), BAS (B = 0.18, bootstrap $SE = .04$, $p < .001$), and T_1-anxiety (B = 1.04, bootstrap $SE = .30$, $p < .001$). We also found a significant interaction between BIS and BAS, where BAS predicted higher levels of T_2-anxiety, but only at higher levels of BIS (B = 0.27, bootstrap $SE = .10$, $p < 0.01$). The results of the bootstrap analysis showed that mCER significantly mediated the relationship between BIS and T_2-anxiety (B = 0.38, bootstrap $SE = 0.18$, bootstrap CI = 0.09–0.83, $p = .01$), as well as the relationship between T_1-anxiety and T_2-anxiety (B = 0.14, bootstrap $SE = 0.05$, bootstrap CI = 0.07–0.25, $p < .001$).

Although our hypothesized model was based on prior theories and empirical findings, given the cross-sectional measurement of mCER and anxiety, we tested an alternative model where anxiety mediates the relationship between BIS and mCER. We assumed that mCER might itself be an artifact of anxiety (Campbell-Sills, Ellard, & Barlow, 2014); this was consistent with the significant path between T_1-anxiety and mCER. Fit indices of the alternative model were as good as our hypothesized model ($\chi^2(1, N = 174) = .09$, $P = 0.76$, $\chi^2/df = .09$, RMSE = 0.00, CFI = 1.00, GFI = 1.00). We also found that T_2-anxiety mediated the relationship between BIS and mCER (B = 1.31, bootstrap $SE = 0.49$, bootstrap CI = 0.46–2.53, $p = .002$), as well as the relationship between T_1-anxiety and mCER (B = 0.44, bootstrap $SE = 0.13$, bootstrap CI = 0.23–0.75, $p = .001$).

4. Discussion

The current study investigated BIS/BAS sensitivity in relation to anxiety symptoms and emotion regulation over a 5-year period. We hypothesized that BIS (at T_1) predicts higher scores on both mCER and anxiety after 5 years (T_2). In addition, we assumed that BIS predicts anxiety indirectly through mCER. Further, we expected BAS and aCER to be unrelated or weakly associated with anxiety.

Our first main finding that BIS significantly predicts T_2-anxiety, even after controlling for T_1-anxiety, supports previous evidence (e.g., Sportel, Nauta, Hullu, Jong, & Hartman, 2011). Dispositional factors such as behavioral inhibition might make individuals more vulnerable to later development of anxiety when facing life tasks and during learning procedures (Mineka & Zinbarg, 2006). The period between late adolescence and young adulthood is an important developmental phase that requires making important decisions regarding educational, occupational and emotional aspects of life (Roisman, Masten, Coatsworth, & Tellegen, 2004). While adolescents face novel and stressful situations that demand an increased level of regulatory effort (Garnefski & Kraaij, 2006), fear of approaching novel situations associated with BIS might pose a risk of developing anxiety symptoms in response to the multidimensional tasks common to this life stage.

Further, the results support our second hypothesis and provide longitudinal evidence for a positive direct link between BIS and various mCER. This is in line with previous findings that showed that BIS is related to higher levels of emotion dysregulation (Hannan & Orcutt, 2013) and mCER strategy rumination (Randles et al., 2010). BIS sensitivity might increase mCER by provoking concerns regarding potential threats and might facilitate catastrophizing due to associated oversensitivity to situations of non-reward or punishment. In addition, mCER might be applied as a problem-solving strategy (Aldao & Nolen-Hoeksema, 2010) or as an attempt to understand negative emotions associated with BIS.

Further, we found that BIS leads to higher levels of anxiety through mCER. Similarly, one study showed that mCER mediates the relationship between punishment sensitivity and anxiety (Tortella-Feliu, Balle, & Sesé, 2010). This finding supports previous evidence on the positive link between emotion dysregulation and anxiety (e.g., Suveg, Morelen,

Brewer, & Thomassin, 2010). In another study, Markarian et al. (2013) reported that emotion dysregulation mediates the relationship between BIS and anxiety. However, they did not test an alternative model investigating the indirect effect of anxiety on BIS and emotion dysregulation. We investigated this alternative model, with BIS leading to higher scores on mCER through T_2-anxiety, which yielded a similar fit. This finding questions the previous correlational findings that overlooked this alternative model on the relationship between anxiety and emotion dysregulation (see Cisler et al., 2010). Anxiety and mCER might overlap or may have a mutual effect on each other, where intense emotions associated with anxiety facilitate mCER and using mCER contributes to higher levels of anxiety (Campbell-Sills et al., 2014). In addition, results might differ for different psychopathologies.

Next, our findings showed that BAS, consistent with Gary's conceptualization of BAS as an impulsivity dimension (Gray, 1994), was unrelated to anxiety. However, similar to Hundt, Nelson-Gray, Kimbrel, Mitchell, and Kwapil (2007), we found that BAS predicted T_2-anxiety only at high levels of BIS. Adolescents with high BIS and BAS might experience more approach-avoidance conflicts that lead to higher levels of distress and anxiety symptoms (Hundt et al., 2007). Further, in line with previous findings (Tull et al., 2010), BAS was unrelated to mCER, but was positively correlated with aCER such as planning, positive reappraisal, and putting into perspective. Similarly, Hasking (2006) found a positive association between BAS and problem solving. Higher levels of positive affect associated with BAS (Hundt, Brown et al., 2013) might facilitate adaptive emotion regulation.

The current study has several limitations. First, our findings were based on self-report data and could benefit from reevaluation using other measurement methods like ecological momentary assessment. A more robust measurement of anxiety including other instruments and measuring specific anxiety symptoms would complement our findings. Second, we had only one measurement for mCER, while multiple assessments of anxiety and mCER help clarifying the direction of anxiety-mCER association. Third, future studies should examine the generalizability and magnitude of our findings through replicating the study with other samples (e.g., clinical samples). Fourth, we applied the ARES that does not measure BIS and FFS separately. Given that current theories of RST distinguish these two systems (Corr & McNaughton, 2008), future studies should apply instruments based on the revised RST (see Corr, 2016) to show whether BIS and FFS have different effects on emotion regulation and anxiety. Finally, while our study focused on better understanding anxiety symptoms, future studies could investigate the generalizability of this model to other psychopathology symptoms.

Our findings highlight the importance of considering emotion regulation for the prediction and treatment of anxiety among individuals with BIS sensitivity. Previous evidence indicates that not all behaviorally inhibited children develop anxiety symptoms (Gladstone, Parker, Mitchell, Wilhelm, & Malhi, 2005). Given the relatively early development of BIS (Kagan, 2008) and the malleability of emotion regulation (Barnow, Löw, Dodek, & Stopsack, 2014), mCER might be a pathway for anxiety development and, therefore, an ideal treatment target among those with BIS sensitivity. Mindfulness-based techniques could be a good treatment option considering their influence on reducing negative emotional reactivity and emotion dysregulation (Shapiro, Carlson, Astin, & Freedman, 2006). Some authors argue that mindfulness might reduce mCER through facilitating acceptance of negative emotional reactivity associated with BIS (Markarian et al., 2013).

5. Conclusion

The current study provides evidence for mCER as an underlying mechanism of the link between BIS and anxiety. Findings contribute to the field by adding longitudinal evidence to previous correlational findings and including various emotion regulation strategies. Results highlight the need for further longitudinal studies to investigate the direction of the relationship between mCER and anxiety.

Acknowledgments and conflicts of interest

The Greifswald family study was supported by the German Research Foundation (Deutsche Forschungsgemeinschaft, BA-2172/2–1, BA2172.8–1. GR1912.7–1).

References

Aldao, A., & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: a transdiagnostic examination. *Behaviour Research and Therapy*, *48*, 974–983.
Aldao, A., & Nolen-Hoeksema, S. (2012). When are adaptive strategies most predictive of psychopathology? *Journal of Abnormal Psychology*, *121*, 276–281.
Aldinger, M., Stopsack, M., Ulrich, I., Reinelt, E., Lang, S., & Barnow, S. (2014). Neuroticism developmental courses–implications for depression, anxiety and everyday emotional experience: A prospective study from adolescence to young adulthood. *BMC Psychiatry*, *14*, 210.
Barnow, S., Löw, C. A., Dodek, A., & Stopsack, M. (2014). Gefühle im Griff–Emotionen intelligent regulieren [managing emotions–emotions under control]. *Psychotherapie, Psychosomatik, Medizinische Psychologie*, *64*, 284–289.
Barnow, S., Lucht, M., & Freyberger, H. J. (2002). Alkoholprobleme im Jugendalter unter Berücksichtigung der Hochrisikogruppe Kinder alkoholkranker Eltern: Ergebnisse einer Familienstudie in Mecklenburg Vorpommern [alcohol problems in adolescence, taking the high risk group of children with alcoholic parents into account]. *Der Nervenarzt*, *73*, 671–679.
Barnow, S., Rüge, J., Spitzer, C., & Freyberger, H. J. (2005). Temperament und Charakter bei Personen mit Borderline-Persönlichkeitsstörung [temperament and character in persons with borderline personality disorder]. *Der Nervenarzt*, *76*, 839–848.
Bijttebier, P., Beck, I., Claes, L., & Vandereycken, W. (2009). Gray's reinforcement sensitivity theory as a framework for research on personality–psychopathology associations. *Clinical Psychology Review*, *29*, 421–430.
Campbell-Sills, L., Ellard, K. K., & Barlow, D. H. (2014). Emotion regulation in anxiety disorders. In J. J. Gross (Ed.), *Handbook of emotion regulation* (pp. 393–412) (2nd ed.). New York, NY: Guilford Press.
Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology*, *67*, 319–333.
Cisler, J., Olatunji, B., Feldner, M., & Forsyth, J. (2010). Emotion regulation and the anxiety disorders: An integrative review. *Journal of Psychopathology and Behavioral Assessment*, *32*, 68–82.
Corr, P. J. (2016). Reinforcement sensitivity theory of personality questionnaires: Structural survey with recommendations. *Personality and Individual Differences*, *89*, 60–64.
Corr, P. J., & McNaughton, N. (2008). Reinforcement sensitivity theory and personality. In P. J. Corr (Ed.), *The reinforcement theory of personality* (pp. 155–187). Cambridge: Cambridge University Press.
Fox, N. A., Henderson, H. A., Marshall, P. J., Nichols, K. E., & Ghera, M. M. (2005). Behavioral inhibition: Linking biology and behavior within a developmental framework. *Annual Review of Psychology*, *56*, 235–262.
Franke, G. H. (1995). *SCL-90-R: The symptom-checklist from Derogatis: German version.* Göttingen: Beltz Test.
Franke, G. H. (2000). *BSI: Brief symptom inventory from Derogatis (short form of SCL-90-R); German version; manual: Beltz test.*
Garnefski, N., & Kraaij, V. (2006). Relationships between cognitive emotion regulation strategies and depressive symptoms: A comparative study of five specific samples. *Personality and Individual Differences*, *40*, 1659–1669.
Garnefski, N., & Kraaij, V. (2007). The cognitive emotion regulation questionnaire. *European Journal of Psychological Assessment*, *23*, 141–149.
Garnefski, N., Kraaij, V., & Spinhoven, P. (2001). Negative life events, cognitive emotion regulation and emotional problems. *Personality and Individual Differences*, *30*, 1311–1327.
Garnefski, N., Kraaij, V., & van Etten, M. (2005). Specificity of relations between adolescents' cognitive emotion regulation strategies and internalizing and externalizing psychopathology. *Journal of Adolescence*, *28*, 619–631.
Gladstone, G. L., Parker, G. B., Mitchell, P. B., Wilhelm, K. A., & Malhi, G. S. (2005). Relationship between self-reported childhood behavioral inhibition and lifetime anxiety disorders in a clinical sample. *Depression and Anxiety*, *22*, 103–113.
Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment*, *26*, 41–54.
Gray, J. A. (1982). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system.* New York, NY: Oxford University Press.
Gray, J. A. (1994). Framework for a taxonomy of psychiatric disorder. In S. H. M. Van Goozen, N. E. Van de Poll, & J. A. Sergeant (Eds.), *Emotions: Essays on emotion theory* (pp. 29–59). Hillsdale, NJ: Erlbaum.
Hannan, S. M., & Orcutt, H. K. (2013). Emotion dysregulation as a partial mediator between reinforcement sensitivity and posttraumatic stress symptoms. *Personality and Individual Differences*, *55*, 574–578.
Hartig, J., & Moosbrugger, H. (2003). The ARES-Scales for measurment of BIS and BAS sensitivity. *Zeitschrift für Differentielle und Diagnostische Psychologie*, *24*, 293–310.

Hasking, P. A. (2006). Reinforcement sensitivity, coping, disordered eating and drinking behaviour in adolescents. *Personality and Individual Differences*, *40*, 677–688.
Hessel, A., Schumacher, J., Geyer, M., & Brähler, E. (2001). Symptom-checklist SCL-90-R: Validation and standardization based on a representative sample of the German population. *Diagnostica*, *47*, 27–39.
Hundt, N. E., Brown, L. H., Kimbrel, N. A., Walsh, M. A., Nelson-Gray, R., & Kwapil, T. R. (2013a). Reinforcement sensitivity theory predicts positive and negative affect in daily life. *Personality and Individual Differences*, *54*, 350–354.
Hundt, N. E., Nelson-Gray, R. O., Kimbrel, N. A., Mitchell, J. T., & Kwapil, T. R. (2007). The interaction of reinforcement sensitivity and life events in the prediction of anhedonic depression and mixed anxiety-depression symptoms. *Personality and Individual Differences*, *43*, 1001–1012.
Hundt, N. E., Williams, A. M., Mendelson, J., & Nelson-Gray, R. (2013b). Coping mediates relationships between reinforcement sensitivity and symptoms of psychopathology. *Personality and Individual Differences*, *54*, 726–731.
John, U., Hensel, E., Lüdemann, J., Piek, M., Sauer, S., Adam, C., Born, G., ... Kessler, C. (2001). Study of Health in Pomerania (SHIP): A health examination survey in an east German region: Objectives and design. *Sozial- und Praventivmedizin*, *46*, 186–194.
Kagan, J. (2008). Behavioral inhibition as a risk factor for psychopathology. In T. P. Beauchaine, & S. P. Hinshaw (Eds.), *Child and adolescent psychopathology* (pp. 157–179). Hoboken, NJ: Wiley.
Leen-Feldner, E. W., Zvolensky, M. J., Feldner, M. T., & Lejuez, C. W. (2004). Behavioral inhibition: Relation to negative emotion regulation and reactivity. *Personality and Individual Differences*, *36*, 1235–1247.
Loch, N., Hiller, W., & Witthöft, M. (2011). The cognitive emotion regulation questionnaire (CERQ). *Zeitschrift für Klinische Psychologie und Psychotherapie*, *40*, 94–106.
Maack, D. J., Tull, M. T., & Gratz, K. L. (2012). Examining the incremental contribution of behavioral inhibition to generalized anxiety disorder relative to other Axis I disorders and cognitive-emotional vulnerabilities. *Journal of Anxiety Disorders*, *26*, 689–695.
Markarian, S. A., Pickett, S. M., Deveson, D. F., & Kanona, B. B. (2013). A model of BIS/BAS sensitivity, emotion regulation difficulties, and depression, anxiety, and stress symptoms in relation to sleep quality. *Psychiatry Research*, *210*, 281–286.
Mausbach, B. T., Roepke, S. K., Depp, C. A., Patterson, T. L., & Grant, I. (2009). Specificity of cognitive and behavioral variables to positive and negative affect. *Behaviour Research and Therapy*, *47*, 608–615.
Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders. *American Psychologist*, *61*, 10–26.
Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin*, *126*, 220–246.
Pickering, A. D., & Corr, P. (2008). J.A.Gray's reinforcement sensitivity theory of personality. *The Sage handbook of personality theory and assessment* (pp. 239–256). London: Sage.
Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, *40*, 879–891.
Randles, D., Flett, G. L., Nash, K. A., McGregor, I. D., & Hewitt, P. L. (2010). Dimensions of perfectionism, behavioral inhibition, and rumination. *Personality and Individual Differences*, *49*, 83–87.
Roisman, G. I., Masten, A. S., Coatsworth, J. D., & Tellegen, A. (2004). Salient and emerging developmental tasks in the transition to adulthood. *Child Development*, *75*, 123–133.
Shapiro, S. L., Carlson, L. E., Astin, J. A., & Freedman, B. (2006). Mechanisms of mindfulness. *Journal of Clinical Psycholology*, *62*, 373–386.
Sportel, B., Nauta, M., Hullu, E., Jong, P., & Hartman, C. (2011). Behavioral inhibition and attentional control in adolescents: Robust relationships with anxiety and depression. *Journal of Child and Family Studies*, *20*, 149–156.
Suveg, C., Morelen, D., Brewer, G. A., & Thomassin, K. (2010). The emotion dysregulation model of anxiety: A preliminary path analytic examination. *Journal of Anxiety Disorders*, *24*, 924–930.
Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Boston: Pearson/Allyn and Bacon.
Tortella-Feliu, M., Balle, M., & Sesé, A. (2010). Relationships between negative affectivity, emotion regulation, anxiety, and depressive symptoms in adolescents as examined through structural equation modeling. *Journal of Anxiety Disorders*, *24*, 686–693.
Tull, M. T., Gratz, K. L., Latzman, R. D., Kimbrel, N. A., & Lejuez, C. W. (2010). Reinforcement sensitivity theory and emotion regulation difficulties: A multimodal investigation. *Personality and Individual Differences*, *49*, 989–994.
Viana, A. G., & Gratz, K. L. (2012). The role of anxiety sensitivity, behavioral inhibition, and cognitive biases in anxiety symptoms: Structural equation modeling of direct and indirect pathways. *Journal of Clinical Psychology*, *68*, 1122–1141.

Appendix 2

Izadpanah, S., Schumacher, M., & Barnow, S. (2017). Anger rumination mediates the relationship between reinforcement sensitivity and psychopathology: Results of a 5-year longitudinal study. *Personality and Individual Differences, 110*, 49-54. doi: 10.1016/j.paid.2017.01.023

Personality and Individual Differences 110 (2017) 49–54

Contents lists available at ScienceDirect

Personality and Individual Differences

journal homepage: www.elsevier.com/locate/paid

Anger rumination mediates the relationship between reinforcement sensitivity and psychopathology: Results of a 5-year longitudinal study

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ARTICLE INFO

Available online 7 February 2017

1. Introduction

Reinforcement sensitivity theory (RST; Gray, 1982), is a biologically-based theory of personality that suggests the "Behavioral Inhibition System" (BIS), together with the "Fight-Flight-Freeze System" (FFFS), and the "Behavioral Approach System" (BAS), control individuals' sensitivity towards signals of punishment and gratification. Although new reconceptualization of RST proposes that FFFS alone mediates the reactions to aversive stimuli and BIS resolves approach-avoidance conflicts (for a review and developments see Corr, 2008), many existing scales still do not capture this change. Hence, here we refer to the original concept of BIS/BAS. Gray's (1994) proposal that anxiety and depression are the result of high BIS is supported by previous findings, while findings on BAS are less consistent (see Bijttebier, Beck, Claes, & Vandereycken, 2009). Some studies support the link between low BAS and depression (e.g., Kimbrel, Nelson-Gray, & Mitchell, 2007), while others do not (Johnson, Turner, & Iwata, 2003). It seems that BAS is a stronger predictor of externalizing rather than internalizing symptoms (Hundt, Kimbrel, Mitchell, & Nelson-Gray, 2008). BIS, but not BAS, predicts anxiety and depression diagnosis (Johnson et al., 2003), while aggression is related to a dominance of BAS over BIS (Quay, 1993). Based on the joint subsystems hypothesis (Corr, 2002), BIS/BAS effects are not independent. However, it is unclear to what extent they exert facilitatory or antagonistic interactive effect for predicting various symptoms. For example, low BAS × high BIS predicts anhedonic depression, while high BIS × high BAS predicts mixed anxiety–depression (Hundt, Nelson-Gray, Kimbrel, Mitchell, & Kwapil, 2007).

Although RST has gained empirical support in predicting psychopathology, the underlying mechanism of this effect is unclear (Bijttebier et al., 2009). Emotion regulation has been suggested as one explanatory construct (e.g., Hannan & Orcutt, 2013), but there is a lack of research on the effect of regulating specific emotions. The emotion of anger offers significant relevance, given the association between BIS/BAS and elevated anger (Harmon-Jones, 2003; Smits & Kuppens, 2005), and their effect on individuals' response towards anger situations (Cooper, Gomez, & Buck, 2008; Smits & Kuppens, 2005). BAS is correlated with left frontal cortical activity, which is associated with anger and aggression (Harmon-Jones & Sigelman, 2001). Furthermore, termination of reward or approach obstruction should cause higher levels of frustration/anger among high BAS individuals (see Carver, 2004). Although both BIS/BAS predict greater anger arousal, BIS leads to an inward anger response such as self-aggression, while BAS predicts an outward anger response and less anger control (Cooper et al., 2008). Rumination is an inward anger response that is defined as repetitively and passively focusing on ones' symptoms of distress and its surrounding circumstances (Nolen-Hoeksema, McBride, & Larson, 1997). Despite previous support for the association between BIS and increased rumination (e.g., Leen-Feldner, Zvolensky, Feldner, & Lejuez, 2004), only one study, to our knowledge, provided cross-sectional evidence for a positive BIS–anger rumination association and an insignificant BAS–anger rumination link (Denson, Pedersen, & Miller, 2006). BIS predicts avoidant (rather than active and problem-focused) coping strategies (Litman, 2006) and ruminative response is also considered as an avoidant strategy towards negative emotions (Stroebe et al., 2007) that results in vulnerability to psychopathology symptoms (Aldao & Nolen-Hoeksema, 2010; Barnow, Aldinger, Ulrich, & Stopsack, 2013).

The role of rumination in psychopathologies such as depression and anxiety is supported by past research (Aldao & Nolen-Hoeksema, 2010; Barnow et al., 2013), while studies on anger rumination are limited so far. Anger rumination increases the intensity of anger experience (Rusting & Nolen-Hoeksema, 1998), and is associated with increased depression (Abdolmanafi, Besharat, Farahani, & Khodaii, 2011; Gilbert, Cheung, Irons, & McEwan, 2005), hostility, and physical/verbal aggression (Anestis, Anestis, Selby, & Joiner, 2009). A ruminative response to negative mood magnifies the effect of the negative mood on thought leading to more severe depression (Nolen-Hoeksema, 1991). While there is no research on anger rumination and anxiety, one study suggests that an inward-directed strategy towards anger contributes to increased anxiety and depression (Koh, Kim, Kim, Park, & Han, 2008). Therefore, systematic studies on the link between anger rumination and various psychopathology symptoms are still missing and these

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http://dx.doi.org/10.1016/j.paid.2017.01.023

studies should consider the multidimensionality of anger rumination. Sukhodolsky, Golub, and Cromwell (2001) suggested that ruminative tendencies towards angry moods and experiences fall under four subscales of "angry afterthoughts" (rethinking about a recent episode of anger), "angry memories" (recalling and getting angry about a distant episode of anger) "thoughts of revenge" (fantasies of taking revenge), and "understanding of causes" (trying to achieve a meaningful understanding of an anger episode). It remains unclear which dimension is more important in relation to BIS and the aforementioned psychopathologies.

In sum, past studies have shown that BIS is associated with an inward anger response, anger rumination (e.g., Denson et al., 2006), and an increased risk for depression and anxiety (Johnson et al., 2003), while BAS is related to an outward anger response and externalizing behaviors such as aggression (Quay, 1993). These findings, coupled with the evidence for anger rumination as a risk factor for depression and anxiety (e.g., Abdolmanafi et al., 2011), suggests anger rumination as a possible underlying mechanism to explain BIS–psychopathology link. Based on the above mentioned findings, this study aimed at testing following hypotheses: (1) BIS (but not BAS) predicts anger rumination, T_2-depression, and T_2-anxiety after a 5-year interval, while BAS predicts increased T_2-aggression and shows only weak or insignificant reverse relationship to T_2-depression; (2) anger rumination predicts T_2-psychopathology; (3) anger rumination mediates the BIS–psychopathology but not BAS–psychopathology link. We also tested the joint subsystem hypothesis to investigate how BIS × BAS interaction predicts each psychopathology symptoms.

2. Materials and methods

2.1. Participants and procedure

The sample was drawn from the population-based Greifswald family study (Aldinger et al., 2014; Barnow et al., 2007; Barnow, Spitzer, Grabe, Kessler, & Freyberger, 2006), a subpopulation from the Study of Health in Pomerania, Germany (SHIP; John et al., 2001). Longitudinal data were collected three times, at 5-year intervals, the second and the third of which (T_1 and T_2) were used in this study.

Between 1997 and 2000 (T_0), 381 offspring from 315 families participated in the study. Between 2005 and 2008, the first follow-up (T_1) was conducted with 334 participants (mean age = 19.56). From 2011 to 2013 (T_2), the participants were investigated again. Data for 85% of T_1 participants were available from this assessment ($N = 284$). Individuals who participated in T_2 did not differ in age from those who dropped out after T_1 ($F = 0.07$, $p = 0.79$). There was an insignificant tendency to more dropouts among men ($\chi = 3.50$, $p = 0.061$) and individuals who did not follow the T_2 assessment had significantly lower BIS ($F = 4.79$, $p = 0.03$) and depression ($F = 9.39$, $p < 0.01$), and higher BAS ($F = 4.40$, $p = 0.04$) at T_1. Furthermore, 11 individuals with missing values for at least one relevant variable were excluded from the analysis, resulting in a final sample of 273 participants (154 women) with a mean age of 19.51 years (14–27) at T_1 and 24.99 years (19–34) at T_2. In this sample, 50 families had participated with two siblings (36.6% of sample) and 2 families participated with three siblings (2.2%).

2.2. Measures

2.2.1. BIS/BAS

At T_1, Action Regulating Emotion Systems (ARES; Hartig & Moosbrugger, 2003), a German alternative to the Behavioral Inhibition/Activation System scales (Carver & White, 1994), was administered. ARES includes 10 items for each BIS (e.g., If I do something wrong, I immediately fear the consequences) and BAS subscale (e.g., Even small incentives can motivate me strongly), ranging from 1 (*strong disagreement*) to 4 (*strong agreement*). ARES shows excellent psychometric properties and a factorial structure consistent with Gray's original BIS and BAS model ($\alpha = 0.89$ and $\alpha = 0.80$, respectively; Hartig & Moosbrugger, 2003). The scale correlated strongly with Eysenck's PEN system, sensation seeking, Big Five, PANAS, and impulsivity scales (Hartig, 2003).

2.2.2. Symptom Checklist-Revised (SCL-90-R) and Brief Symptom Inventory (BSI)

T_1-psychopathology and T_2-psychopathology were measured with the German version of the SCL-90-R (Franke, 1995) and its short form, the BSI (Franke, 2000), respectively. Both versions are comparable and measure psychopathology with nine scales assessing symptoms over the last seven days (Franke, 1995, 2000). Items are rated on a five-point Likert scale, ranging from *not at all* (0) to *extremely* (4). The depression, anxiety, and aggression subscales were used in this study. These scales have adequate internal consistency in SCL-90-R and BSI ($0.77 \le \alpha \le 0.87$ and $0.54 \le \alpha \le 0.82$, respectively), and show good one-week stability ($0.78 \le r \le 0.92$ and $0.88 \le r \le 0.92$, respectively) (Franke, 1995, 2000). To facilitate comparability of T_1-psychopathology and T_2-psychopathology, we extracted BSI items from SCL-90-R and summed them to produce the T_1-anxiety score.

2.2.3. The Anger Rumination Scale (ARS)

(ARS; Sukhodolsky et al., 2001) was applied at T_2 and comprises 19 items rated with a four-point Likert scale ranging from 1 (*almost never*) to 4 (*almost always*). The items measure four factors of "angry afterthoughts" with 6 items (e.g., I re-enact the anger episode in my mind after it has happened), "angry memories" with 5 items (e.g., I keep thinking about events that angered me for a long time), "thoughts of revenge" with 4 items (e.g., I have long living fantasies of revenge after the conflict is over), and "understanding causes" with 4 items (e.g., I think about the reasons people treat me badly). All subscales show adequate validity and reliability (Sukhodolsky et al., 2001).

2.3. Statistical analysis

We analyzed data using IBM SPSS version 20 and analysis of movement structure (AMOS) version 22. We calculated descriptive statistics for each variable and Pearson correlation coefficients between the variables. SEM was applied to design and test the hypothesized model in AMOS with a 95% confidence interval and using the following fit indices: chi-square/*df* ratio < 2.0, Comparative Fit Index (CFI) > 0.90, Goodness of Fit Index (GFI) > 0.90, and Root Mean Square Error of Approximation (RMSEA) < 0.08 (Tabachnick & Fidell, 2007). Using this method allows testing multiple mediators and dependent variables simultaneously (Dattalo, 2013). A SPSS macro called PROCESS was used to determine the significance of individual indirect effects through bootstrapping with 5000 resamples (Preacher & Hayes, 2008). A significant indirect effect is implied if the confidence interval does not include zero.

3. Results

Table 1 illustrates Cronbach's Alpha reliability coefficients, together with means and standard deviations for study variables, and the correlation coefficients. The T_1-anxiety, T_1-depression, and T_1-aggression scores of 35, 28, and 24 participants, as well as T_2-anxiety, T_2-depression, and T_2-aggression of 18, 28, and 22 participants respectively, were within the clinical range ($t \ge 63$, see Franke, 2000). BIS was associated with more depression, anxiety, and aggression at both assessment points and with higher scores on all anger rumination scales. BAS was negatively associated with T_1-depression and thoughts of revenge. Furthermore, all anger rumination subscales were positively associated with depression, anxiety, and aggression at both T_1 and T_2.

Table 1
Correlation coefficients between study variables.

Variables	1	2	3	4	5	6	7	8	9	10	11	12	13
First assessment													
1 ARES-BIS		−0.23***	0.43***	0.31***	0.27***	0.33***	0.33**	0.25***	0.37***	0.19**	0.31***	0.29***	0.36***
2 ARES-BAS			−0.20***	−0.03	−0.13	−0.07	0.01	0.05	−0.09	−0.15*	−0.08	−0.04	−0.10
3 SCL-90-Depression[a]				0.55***	0.66***	0.33***	0.27***	0.39***	0.36***	0.26***	0.32***	0.35***	0.38***
4 SCL-90-Anxiety[a]					0.57***	0.31***	0.42***	0.35***	0.26***	0.21***	0.24***	0.21***	0.27***
5 SCL-90-Aggression[a]						0.30***	0.37***	0.44***	0.26***	0.27***	0.27***	0.26***	0.30***
Second assessment													
6 BSI-Depression							0.70***	0.58***	0.40***	0.27***	0.40***	0.36***	0.43***
7 BSI-Anxiety								0.63***	0.38***	0.32***	0.40***	0.36***	0.43***
8 BSI-Aggression									0.42***	0.37***	0.45***	0.38***	0.48***
9 ARS-Afterthoughts										0.51***	0.75***	0.76***	0.93***
10 ARS-Revenge											0.51***	0.47***	0.66***
11 ARS-Memories												0.69***	0.89***
12 ARS-Causes													0.87***
13 ARS-Total													
Mean	1.35	2.25	2.62	2.30	2.05	0.35	0.33	0.28	1.98	1.42	2.09	2.20	1.94
(SD)	(0.53)	(0.38)	(3.23)	(2.56)	(2.27)	(0.60)	(0.45)	(0.42)	(0.72)	(0.45)	(0.71)	(0.68)	(0.56)
Cronbach alpha	0.87	0.65	0.81	0.69	0.66	0.86	0.77	0.67	0.86	0.59	0.86	0.72	0.93

Note. Action Regulating Emotion Systems (ARES); Behavioral Inhibition System (BIS); Behavioral Activation System (BAS); Symptom Checklist-90 (SCL-90); Brief Symptom Inventory (BSI); Anger Rumination Scale (ARS).
[a] BSI items were extracted and summed.
* $p < 0.05$.
** $p < 0.01$.
*** $p < 0.001$.

3.1. Testing the model

We tested an initial model with paths from BIS/BAS to both anger rumination and T_2-psychopathology, and paths from anger rumination to T_2-psychopathology. We controlled for T_1-psychopathology by drawing a path from each symptom to the same one at T_2. This Model showed an excellent fit: $\chi^2/df = 1.51$, RMSE = 0.04, CFI = 0.99, GFI = 0.97). Testing a more parsimonious model including significant paths of the initial model resulted in an excellent fit as well: $\chi^2/df = 1.38$, RMSE = 0.04, CFI = 0.99, GFI = 0.96. Given that Chi square difference for two models was not significant ($\chi^2 = 7.27$, $df = 9$, p = NS), the more parsimonious model was accepted and reported further in detail (Fig. 1).

3.2. BIS/BAS as predictor

BIS predicted higher anger rumination (B = 0.27, *SE* = 0.07, $p < 0.001$), depression (B = 0.12, *SE* = 0.06, $p = 0.04$), and anxiety (B = 0.10, *SE* = 0.04, $p = 0.02$) after five years, while BAS predicted higher scores on aggression (B = 0.11, *SE* = 0.05, $p = 0.02$) but not depression, anxiety, and anger rumination ($p > 0.10$). BIS × BAS interaction predicted higher scores on anxiety (B = 0.04, *SE* = 0.01, $p < 0.001$) but it did not predict anger rumination, depression, and aggression ($p > 0.10$). Further analysis using PROCESS macro showed that BAS predicted anxiety only at high levels of BIS (B = 0.16, bootstrap *SE* = 0.07, bootstrap CI = 0.02–0.30, $p = 0.02$), but not at low and average BIS ($p > 0.10$). BIS predicted anxiety at low (B = 0.13, bootstrap *SE* = 0.06, bootstrap CI = 0.01–0.24, $p = 0.04$), average (B = 0.20, bootstrap *SE* = 0.05, bootstrap CI = 0.10–0.30, $p < 0.001$), and most strongly at high levels of BAS (B = 0.28, bootstrap *SE* = 0.06, bootstrap CI = 0.16–0.39, $p < 0.001$).

3.3. T_1-psychopathology as predictor

Only T_1–depression predicted more anger rumination (B = 0.05, *SE* = 0.01, $p < 0.001$). T_1-aggression predicted T_2-aggression (B = 0.03, *SE* = 0.01, $p < 0.001$), T_1-anxiety predicted T_2-anxiety (B = 0.03, *SE* = 0.01, $p < 0.001$), and T_1-depression predicted T_2-depression scores (B = −0.03, *SE* = 0.01, $p < 0.02$).

3.4. Anger rumination as predictor

Anger rumination was associated with higher scores on T_2–depression (B = 0.78, *SE* = 0.16, $p < 0.001$) T_2-aggression (B = 0.60, *SE* = 0.11, $p < 0.001$), and T_2-anxiety (B = 0.06, *SE* = 0.11, $p < 0.01$).

3.5. Indirect effects

Bootstrapping results showed that anger rumination mediated the link between BIS and T_2-anxiety (B = 0.07, bootstrap *SE* = 0.01, bootstrap CI = 0.03–0.15, $p < 0.001$), T_2-depression (B = 0.10, bootstrap *SE* = 0.02, bootstrap CI = 0.04–0.22, $p < 0.001$), and T_2-aggression (B = 0.09, bootstrap *SE* = 0.02, bootstrap CI = 0.04–0.16, $p < 0.001$). There was no significant indirect effect of anger rumination on BAS–psychopathology link ($p > 0.10$).

Given that AMOS only provides the sum of all indirect effects, we applied the SPSS macro PROCESS (Preacher & Hayes, 2008) to assess the significance of individual indirect effects. We conducted three separate mediation analyses with each psychopathology symptom as dependent variable, BIS as independent variable, and anger rumination subscales as mediators, while controlling for T_1-psychopathology. Results of this analysis revealed that angry memories (B = 0.12, bootstrap *SE* = 0.05, bootstrap CI = 0.03–0.24) and thoughts of revenge (B = 0.06, bootstrap *SE* = 0.03, bootstrap CI = 0.01–0.14) mediated the link between BIS and T_2-aggression. A significant total effect (B = 0.11, bootstrap *SE* = 0.04, bootstrap CI = 0.02–0.20) and an insignificant direct effect (B = 0.03, bootstrap *SE* = 0.04, bootstrap CI = −0.05-0.11) indicated a full mediation.

Furthermore, after controlling for T_1-depression, angry memories mediated the link between BIS and T_2–depression (B = 0.04, bootstrap *SE* = 0.03, bootstrap CI = 0.02–0.15). Significant total (B = 0.26, bootstrap *SE* = 0.07, bootstrap CI = 0.12–0.39) and direct effect of BIS on T_2-depression (B = 0.17, bootstrap *SE* = 0.07, bootstrap CI = 0.03–0.30) indicated a partial mediation. Angry memories also partially mediated the BIS–anxiety link (B = 0.05, bootstrap *SE* = 0.02, bootstrap CI = 0.01–0.10) with a significant total (B = 0.19, bootstrap *SE* = 0.05, bootstrap CI = 0.09–0.28) and direct effect (B = 0.12, bootstrap *SE* = 0.05, bootstrap CI = 0.03–0.22). There was no indirect effect for angry

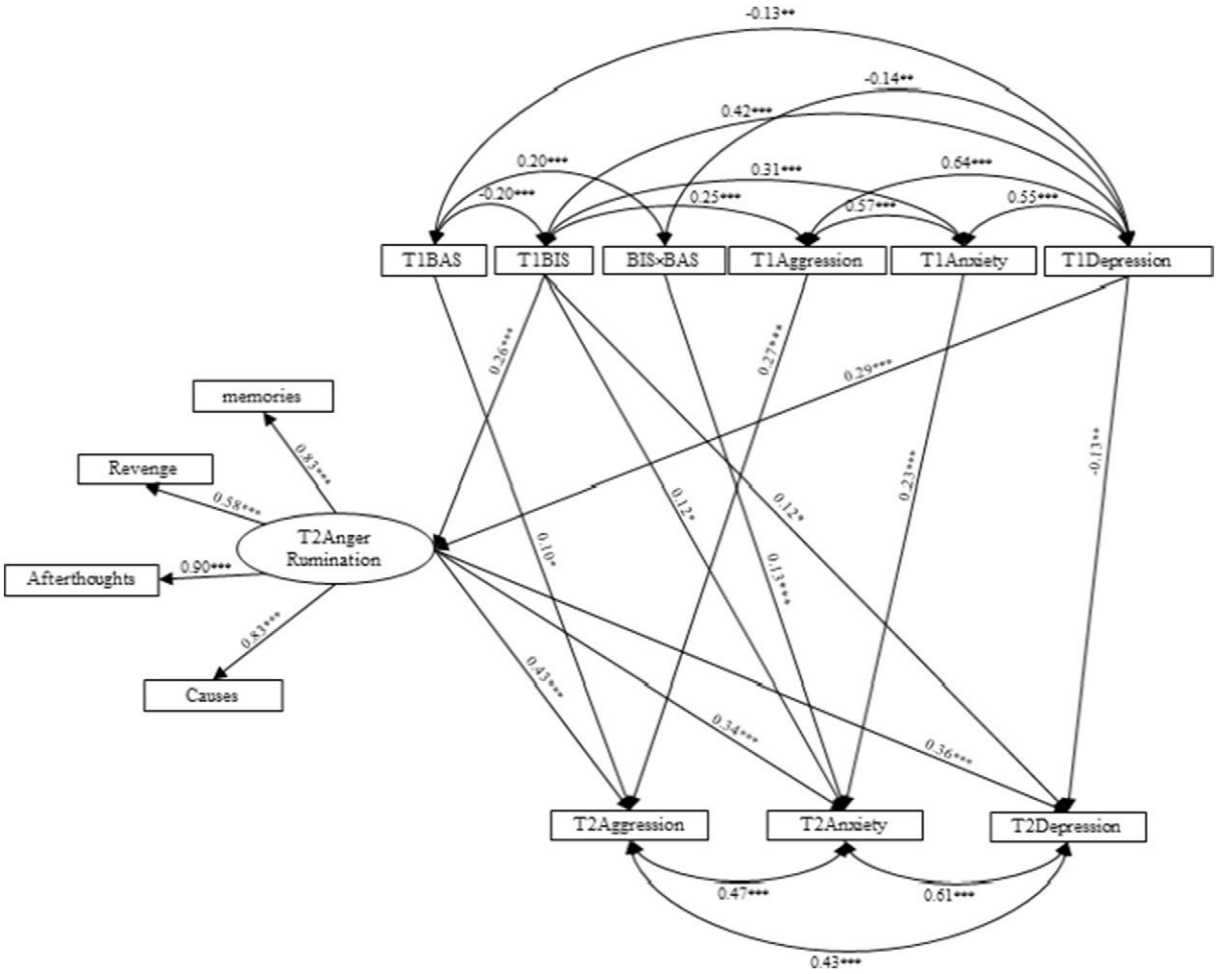

Fig. 1. Standardized regression coefficients for the parsimonious model. *$p < 0.05$. **$p < 0.01$. ***$p < 0.001$.

afterthoughts and understanding of causes on BIS–psychopathology as the confidence intervals for their indirect effects included zero.

4. Discussion

This study provided evidence for the longitudinal effect of BIS on depression and anxiety, and the effect of BAS on aggression, after controlling for baseline psychopathology. Further, anger rumination mediated the link between BIS and depression, anxiety, and aggression.

Results showed that, after controlling for T_1-psychopathology, BIS predicted higher scores on T_2-depression and T_2-anxiety, but not T_2-aggression, while BAS only predicted higher scores on T_2-aggression. These findings strengthen previous evidence that supports BIS as a risk factor for depression and anxiety (e.g., Schofield, Coles, & Gibb, 2009), and relates adults' externalizing symptoms to high BAS but not low BIS (Hundt et al., 2008). It seems that BAS is more directly associated with aggression compared to the BIS–aggression link, which seems to be mediated through other constructs such as general negative affect (Harmon-Jones, 2003). Past literature on BAS–depression link have found weak or insignificant association (see Bijttebier et al., 2009). In this study, although there was a negative correlation at T_1, BAS did not longitudinally predict depression. This brings up the question if cross sectional BAS–depression associations might be explained by shared characteristics of depression and BAS or if this link might be indirect and mediated through other constructs such as positive experience and expectancies (see Beevers & Meyer, 2002). Further, BAS might be a stronger predictor of anhedonic depression (Bijttebier et al., 2009).

Furthermore, BIS × BAS interaction predicted anxiety but not aggression and depression. Similarly, Hundt et al. (2007), found that BIS effect on mixed depression/anxiety symptoms was strongest at high BAS, and the same effect for BAS was only significant at high BIS. BAS sensitivity seems to be important for the development of anxiety only when combined with high BIS. Daily life situations entail a mixture of appetitive and aversive stimuli that based on joint subsystems hypothesis, result in an interactive effect of BIS/BAS. Based on this hypothesis, with weak aversive stimuli, BAS impairs BIS-mediated behavior. However, we found a facilitating effect of BIS on BAS–anxiety link. This finding provides evidence for the BIS/BAS joint effect. However, this interaction effect on other psychopathology symptoms such as depression might be conditioned on environmental factors such as low life stress (Hundt et al., 2007). As Hundt et al. (2007) argued, individuals with both high BIS and BAS might experience elevated distress as a result of experiencing more frequent approach-avoidance conflicts. When both aversion and approach are at high levels, the antagonistic effect of one system on another might be impaired, resulting in a prolonged conflict state and distress.

Finally, our study was the first to investigate the indirect effect of anger rumination on the link between BIS/BAS and psychopathology. We found an indirect effect of angry memories and thoughts of revenge on BIS–aggression link. Similar to our findings, BIS/BAS have shown positive relationship with increased vengeance or revenge seeking. (Johnson, Kim, Giovannelli, & Cagle, 2010). Furthermore, anger rumination has been associated with higher hostility and physical/verbal aggression (Anestis et al., 2009). Our results were complementary to this previous finding by reporting results on anger rumination subscales, and showing that anger rumination is also related to depression and

anxiety. Angry memories mediated the link between BIS and depression and anxiety, controlling for T_1-psychopathology. While angry memories are important aspects of forgiving (Barber, Maltby, & Macaskill, 2005), individuals with high BIS show lower self and situational forgiveness (Johnson et al., 2010), which in turn can elevate anger rumination and increase their vulnerability to psychopathology symptoms (Barber et al., 2005). Given that BIS, based on the original conceptualization, is characterized by sensitivity to punishment and situations with no reward (Gray, 1994), individuals with higher punishment sensitivity might not react actively to anger situations due to fear of eliminating a positive state (e.g., positive attention) or receiving negative consequences such as counter anger. However, considering the inadequacy of this approach in eliminating anger or resolving the situation, the emotion will continue to be processed, thus facilitating anger rumination. In general, although both BIS/BAS have been related to elevated anger (Smits & Kuppens, 2005), those with BIS sensitivity seem to apply a more avoidant coping and engage less often in active and problem-focused coping (Hundt, Williams, Mendelson, & Nelson-Gray, 2013). This avoidant approach towards anger eliciting situations, which might be a result of fear of causing further anxiety-provoking stimuli, can facilitate applying more passive alternatives to deal with anger such as rumination. As we expected, anger rumination did not mediate BAS–psychopathology link. This supports the differential functioning of BIS and BAS systems (Gray, 1994).

This study had several limitations. First, self-report measures are sensitive to social desirability biases. Second, we only used the "ARES" to measure BIS/BAS, while different measures of BIS/BAS do not seem to measure the exact same construct (Krupić, Corr, Ručević, Križanić, & Gračanin, 2016). Appling multiple questionnaires based on the revised RST (Corr, 2016) facilitates investigating convergent validity of the scales, together with distinct effect of FFFS and its interaction with BIS/BAS. Corr and Cooper (2016) developed a questionnaire based on a more comprehensive model of RST that integrates the most recent RST reconceptualization. Third, given that we did not measure anger rumination at T_1, we could not make longitudinal conclusions about BIS–anger rumination link. Fourth, while we focused on anger rumination, it might be fruitful to investigate how BIS/BAS are related to other types of anger regulation. Finally, future research should test the indirect effect of anger rumination on BIS–depression link, controlling for depressive rumination.

Despite these limitations, our findings contribute to the literature on BIS–psychopathology link by providing evidence for anger rumination as one underlying mechanism. One implication would be including psycho-educational material about anger rumination in depression, anxiety, and aggression treatment, or considering anger rumination a preventive target among individuals with high BIS. It has been shown that active coping is the best strategy for controlling anger (Maxwell & Siu, 2008). Since our findings revealed the relevance of angry memories and thoughts of revenge for psychopathology, cultivating forgiveness (e.g., through meditation) might be of special psychotherapeutic benefit (Menahem & Love, 2013).

Acknowledgments

The Greifswald family study was supported by the German Research Foundation (Deutsche Forschungsgemeinschaft, BA-2172/2-1, BA2172.8-1.GR1912.7-1).

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.paid.2017.01.023.

References

Abdolmanafi, A., Besharat, M. A., Farahani, H., & Khodaii, M. R. (2011). The moderating role of locus of control on the relationship between anger rumination and depression in patients with major depression disorder. *Procedia - Social and Behavioral Sciences*, *30*, 302–306.

Aldao, A., & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: A transdiagnostic examination. *Behaviour Research and Therapy*, *48*, 974–983.

Aldinger, M., Stopsack, M., Ulrich, I., Reinelt, E., Lang, S., & Barnow, S. (2014). Neuroticism developmental courses–implications for depression, anxiety and everyday emotional experience: A prospective study from adolescence to young adulthood. *BMC Psychiatry*, *14*, 210.

Anestis, M. D., Anestis, J. C., Selby, E. A., & Joiner, T. E. (2009). Anger rumination across forms of aggression. *Personality and Individual Differences*, *46*, 192–196.

Barber, L., Maltby, J., & Macaskill, A. (2005). Angry memories and thoughts of revenge: The relationship between forgiveness and anger rumination. *Personality and Individual Differences*, *39*, 253–262.

Barnow, S., Spitzer, C., Grabe, H. J., Kessler, C., & Freyberger, H. J. (2006). Individual characteristics, familial experience, and psychopathology in children of mothers with borderline personality disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*, 965–972.

Barnow, S., Herpertz, S. C., Spitzer, C., Stopsack, M., Preuss, U. W., Grabe, H. J., ... Akiskal, H. S. (2007). Temperament and character in patients with borderline personality disorder taking gender and comorbidity into account. *Psychopathology*, *40*, 369–378.

Barnow, S., Aldinger, M., Ulrich, I., & Stopsack, M. (2013). Emotion regulation in depression: An overview of results using various methods [Emotionsregulation bei Depression: Ein multimethodaler Überblick]. *Psychologische Rundschau*, *64*, 235–243.

Beevers, C. G., & Meyer, B. (2002). Lack of positive experiences and positive expectancies mediate the relationship between BAS responsiveness and depression. *Cognition & Emotion*, *16*, 549–564.

Bijttebier, P., Beck, I., Claes, L., & Vandereycken, W. (2009). Gray's reinforcement sensitivity theory as a framework for research on personality–psychopathology associations. *Clinical Psychology Review*, *29*, 421–430.

Carver, C. S. (2004). Negative affects deriving from the behavioral approach system. *Emotion*, *4*, 3–22.

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology*, *67*, 319–333.

Cooper, A., Gomez, R., & Buck, E. (2008). The relationships between the BIS and BAS, anger and responses to anger. *Personality and Individual Differences*, *44*, 403–413.

Corr, P. J. (2002). J. A. Gray's reinforcement sensitivity theory: Tests of the joint subsystems hypothesis of anxiety and impulsivity. *Personality and Individual Differences*, *33*, 511–532.

Corr, P. J. (2008). *The reinforcement sensitivity theory of personality* (1. publ. ed.). Cambridge [u.a.]: Cambridge Univ. Press, XVIII (537 S).

Corr, P. J. (2016). Reinforcement sensitivity theory of personality questionnaires: Structural survey with recommendations. *Personality and Individual Differences*, *89*, 60–64.

Corr, P. J., & Cooper, A. J. (2016). The reinforcement sensitivity theory of personality questionnaire (RST-PQ): Development and validation. *Psychological Assessment*, *28*, 1427–1440.

Dattalo, P. (2013). *Analysis of multiple dependent variables.* Oxford University Press.

Denson, T. F., Pedersen, W. C., & Miller, N. (2006). The displaced aggression questionnaire. *Journal of Personality and Social Psychology*, *90*, 1032–1051.

Franke, G. H. (1995). *SCL-90-R: The symptom-checklist from Derogatis: German version.* Göttingen: Beltz Test.

Franke, G. H. (2000). *BSI: Brief symptom inventory from Derogatis (short form of SCL-90-R); German version; manual: Beltz test.*

Gilbert, P., Cheung, M., Irons, C., & McEwan, K. (2005). An exploration into depression-focused and anger-focused rumination in relation to depression in a student population. *Behavioural and Cognitive Psychotherapy*, *33*, 273–283.

Gray, J. A. (1982). *The neuropsychology of anxiety: An enquiry into the functions of the septo-hippocampal system.* New York, NY: Oxford University Press.

Gray, J. A. (1994). Framework for a taxonomy of psychiatric disorder. In S. H. M. Van Goozen, N. E. Van de Poll, & J. A. Sergeant (Eds.), *Emotions: Essays on emotion theory* (pp. 29–59). Hillsdale, NJ: Erlbaum.

Hannan, S. M., & Orcutt, H. K. (2013). Emotion dysregulation as a partial mediator between reinforcement sensitivity and posttraumatic stress symptoms. *Personality and Individual Differences*, *55*, 574–578.

Harmon-Jones, E. (2003). Anger and the behavioral approach system. *Personality and Individual Differences*, *35*, 995–1005.

Harmon-Jones, E., & Sigelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology*, *80*, 797–803.

Hartig, J. (2003). *Sensitivity to reward and punishment as the basis of fundamental personality dimensions: A contribution to the study of Grays amplifier sensitivity theory.* Frankfurt: Goethe-University.

Hartig, J., & Moosbrugger, H. (2003). The ARES-Scales for measurment of BIS and BAS sensitivity. *Zeitschrift für Differentielle und Diagnostische Psychologie*, *24*, 293–310.

Hundt, N. E., Nelson-Gray, R. O., Kimbrel, N. A., Mitchell, J. T., & Kwapil, T. R. (2007). The interaction of reinforcement sensitivity and life events in the prediction of anhedonic depression and mixed anxiety-depression symptoms. *Personality and Individual Differences*, *43*, 1001–1012.

Hundt, N. E., Kimbrel, N. A., Mitchell, J. T., & Nelson-Gray, R. O. (2008). High BAS, but not low BIS, predicts externalizing symptoms in adults. *Personality and Individual Differences*, *44*, 565–575.

Hundt, N. E., Williams, A. M., Mendelson, J., & Nelson-Gray, R. (2013). Coping mediates relationships between reinforcement sensitivity and symptoms of psychopathology. *Personality and Individual Differences*, *54*, 726–731.

John, U., Greiner, B., Hensel, E., Ludemann, J., Piek, M., Sauer, S., ... Kessler, C. (2001). Study of health in Pomerania (SHIP): A health examination survey in an east German region: Objectives and design. *Sozial- und Präventivmedizin*, *46*, 186–194.

Johnson, S. L., Turner, R. J., & Iwata, N. (2003). BIS/BAS levels and psychiatric disorder: An epidemiological study. *Journal of Psychopathology and Behavioral Assessment*, *25*, 25–36.

Johnson, J. L., Kim, L. M., Giovannelli, T. S., & Cagle, T. (2010). Reinforcement sensitivity theory, vengeance, and forgiveness. *Personality and Individual Differences*, *48*, 612–616.

Kimbrel, N. A., Nelson-Gray, R. O., & Mitchell, J. T. (2007). Reinforcement sensitivity and maternal style as predictors of psychopathology. *Personality and Individual Differences*, *42*, 1139–1149.

Koh, K. B., Kim, D. K., Kim, S. Y., Park, J. K., & Han, M. (2008). The relation between anger management style, mood and somatic symptoms in anxiety disorders and somatoform disorders. *Psychiatry Research*, *160*, 372–379.

Krupić, D., Corr, P. J., Ručević, S., Križanić, V., & Gračanin, A. (2016). Five reinforcement sensitivity theory (RST) of personality questionnaires: Comparison, validity and generalization. *Personality and Individual Differences*, *97*, 19–24.

Leen-Feldner, E. W., Zvolensky, M. J., Feldner, M. T., & Lejuez, C. W. (2004). Behavioral inhibition: Relation to negative emotion regulation and reactivity. *Personality and Individual Differences*, *36*, 1235–1247.

Litman, J. A. (2006). The COPE inventory: Dimensionality and relationships with approach- and avoidance-motives and positive and negative traits. *Personality and Individual Differences*, *41*, 273–284.

Maxwell, J. P., & Siu, O. L. (2008). The Chinese coping strategies scale: Relationships with aggression, anger, and rumination in a diverse sample of Hong Kong Chinese adults. *Personality and Individual Differences*, *44*, 1049–1059.

Menahem, S., & Love, M. (2013). Forgiveness in psychotherapy: The key to healing. *Journal of Clinical Psychology*, *69*, 829–835.

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, *100*, 569–582.

Nolen-Hoeksema, S., McBride, A., & Larson, J. (1997). Rumination and psychological distress among bereaved partners. *Journal of Personality and Social Psychology*, *72*, 855–862.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*, *40*, 879–891.

Quay, H. C. (1993). The psychobiology of undersocialized aggressive conduct disorder: A theoretical perspective. *Development and Psychopathology*, *5*, 165–180.

Rusting, C. L., & Nolen-Hoeksema, S. (1998). Regulating responses to anger: Effects of rumination and distraction on angry mood. *Journal of Personality and Social Psychology*, *74*, 790.

Schofield, C. A., Coles, M. E., & Gibb, B. E. (2009). Retrospective reports of behavioral inhibition and young adults' current symptoms of social anxiety, depression, and anxious arousal. *Journal of Anxiety Disorders*, *23*, 884–890.

Smits, D. J. M., & Kuppens, P. (2005). The relations between anger, coping with anger, and aggression, and the BIS/BAS system. *Personality and Individual Differences*, *39*, 783–793.

Stroebe, M., Boelen, P. A., Hout, M., Stroebe, W., Salemink, E., & Bout, J. (2007). Ruminative coping as avoidance. *European Archives of Psychiatry and Clinical Neuroscience*, *257*, 462–472.

Sukhodolsky, D. G., Golub, A., & Cromwell, E. N. (2001). Development and validation of the anger rumination scale. *Personality and Individual Differences*, *31*, 689–700.

Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics* (5th ed.). Boston: Pearson/Allyn and Bacon.

Appendix 3

Izadpanah, S., Aldinger, M., Arens, E., Stopsack, M., I., U., Hansenne, M., Grabe, H. J., & Barnow, S. (2016). Adolescent harm avoidance as a longitudinal predictor of maladaptive cognitive emotion regulation in adulthood: The mediating role of inhibitory control. *Journal of Adolescence, 52,* 49-59. doi: 10.1016/j.adolescence.2016.07.006

Journal of Adolescence 52 (2016) 49–59

Contents lists available at ScienceDirect

Journal of Adolescence

journal homepage: www.elsevier.com/locate/jado

Adolescent harm avoidance as a longitudinal predictor of maladaptive cognitive emotion regulation in adulthood: The mediating role of inhibitory control

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ARTICLE INFO

Article history:
Available online 3 August 2016

Keywords:
Harm avoidance
Temperament
Cognitive emotion regulation
Inhibitory control
Impulsivity
Gender differences

ABSTRACT

The current study investigates the effect of adolescent harm avoidance (HA) on maladaptive cognitive emotion regulation strategies (mCER) in early adulthood. The mediating role of inhibitory control and the moderating effect of gender on this link were also examined. Longitudinal data from 261 adolescents (147 female) were collected in three phases (T0, T1 and T2) over approximately 10 years. Results revealed that, after controlling for HA in adulthood (T2), female adolescents' HA (T0) significantly predicted mCER strategies after 10 years (T2), whereas male adolescents' HA only predicted catastrophizing. In addition, attentional impulsivity (T1) significantly mediated the relation between HA and mCER, though only among women. There was no significant indirect effect for emotional interference and stop-signal reaction time. Results revealed gender and measure specific associations between HA and inhibitory control and suggest that HA could induce inhibitory deficits leading to mCER.

Cognitive emotion regulation strategies are defined as cognitive strategies for responding to stressful events and handling emotionally arousing information (Garnefski, Kraaij, & Spinhoven, 2001). Maladaptive cognitive emotion regulation strategies (mCER) such as self-blame, rumination, catastrophizing, blaming others and suppression have been shown to have a detrimental impact on mental health (Barnow, Aldinger, Ulrich, & Stopsack, 2013; Izadpanah et al., 2016) and quality of life (Elphinston, Feeney, Noller, Connor, & Fitzgerald, 2013) among early adolescents and young adults. Studying the period between adolescence and adulthood seems to be important for understanding the development of cognitive emotion

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http://dx.doi.org/10.1016/j.adolescence.2016.07.006
0140-1971/

regulation strategies as the application of these strategies increases from adolescence to adulthood and these strategies are improved, modified and mastered within this period (Garnefski & Kraaij, 2006). However, the existing literature on the predictors and underlying mechanism of mCER in adolescence and adulthood is surprisingly scarce (Jose, Wilkins, & Spendelow, 2012; Zetsche & Joormann, 2011). Understanding these mechanisms is of great importance for early treatment of psychopathology among at risk adolescents as it creates implications for designing preventive programs (Aldao & Nolen-Hoeksema, 2010; Barnow, Löw, Dodek, & Stopsack, 2014).

Developmental predictors of mCER

Temperament has been proposed as a blueprint and foundation for emotional development (Southam-Gerow & Kendall, 2002). Previous researchers have studied the temperamental construct of negative affectivity in relation to emotion regulation (Tortella-Feliu, Balle, & Sesé, 2010). Harm avoidance (HA) is one of these constructs and has also been closely related to trait anxiety (e.g., Caci, Robert, & Boyer, 2004). HA has been associated with psychopathologies characterized by emotional dysregulation, such as major depressive disorder (Barnow, Rüge, Spitzer, & Freyberger, 2005; Cloninger, Svrakic, & Przybeck, 2006). High scores in HA reflect traits like fearfulness of uncertainty, shyness with strangers, fatigability, and anticipatory worries (Cloninger, 1994a). In addition, HA has been related to strong reactions and attentional biases towards negative stimuli (Cloninger, 1987). Limited studies have investigated the association between HA and emotion regulation (Manfredi et al., 2011; Schreiber, Grant, & Odlaug, 2012). Schreiber et al. (2012) provided support for the association between HA and difficulties in emotion regulation. In their study, 194 young adults were separated into low, average, and high levels of emotion dysregulation. They found higher levels of HA among those with more emotion regulation difficulties. To our knowledge, the only study testing the relation between HA and mCER, concentrated exclusively on rumination (Manfredi et al., 2011). In their study, Manfredi et al. (2011) found that adults with higher levels of HA scored higher in the mCER strategy "rumination". So far, no study to our knowledge investigates the relationship between HA and other mCER strategies. However, adolescents with higher HA might develop a tendency to use strategies such as rumination, self-blame, and catastrophizing as dysfunctional means of coping with their worries, uncertainty or shyness——all features of trait HA (Carleton, Sharpe, & Asmundson, 2007; Henderson, 2002; Liao & Wei, 2011). Further, given the association between negative affectivity and mCER strategies such as self-blame, blaming others and catastrophizing (Gilbert & Miles, 2000; Gunthert, Cohen, & Armeli, 1999; Martin & Dahlen, 2005), we assumed that HA—as a construct of negative affectivity—might also be associated with higher usage of these strategies. Although the above mentioned cross-sectional findings support a positive link between HA and emotion dysregulation among young adults, no study has longitudinally investigated the influence of adolescent HA on emotion dysregulation in adulthood.

Inhibitory control as an underlying mechanism

Although Manfredi et al. (2011) and Schreiber et al. (2012) provided initial evidence suggesting a relationship between HA and emotion dysregulation, the mechanism behind this link remains unclear. One possible mechanism for this association might rely on the implications of inhibitory control. Inhibitory control refers to the ability to suppress inappropriate responses or attention tendencies in order to act appropriately on the task at hand (Dempster, 1992; Nigg, 2000). Although no study has investigated the link between HA and inhibitory control deficits, past evidence suggests such an association (Hansenne, 1999; Most, Chun, Johnson, & Kiehl, 2006). A vast majority of studies have demonstrated that chronic negative affect contributes to inhibitory control deficits (Zetsche, D'Avanzato, & Joormann, 2012). However, there are no studies on the link between trait negative affect and inhibitory control. It has been suggested that affective states associated with personality traits might be a potential mechanism that leads to inhibitory control deficits (Hahn, Buttaccio, Hahn, & Lee, 2015; Watson & Clark, 1992). Similarly, in line with the state-trait model of anxiety (Eysenck, 1982), HA might predict performance impairment in inhibitory control tasks through determining state anxiety (see also Matthews & Deary, 2000b, pp. 70–90). Accordingly, Matthews, Joyner, Gilliland, Huggins, and Falconer (1999) showed that trait negative affectivity (measured with neuroticism) predicted higher levels of distraction and interfering cognitions during an Emotional Stroop Task, which might interfere with inhibitory control processes. Further, HA is characterized by attentional bias towards negative stimuli, which can slow down the process of naming the color of emotional words and result in lower inhibitory control of the emotional stimuli (Matthews & Deary, 2000a). Accordingly, past findings have shown that individuals with high HA have difficulty inhibiting irrelevant information when searching for targets during an attentional task (Most et al., 2006; Most, Chun, Widders, & Zald, 2005). HA has shown to be associated with an automated pattern of attending to neutral and emotional stimuli (Hansenne et al., 2003; Mardaga & Hansenne, 2009) and a strong attentional bias towards emotionally negative stimuli (Cloninger, 1994b; Zhang et al., 2013), both of which might cause difficulties in suppressing irrelevant information and facilitate the interference of negative emotional information leading to inhibitory control deficits (Matthews & Deary, 2000a; Weierich, Treat, & Hollingworth, 2008). In addition, HA has been associated with higher levels of self-report inhibitory control (Schreiber et al., 2012). Higher HA scores have also been associated with psychopathologies characterized by low inhibitory control (Kusunoki et al., 2000; Lyoo, Lee, Kim, Kong, & Kwon, 2001).

Further, numerous studies support the proposition that inhibition deficits are related to the mCER strategy rumination (Joormann, 2006; Whitmer & Banich, 2007; Zetsche et al., 2012). For instance, Joormann (2006) showed that an inhibition deficit as assessed by negative priming was associated with more rumination. Similarly, deficits in inhibiting neutral

(Whitmer & Banich, 2007) and emotionally negative information (De Lissnyder et al., 2012; Donaldson, Lam, & Mathews, 2007; Joormann & Gotlib, 2010; Zetsche et al., 2012) have been associated with increased rumination. Accordingly, it has been argued that deficits in inhibitory control facilitate mCER by impairing the access to mood-incongruent material (Joormann, 2010). Although inhibitory control has only been investigated in relation to rumination, we assumed that inhibitory control deficits might be related to other mCER such as self-blame, blaming others, and catastrophizing as these strategies have a cognitive nature that consists of recurrent dysfunctional thoughts (Garnefski et al., 2001). Therefore, low inhibitory control might facilitate the increased interference of these thoughts and thereby increase vulnerability to mCER.

All in all, past findings imply a positive link between HA and inhibitory deficits and between HA and mCER (e.g., Manfredi, et al., 2011; Most et al., 2006). These findings, coupled with evidence suggesting that inhibition deficits underlie mCER (e.g., Joormann, 2010), point to the possibility that inhibition deficits might mediate the relation between HA and mCER. This is in accordance with the theoretical background, which proposes that negative affectivity contributes to inhibition deficits, which in turn increases vulnerability to mCER and reduces the chance of applying more functional emotion regulation strategies (Joormann, 2010). It is worth noting that, given the insignificant or low association between various measures of inhibitory control, it seems that different measures of inhibitory control test different underlying components of this construct (Khng & Lee, 2014; Reynolds, Ortengren, Richards, & de Wit, 2006). Considering this point, the current study is the first study that longitudinally investigates the distinctive association between HA, inhibitory control, and mCER, including various measures of inhibitory control and various mCER strategies. This multimethod assessment enables us to investigate how HA is associated with well-known state-dependent experimental tasks of inhibitory control (Emotional Stroop and Stop-Signal Task) (Miyake et al., 2000), as well as with self-reported, and less state-dependent inhibitory control (Barrat impulsiveness scale; Patton, Stanford, & Barratt, 1995).

Gender differences

Studying the development of emotion regulation without taking gender differences into account might be misleading (Cole, 2014). Women report higher levels of HA (Al-Halabí et al., 2011; Cloninger et al., 2006), which might predispose them to more adverse health outcomes (Cloninger, Bayon, & Svrakic, 1998). Further, women engage more strongly in their negative emotions than men do and adopt more internally focused and passive responses to emotions (Tamres, Janicki, & Helgeson, 2002). This tendency might also be related to men's traditional gender roles, which require more active and agentic responses on their part, such as problem-solving or reappraisal with the aim of changing the situation that triggered the emotion (Butler & Nolen-Hoeksema, 1994; Tamres et al., 2002). This internalizing approach, along with higher levels of HA, can lead to higher levels of mCER among women (Nolen-Hoeksema & Aldao, 2011; Zlomke & Hahn, 2010) as well as a higher prevalence of disorders characterized by emotional disturbance compared to men (Tomko, Trull, Wood, & Sher, 2013). According to these findings, recent neuropsychological evidence highlights the importance of considering gender differences when studying HA associations (Li, Qin, Jiang, Zhang, & Yu, 2012). Therefore, we examined the moderating effect of gender on the pathways in our model that included HA.

The current study

Fig. 1 illustrates the aims of the present study. Considering the above mentioned findings, the current study investigates:

- adolescent HA (at T_0) as a predictor of adulthood mCER over a period of 10 years (at T_2);
- adolescent HA as a predictor of various inhibitory control measures assessed after a 5-year interval (at T_1);
- how various inhibitory control measures predict mCER when measured after 5 years (at T_2);
- the role of inhibitory control in the relationship between HA and mCER using a multimethod assessment of inhibitory control, taking the moderating effect of gender into account.

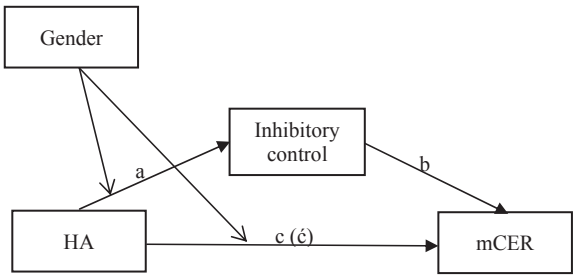

Fig. 1. Hypothesized moderated mediation model representing inhibitory control as mediator and gender as moderator. Harm avoidance (HA); maladaptive cognitive emotion regulation (mCER). Direct effect of HA on inhibitory control (a); direct effect of inhibitory control on mCER (b); total effect of HA on mCER (c); direct effect of HA on mCER, controlling for inhibitory control (ć).

Method

Participants

The sample was drawn from the population-based Greifswald family study (Aldinger et al., 2014; Barnow et al., 2005). Between 1997 and 2000 (T_0), 315 families (with 381 offspring, mean age = 15.1, $SD = 2.3$) participated in the family study. The first follow up (T_1), conducted about five years later between 2005 and 2008 (mean $interval_{T1-T0}$ = 53.18 months, SD_{T1-T0} = 12.97), included 87.7% of offspring ($n = 334$, mean age = 19.6, $SD = 2.4$). From May 2011 to April 2014 they were investigated a third time (mean $interval_{T2-T1}$ = 65.63 months, SD_{T2-T1} = 8.14) and, from this assessment, data for 85% of T_1 offspring participants are available ($n = 284$). Those who participated in all three assessments did not differ from individuals who dropped out after T_0 concerning gender ($\chi^2 = 2.37$, $p = .146$), age ($F = 2.05$, $p = .153$), and HA ($F = .59$, $p = .55$) at T_0. Further, 23 individuals had missing values for at least one of the relevant variables and were excluded from the analysis, which resulted in a final sample of 261 participants (147 women) between 19 and 34 years old (mean age = 24.93, $SD = 2.42$). The mean age of this final sample at T_0 and T_1 was 15.03 (11–21) and 19.43 (14–27) years old, respectively. All participants gave informed consent and the study was approved by the local ethics committee of Ruprecht-Karls-University Heidelberg.

Materials and procedure

Assessment at T_0. Participants between 16 and 21 years old completed the Temperament and Character Inventory (TCI), which is a self-administered true-false questionnaire based on Cloninger's psychobiological model (Cloninger, Svrakic, & Przybeck, 1993). This questionnaire includes 240 items measuring three dimensions of character and four dimensions of temperament. Temperament dimensions consist of HA, novelty seeking, reward dependence, and persistence (Cloninger et al., 1993). The German version of TCI has good psychometric properties with internal consistencies ranging from $\alpha = .66$ for reward dependence to $\alpha = .83$ for novelty seeking (Richter, Eisemann, & Richter, 2000). HA was the variable of interest in the current study and has shown acceptable internal consistency and 54-day stability ($\alpha = .76$, $r = .72$, respectively; Richter et al., 2000). Due to age restrictions of the TCI, participants younger than 15 completed the German version of Junior TCI (JTCI), which is an adapted version of TCI with 108 items (Luby, Svrakic, McCallum, Przybeck, & Cloninger, 1999) (see Table 1). The German version of the JTCI measures the same scales as the adult version, and its HA scale has also shown good internal consistency and two-week stability ($\alpha = .81$, $r = .88$; Schmeck, Goth, Poustka, & Cloninger, 2001). Z score transformation of the HA scales in JTCI and TCI were combined to obtain one value for the different age groups.

Assessment at T_1. In our inhibition control battery, we first utilized the Stop-Signal Task (Logan, 1994). Following the suggestion of Logan, Schachar, and Tannock (1997), the tracking version of this task was applied with a variant stop-signal delay. The task requires individuals to suppress a primary ongoing go response whenever a sudden auditory stimulus is presented. Stop-signal reaction time (SSRT) was calculated as a measure of the time individuals need to inhibit the primary go response by subtracting the mean of delay from the mean reaction time. Longer SSRT indicates poorer inhibitory control.

In addition, the Emotional Stroop was applied as another measure for inhibition control (see Miyake et al., 2000). The task contained 114 (59 emotionally negative and 55 neutral) words that were presented in different colors. Participants named the words' colors after being presented with each word. In order to obtain the mean interference score, we calculated the mean reaction time for each word and the difference of reaction time between emotional and neutral words. A longer reaction time for emotional words, compared to neutral ones, indicates more difficulty in inhibiting the interference of emotional stimuli.

We further used the Barratt Impulsiveness Scale, version 11 (BIS; Patton et al., 1995), which has been regarded in literature as a self-report measure of inhibitory control (Enticott, Ogloff, & Bradshaw, 2006). The BIS has 30 items with a four-point Likert scale (*never*, *occasionally*, *often*, *almost always*) and shows adequate psychometric properties ($.79 \leq \alpha \leq .83$; Patton et al., 1995). The BIS covers three sub-traits of motor, attentional and non-planning impulsiveness.

Assessment at T_2. Participants completed the Cognitive Emotion Regulation Questionnaire (CERQ) (Garnefski et al., 2001). The CERQ consists of 36 items that are rated on a scale from 1 (*sometimes*) to 5 (*always*) and measures nine cognitive strategies of positive reappraisal, acceptance, refocus on planning, positive refocus, putting into perspective, self-blame, rumination, catastrophizing, and blaming others. According to its goals, the present study was interested in the four latter strategies,

Table 1
Constructs and their respective measures that were applied in three measurement points.

Constructs	Measure	Measurement point
Harm avoidance		T_0
age < 16	Junior temperament and character inventory	
age ≥ 16	Temperament and character inventory	
Inhibitory control	Stop signal task	T_1
	Emotional stroop	
	Barrat impulsiveness scale	
Emotion regulation	Cognitive emotion regulation questionnaire	T_2
Harm avoidance	Temperament and character inventory	

Note. The first level of assessment (T_0); the first follow up (T_1); the second follow up (T_2).

which are known to be dysfunctional. The German translation of CERQ showed adequate internal consistency (.73 < α < .60) (Loch, Hiller, & Witthöft, 2011). In addition, at this measurement point, all participants completed the TCI a second time.

Data analysis

All analyses were conducted using IBM statistics 20 and the SPSS macro PROCESS (A. F. Hayes, 2013). First, given the comparability of TCI and JTCI (Luby et al., 1999), the z scores of HA scales for both age groups were produced and combined. Next, we determined the mean and standard deviation for each variable and also performed bivariate correlations to examine the association between variables. Then, in order to analyze the moderated mediation hypothesis, we used SPSS macro PROCESS (A. F. Hayes, 2013) and applied the bootstrapping method introduced by Preacher and Hayes (2008), through which we obtained 5000 resamples of data and estimated indirect effects. Moderated mediation analysis is subsumed under the category of conditional indirect effects (Preacher, Rucker, & Hayes, 2007), for which using the bootstrapping method is strongly recommended (Mackinnon, Lockwood, & Williams, 2004). Bootstrapping method is less vulnerable to Type II error compared to other methods, which results in a higher statistical power (Preacher & Hayes, 2008). Applying PROCESS macro facilitates the estimation of conditional indirect and direct effects and examines the significance of effects at different values of moderator variables. The degree of an indirect effect might vary at different moderator values (A. F. Hayes, 2013). This means that the mediational model might hold for one group but not for another, or that the magnitude of the indirect effect might differ according to the values of the moderator. The presence of a significant effect can be inferred if the confidence interval does not include zero. In the current study, an alpha of .05 was set for statistical significance. Age and financial status were included as covariates in the moderated mediation analysis.

Results

Sample characteristics

Our sample consisted of German participants, 64% of whom reporting being married or in a committed relationship and 36% reported being single. Further, 17.3% perceived their financial status to be poor or very poor, 79% reported having an average or good financial status, and 36.8% reported having a good or very good financial status. Table 2 shows means and standard deviations for all study variables. Female participants had significantly higher scores on rumination, catastrophizing, total scores of mCER (T_2), and HA (at T_0 and T_2) than male participants, whereas non-planning impulsivity (T_1) was significantly higher among men.

Table 2
Means and standard deviations for variables of the study across three measurement points.

	Total ($N = 261$)	Female ($n = 147$)	Male ($n = 114$)	t
	M (SD)	M (SD)	M (SD)	
Age in years at T_0	15.03 (2.28)	15.22 (2.27)	14.80 (2.28)	1.48
Age in years at T_1	19.43 (2.34)	19.59 (2.36)	19.23 (2.30)	1.25
Age in years at T_2	24.93 (2.42)	25.10 (2.45)	24.73 (2.37)	1.22
Harm avoidance (T_0)				
JTCI/TCI[a]	0.0 (1.0)	0.14 (1.00)	−0.18 (0.97)	2.55*
JTCI[b]	6.61 (3.86)	7.06 (3.93)	6.09 (3.75)	1.42
TCI[c]	14.35 (6.95)	15.44 (6.90)	12.80 (6.79)	2.18*
Harm avoidance (T_2)				
TCI	8.37 (3.65)	9.11 (3.60)	7.41 (3.50)	−3.83***
Inhibitory control (T_1)				
Total score (BIS)	64.29 (9.81)	63.45 (10.46)	65.37 (8.84)	−1.57
Attentional (BIS)	16.28 (3.21)	16.33 (3.47)	16.23 (2.83)	0.25
Motor (BIS)	23.10 (4.37)	22.73 (4.62)	23.58 (4.00)	−1.56
Non-planning (BIS)	24.90 (4.40)	24.39 (4.58)	25.56 (4.08)	−2.14*
SSRT	331.17 (69.61)	333.82 (69.61)	327.76 (69.76)	0.69
Mean interference (ES)	1.46 (26.03)	1.11 (23.86)	1.92 (28.69)	−0.25
CERQ (T_2)				
Self-blame	2.99 (2.13)	3.12 (2.34)	2.83 (1.82)	1.06
Rumination	4.20 (2.72)	4.61 (2.81)	3.67 (2.52)	2.80**
Catastrophizing	2.28 (2.18)	2.52 (2.26)	1.97 (2.05)	1.97*
Blaming others	2.09 (2.08)	2.14 (2.24)	2.03 (1.86)	0.45
Total maladaptive	11.56 (6.61)	12.38 (7.08)	10.51 (5.81)	2.28*

Note. The first level of assessment (T_0); the first follow up (T_1); the second follow up (T_2); Junior Temperament and Character Inventory (JTCI); Temperament and Character Inventory (TCI); Barratt Impulsiveness Scale (BIS); Stop-Signal Reaction Time (SSRT); Emotional Stroop (ES); Cognitive Emotion Regulation Questionnaire (CERQ).
*$p \leq .05$. **$p \leq .01$. ***$p \leq .001$.
[a] Aggregated z scores of harm avoidance (JTCI/TCI) for the total sample.
[b] Harm avoidance measured among individuals with age $T_0 < 16$.
[c] Harm avoidance measured among individuals with age $T_0 \geq 16$.

Correlational analysis

Table 3 represents the correlation coefficients between the study variables. Female adolescents' HA (at T_0) significantly correlated with lower inhibitory control at T_1 as measured with attentional impulsivity (BIS). Among male adolescents, HA was associated with a better inhibitory control as shown by lower emotional interference (measured with ES). However, HA did not show any significant relationship to other measures of inhibitory control. Regarding emotion regulation, female adolescents' HA (at T_0) significantly predicted higher levels of total mCER, self-blame, rumination, catastrophizing, and blaming others after 10 years (at T_2). Further, there was a cross-sectional correlation between adult females' HA and all mCER strategies (both measured at T_2). Among male adolescents, HA at T_0 was associated with higher scores on the mCER strategy catastrophizing, and HA at T_2 was associated with higher levels of all mCER strategies except for blaming others. In addition, among inhibitory control measures, female adolescents' attentional impulsivity (at T_1) was significantly correlated with higher scores on rumination, self-blame, catastrophizing, and blaming others in their early adulthood (at T_2) whereas, among male adolescents, attentional impulsivity was only associated with rumination.

Moderated mediation analysis

In keeping with new quantitative texts (A. F. Hayes, 2013) that reject the necessity of fulfilling the assumptions from Baron and Kenny (1986) for conducting a mediation analysis, we included all mediators in the model simultaneously. We further added the effect of gender as moderator to the pathways that included HA as predictor (see Fig. 1). We also analyzed age and financial status as covariates. Additionally, we controlled for HA at T_2 to test if the effect of HA at T_0 on mCER is longitudinal or if it is a result of stability in HA. Regarding the first stage of moderated mediation analysis (path a in Fig. 1), results showed that HA × gender interaction did not significantly predict inhibitory control measures ($p \geq .10$), but there was a conditional direct effect of HA on inhibitory control as measured with attentional impulsivity. That is, HA predicted higher levels of attentional impulsivity among female adolescents but not among male adolescents (Table 4). Similarly, the effect of HA on emotional interference was conditioned on gender. In other words, HA was associated with lower levels of emotional interference among men (B = −6.18, $SE = 2.51$, $t = -2.46$, $p = .014$), while this effect was not significant for women (B = −0.69, $SE = 2.15$, t = −0.32, $p = .75$). The coefficients for the effects of HA on other inhibitory control measures were not significant ($p \geq .10$) and were also not gender-dependent. Further, adolescents' attentional impulsivity was significantly associated with higher levels of mCER in their early adulthood (B = .43, $SE = 0.14$, 95% CI [0.25−0.82], $p < .01$), while non-planning impulsivity showed a small association with lower levels of mCER (B = −.022, $SE = 0.11$, 95% CI [−0.43 to −0.01], $p = .04$). Emotional interference, SSRT and motor impulsivity showed an insignificant association with mCER ($p \geq .11$). Further, HA × gender interaction did not predict mCER but results showed that, after controlling for HA at T_2, there was a conditional total effect of HA at T_0 on mCER (Table 5). That is, female adolescents' HA significantly predicted higher mCER in their early adulthood. Table 5 illustrates the results of the second stage of moderated mediation analysis for attentional impulsivity as mediator (path b and c in Fig. 1).

Further, results revealed a conditional indirect effect in the predicted direction (Table 5). Among females, attentional impulsivity mediated the link between HA and mCER, but this indirect effect was not significantly different from zero among males. The results indicate a full mediation, given that the effect of HA on mCER became insignificant after controlling for attentional impulsivity (B = 0.92, $p = .09$). Additionally, confidence intervals for indirect effects of other inhibitory measures

Table 3
Correlation coefficients for study variables among female and male participants ($N = 261$).

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1. Age (T_0)	–	.08	−.07	−.01	.01	.08	−.12	−.05	.16*	.03	−.08	−.16	−.13	−.11
2. Harm avoidance (T_0)[a]	−.01	–	.43***	.06	.24**	−.10	.06	.07	−.02	.22**	.18*	.22**	.19*	.27***
3. Harm avoidance (T_2)	−.02	.34***	–	.04	.25**	−.14	.04	−.03	.05	.23**	.24**	.37***	.30***	.39***
Inhibition (T_1)														
4. Total (BIS)	−.14	.12	−.10	–	.76***	.86***	.84***	−.15	.08	.10	.12	.11	.10	.15
5. Attentional (BIS)	−.12	.15	.00	.73***	–	.51***	.46***	−.15	.09	.19*	.21**	.27***	.18*	.29***
6. Motor (BIS)	−.05	.01	−.14	.86***	.51***	–	.57***	−.12	.05	.07	.10	.02	.01	.07
7. Non-planning (BIS)	−.16	.13	−.09	.82***	.38***	.53***	–	−.10	.06	.01	.02	.03	.08	.05
8. SSRT	.05	−.02	.17	.11	.20*	.15	−.04	–	−.04	.09	−.11	−.10	−.04	−.06
9. Mean interference (ES)	−.01	−.21*	−.04	.00	−.02	−.02	.04	.10	–	.04	.09	.09	.07	.10
CERQ (T_2)														
10. Self-Blame	.15	.10	.26**	−.03	.06	.01	−.10	−.02	−.07	–	.40***	.48***	.13	.68***
11. Rumination	−.01	.06	.19*	.09	.20*	.12	−.06	.07	.08	.34***	–	.57***	.31***	.81***
12. Catastrophizing	.02	.22*	.24**	.06	.15	−.00	.02	.10	.06	.27***	.44***	–	.39***	.82***
13. Blaming others	.09	.13	.00	.03	.11	.01	−.01	.14	.01	.02	.27**	.52***	–	.61***
14. Total maladaptive	.08	.17	.25**	.06	.19*	.06	−.05	.11	.04	.57***	.79***	.80***	.63***	–

Note. Correlation coefficients between study variables among female participants are presented above the diagonal and those of male participants are presented below the diagonal.
*p ≤ .05. **p ≤ .01. ***p ≤ .001.
[a] Aggregated z score of harm avoidance (JTCI/TCI) for the total sample. The first level of assessment (T_0); the first follow up (T_1); the second follow up (T_2); Barratt Impulsiveness Scale (BIS); Stop-Signal Reaction Time (SSRT); Emotional Stroop (ES); Cognitive Emotion Regulation Questionnaire (CERQ).

Table 4
Regression results for the first-stage pathway of moderated mediation model (path a in Fig. 1) with attentional impulsivity as mediator.

Predictors	Outcome	R	R^2	F	P
	Attentional impulsivity (T_1)	.25	.06	2.80	.01
		B	SE	t	p
Constant		17.82	1.59	11.23	<.001
Age		−0.06	0.09	−0.68	.50
Financial status		−0.35	0.23	−1.47	.14
Harm avoidance (T_2)		0.07	0.06	1.10	.27
Harm avoidance (T_0)		0.36	0.31	1.16	.25
Gender		−0.23	0.41	−0.55	.58
Harm avoidance × Gender		0.35	0.40	0.88	.38
Conditional effect of harm avoidance (at T_0) on attentional impulsivity					
Men		0.36	0.31	1.16	.25
Women		0.72	0.28	2.60	.01

Note. All path coefficients are unstandardized. The first level of assessment (T_0); the first follow up (T_1); the second follow up (T_2).

Table 5
Regression results of moderated mediation model for the pathways predicting mCER (paths b and c in Fig. 1), along with conditional total, direct, and indirect effects of harm avoidance on mCER.

Predictors	Outcome	*R*	R^2	*F*	*P*
	mCER (T_2)	.43	.18	5.55	<.001
		B	*SE*	*t*	*p*
Constant		16.77	4.75	3.53	<.001
Age		−0.11	0.17	−0.69	.49
Financial status		−1.44	0.46	−3.16	<.01
Harm avoidance (T_2)		0.45	0.12	3.88	<.001
Harm avoidance (T_0)		0.52	0.60	0.86	.39
Attentional impulsivity (T_1)		0.43	0.14	2.99	<.01
Gender		0.42	0.78	0.54	.59
Harm avoidance × Gender		0.41	0.77	0.53	.60
Conditional total effect of harm avoidance (T_0) on mCER					
Men		0.42	0.60	0.70	.48
Women		1.09	0.53	2.03	.04
Conditional direct effect of harm avoidance (T_0) on mCER, controlling for attentional impulsivity					
Men		0.52	0.60	0.86	.39
Women		0.92	0.54	1.71	.09
Conditional indirect effect of harm avoidance (T_0) on mCER through attentional impulsivity					
		Effect	Boot *SE*[a]	Boot CI[b]	
Men		0.16	0.14	−.05 to .52	
Women		0.31	0.18	.05 to .78	

Note. All path coefficients are unstandardized.
[a] Bias corrected bootstrapped standard error.
[b] Bias corrected bootstrapped confidence interval. The first level of assessment (T_0); the first follow up (T_1); the second follow up (T_2); maladaptive cognitive emotion regulation (mCER).

included zero for both men and women, which suggests that these measures did not significantly mediate the link between HA and mCER. Examination of covariates indicated that non-planning impulsivity was higher among men (B = −1.22, *SE* = .55, *t* = −2.18, *p* = .03) and that it also slightly reduced with age (B = −0.25, *SE* = 0.12, *t* = −2.14 *p* = .03). In addition, a worse financial status significantly predicted higher levels of motor impulsivity (B = −1.13, *SE* = 0.32, *t* = −3.56 *p* < .001), non-planning impulsivity (B = −0.64, *SE* = 0.32, *t* = −1.99 *p* = .048), and mCER (B = −1.44, *SE* = 0.46, *t* = −3.16, *p* < .01).

Discussion

In the current study, we investigated the influence of adolescent HA (T_0) on young adulthood mCER over a period of 10 years. In addition, we explored the indirect effect of various measures of inhibitory control on relationship between HA and mCER. First, we found that female adolescents' HA significantly predicted higher levels of mCER in their early adulthood. We also found that this link was mediated by attentional impulsivity.

Our first main finding showed that adolescents' HA was associated with a higher score on strategies such as catastrophizing, rumination, self-blame, and blaming others after around 10 years, in early adulthood. However, this was true mostly among women, while HA was only associated with catastrophizing among male participants. However, the cross-sectional correlations between adulthood HA and mCER strategies were significant among both women and men. Importantly, the longitudinal effect of HA (T_0) on mCER was still significant after controlling for HA at T_2. Our findings were in accordance with Schreiber et al. (2012), who found a significant positive relationship between HA and emotion dysregulation. Similarly, Manfredi et al. (2011) reported a cross-sectional association between HA and higher rumination scores. However, the above mentioned studies did not include various mCER and also did not consider gender effect in their analysis. In accordance with previous arguments, our findings highlight the importance of differentiating effect of gender when studying HA associations (Li et al., 2012). One reason for this gender effect might be related to women reporting higher levels of HA (Al-Halabí et al., 2011) and mCER strategies (Zlomke & Hahn, 2010). Another explanation might rely on men and women's different approaches toward stressors (Tamres et al., 2002). In a meta-analysis, Tamres et al. (2002) showed that women appraise stressors as being more severe than men. Women also used more rumination and self-blame and engaged more often in uncontrollable stressors, while men tended to withdraw or avoid such situations. This approach can put female adolescents at risk of increased emotional distress particularly during the period between adolescence and young adulthood when they face important developmental stressors and are required to make important decisions regarding educational, occupational, and emotional aspects of life (Roisman, Masten, Coatsworth, & Tellegen, 2004). While adolescents come across novel and stressful situations that require an increased level of regulatory effort (Garnefski & Kraaij, 2006), HA with its associated fear of uncertainty and negative bias, might lead to more emotional distress among women with high HA and facilitate using mCER. It is important for future research to investigate the possibility if male adolescents with high HA might experience less emotional distress than their female counterparts and therefore apply mCER less frequently, or if they deal with their distress through other strategies that have not been included in the current study.

Our second main finding demonstrated that lack of attentional focus and experiencing intrusive thoughts—as measured by the attentional impulsivity subscale of BIS—mediated the link between HA and mCER. This finding was consistent with Schreiber et al. (2012), who found a significant correlation between HA and higher levels of both attentional impulsivity and emotion regulation difficulties. Negative affectivity related to HA might contribute to impulsive attention (Smallwood, Fitzgerald, Miles, & Phillips, 2009). Similarly, the avoidance tendency associated with HA might lead to impulsive attention as a method for avoiding unwanted experiences (Berghoff, Pomerantz, Pettibone, Segrist, & Bedwell, 2012). The avoidance tendency can also lead to thought intrusion and thought disinhibition (Wegner, 1994, 1997), which in turn can facilitate mCER such as rumination as a method to understand and process those uninhibited thoughts.

Thus, our findings imply the importance of a specific inhibitory mechanism in the context of HA and mCER. Behavioral tasks and self-report measures of inhibitory control have different underlying mechanisms (Reynolds et al., 2006), as reflected in the insignificant or low bivariate correlation between measures of BIS, SST, and ES in previous research (Cheung, Mitsis, & Halperin, 2004; Enticott et al., 2006). While performance-based measures of inhibitory control are influenced by temporary fluctuations, self-report measures cover broad periods of time and measure a more stable (trait-dependent) aspect of inhibition (Dougherty, Mathias, Marsh, & Jagar, 2005). Therefore, less stability and more state-dependency of behavioral measures might play a role in low or insignificant correlations between HA and behavioral measures of inhibitory control, particularly over long intervals. Additionally, in accordance with past theories (Matthews & Deary, 2000b, pp. 70–90), performance in these tasks might be more influenced by the negative affective state rather than the trait. In other words, the effect of trait HA on performance-based inhibitory control tasks might be mediated by the state negative affect while doing the task. Future studies can answer this question by assessing state negative affect before or after completing tasks. Further, in this study, we applied the Emotional Stroop and Stop-Signal Task that measure the controlled, deliberate suppression of prepotent response rather than reactive inhibition, which seems to be a residual aftereffect of processing, is unintentional, and is measured through other tasks such as negative priming (Miyake et al., 2000). Considering that HA is associated with an automatic pattern of attending to stimuli, HA might be related to less intentional kind of inhibition deficit that is not assessed in Emotional Stroop and Stop-Signal Task, but might be recognizable by the person and therefore reflected in a self-report measure. In line with this assumption, Taylor et al. (2008) have argued that the attentional impulsivity subscale might represent some levels of disturbances in executive functioning.

Further, our study provides gender and measure-specific findings for the association between HA and inhibitory control. We found that HA was associated with more trait-based inhibitory control—as measured with attentional impulsivity—among female adolescents, while HA was associated with better performance-based inhibitory control among male adolescents——as measured with Emotional Stroop. This finding is in line with gender socialization theory, according to which men learn to have more control over their emotions and use more active and instrumental coping behaviors; while women use more passive and emotion-focused coping strategies, because their traditional role does not prescribe emotional inhibition (Matud, 2004). It can be argued that male adolescents might have counteracted the influence of HA by developing more emotional inhibition as a response to socialization processes that make it difficult for men to accept and express fear and weakness. However, these associations were moderate to small and need to be replicated in future studies. Additionally, testing this hypothesis among a clinical sample of adolescents might yield different results. Finally, although this finding should be interpreted with caution, it suggests that HA may lead to gender-specific inhibitory control outcomes.

Our results should be concluded by considering several limitations of this study. First, the lack of multiple assessments for inhibitory control and mCER restricts conclusions about cause-effect relationships. Therefore, further research needs to extend

and examine our results, applying multiple measurements of each construct. Second, we measured HA using TCI and its adapted version JTCI to measure HA in adolescents older and younger than 16 years old, respectively. Although both measures have similar psychometric features and identical scales (Luby et al., 1999), this aspect of our methods might limit our ability to draw firm conclusions. Third, self-report measures limit the interpretation of the results due to possible interfering factors such as social desirability. Fourth, the extent to which our results can be generalized beyond the scope of our sample remains unclear.

In spite of these limitations, the current study supports the importance of adolescent HA as a predictor of female adults' mCER. Further, although the effect size was small, the current study offers new insight in the specific but minor role of attentional impulsivity in the association between HA and mCER. HA is considerably stable across the lifespan (Josefsson et al., 2013), and its maladaptive influence might be better controlled by targeting mediating pathways. Early recognition of temperamental risk factors permits the possibility of preventing pathological trajectories by providing early interventions (Hirshfeld-Becker & Biederman, 2002). While effective emotion regulation has been identified as an optimal target for psychological intervention (Barnow et al., 2014), the present study suggests that focusing on enhancement of attentional control might have a buffering effect against development of mCER. It has been shown that clinical interventions such as mindfulness facilitate the application of higher level executive attention for regulating automatic emotional responses (Jha, Krompinger, & Baime, 2007) and improve both attentional control (Lattimore, Fisher, & Malinowski, 2011) and emotion regulation ability (A. M. Hayes & Feldman, 2004). Future research should replicate this study by including multiple measurements of each construct and using measures of less intentional inhibition. Further, we used a community-based sample and replicating this study with a clinical sample would provide valuable complementary information.

Acknowledgment

The Greifswald family study was supported by the German Research Foundation (Deutsche Forschungsgemeinschaft, BA-2172/2-1, BA2172.8-1. GR1912.7-1).

References

Al-Halabí, S., Herrero, R., Sáiz, P. A., García-Portilla, M. P., Errasti, J. M., Corcoran, P., et al. (2011). A cross-cultural comparison between Spain and the USA: Temperament and character distribution by sex and age. *Psychiatry Research, 186*, 397–401. http://dx.doi.org/10.1016/j.psychres.2010.07.021.

Aldao, A., & Nolen-Hoeksema, S. (2010). Specificity of cognitive emotion regulation strategies: A transdiagnostic examination. *Behaviour Research and Therapy, 48*, 974–983. http://dx.doi.org/10.1016/j.brat.2010.06.002.

Aldinger, M., Stopsack, M., Ulrich, I., Appel, K., Reinelt, E., Wolff, S., et al. (2014). Neuroticism developmental courses – Implications for depression, anxiety and everyday emotional experience: A prospective study from adolescence to young adulthood. *BMC Psychiatry, 14*, 210. http://dx.doi.org/10.1186/s12888-014-0210-2.

Barnow, S., Aldinger, M., Ulrich, I., & Stopsack, M. (2013). Emotion regulation in depression: An overview of results using various methods. *Psychologische Rundschaw, 64*, 235–243. http://dx.doi.org/10.1026/0033-3042/a000172.

Barnow, S., Löw, C. A., Dodek, A., & Stopsack, M. (2014). Managing emotions-emotions under control [Gefühle im Griff-Emotionen intelligent regulieren]. *Psychotherapie, Psychosomatik, medizinische Psychologie, 64*, 284–289. http://dx.doi.org/10.1055/s-0033-1363683.

Barnow, S., Rüge, J., Spitzer, C., & Freyberger, H. J. (2005). Temperament and character in individuals with borderline personality disorder [Temperament und Charakter bei Personen mit Borderline-Persönlichkeitsstörung]. *Der Nervenarzt, 76*, 839–848. http://dx.doi.org/10.1007/s00115-004-1810-8.

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*, 1173–1182. http://dx.doi.org/10.1037/0022-3514.51.6.1173.

Berghoff, C. R., Pomerantz, A. M., Pettibone, J. C., Segrist, D. J., & Bedwell, D. R. (2012). The relationship between experiential avoidance and impulsiveness in a nonclinical sample. *Behaviour Change, 29*, 25–35. http://dx.doi.org/10.1017/bec.2012.6.

Butler, L., & Nolen-Hoeksema, S. (1994). Gender differences in responses to depressed mood in a college sample. *Sex Roles, 30*, 331–346. http://dx.doi.org/10.1007/BF01420597.

Caci, H., Robert, P., & Boyer, P. (2004). Novelty seekers and impulsive subjects are low in morningness. *European Psychiatry, 19*, 79–84. http://dx.doi.org/10.1016/j.eurpsy.2003.09.007.

Carleton, R. N., Sharpe, D., & Asmundson, G. J. G. (2007). Anxiety sensitivity and intolerance of uncertainty: Requisites of the fundamental fears? *Behaviour Research and Therapy, 45*, 2307–2316. http://dx.doi.org/10.1016/j.brat.2007.04.006.

Cheung, A. M., Mitsis, E. M., & Halperin, J. M. (2004). The relationship of behavioral inhibition to executive functions in young adults. *Journal of Clinical and Experimental Neuropsychology, 26*, 393–404. http://dx.doi.org/10.1080/13803390490510103.

Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants: A proposal. *Archives of General Psychiatry, 44*, 573–588. http://dx.doi.org/10.1001/archpsyc.1987.01800180093014.

Cloninger, C. R. (1994a). *The temperament and character inventory (TCI): A guide to its development and use*. St. Louis, MO: Center for Psychobiology of Personality, Washington University.

Cloninger, C. R. (1994b). Temperament and personality. *Current Opinion in Neurobiology, 4*, 266–273. http://dx.doi.org/10.1016/0959-4388(94)90083-3.

Cloninger, C. R., Bayon, C., & Svrakic, D. M. (1998). Measurement of temperament and character in mood disorders: A model of fundamental states as personality types. *Journal of Affective Disorders, 51*, 21–32. http://dx.doi.org/10.1016/S0165-0327(98)00153-0.

Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry, 50*, 975–990. http://dx.doi.org/10.1001/archpsyc.1993.01820240059008.

Cloninger, C. R., Svrakic, D. M., & Przybeck, T. R. (2006). Can personality assessment predict future depression? A twelve-month follow-up of 631 subjects. *Journal of Affective Disorders, 92*, 35–44. http://dx.doi.org/10.1016/j.jad.2005.12.034.

Cole, P. M. (2014). Moving ahead in the study of the development of emotion regulation. *International Journal of Behavioral Development, 38*, 203–207. http://dx.doi.org/10.1177/0165025414522170.

De Lissnyder, E., Koster, E. H. W., Goubert, L., Onraedt, T., Vanderhasselt, M.-A., & De Raedt, R. (2012). Cognitive control moderates the association between stress and rumination. *Journal of Behavior Therapy and Experimental Psychiatry, 43*, 519–525. http://dx.doi.org/10.1016/j.jbtep.2011.07.004.

Dempster, F. N. (1992). The rise and fall of the inhibitory mechanism: Toward a unified theory of cognitive development and aging. *Developmental Review, 12*, 45–75. http://dx.doi.org/10.1016/0273-2297(92)90003-K.

Donaldson, C., Lam, D., & Mathews, A. (2007). Rumination and attention in major depression. *Behaviour Research and Therapy, 45*, 2664–2678. http://dx.doi.org/10.1016/j.brat.2007.07.002.

Dougherty, D. M., Mathias, C. W., Marsh, D. M., & Jagar, A. A. (2005). Laboratory behavioral measures of impulsivity. *Behavior Research Methods, 37*, 82–90. http://dx.doi.org/10.3758/bf03206401.

Elphinston, R. A., Feeney, J. A., Noller, P., Connor, J. P., & Fitzgerald, J. (2013). Romantic jealousy and relationship satisfaction: The costs of rumination. *Western Journal of Communication, 77*, 293–304. http://dx.doi.org/10.1080/10570314.2013.770161.
Enticott, P. G., Ogloff, J. R. P., & Bradshaw, J. L. (2006). Associations between laboratory measures of executive inhibitory control and self-reported impulsivity. *Personality and Individual Differences, 41*, 285–294. http://dx.doi.org/10.1016/j.paid.2006.01.011.
Eysenck, M. W. (1982). *Attention and arousal: Cognition and performance*. New York: Springer.
Garnefski, N., & Kraaij, V. (2006). Relationships between cognitive emotion regulation strategies and depressive symptoms: A comparative study of five specific samples. *Personality and Individual Differences, 40*, 1659–1669. http://dx.doi.org/10.1016/j.paid.2005.12.009.
Garnefski, N., Kraaij, V., & Spinhoven, P. (2001). Negative life events, cognitive emotion regulation and emotional problems. *Personality and Individual Differences, 30*, 1311–1327. http://dx.doi.org/10.1016/S0191-8869(00)00113-6.
Gilbert, P., & Miles, J. N. V. (2000). Sensitivity to social put-down: It's relationship to perceptions of social rank, shame, social anxiety, depression, anger, and self-other blame. *Personality and Individual Differences, 29*, 757–774. http://dx.doi.org/10.1016/S0191-8869(99)00230-5.
Gunthert, K. C., Cohen, L. H., & Armeli, S. (1999). The role of neuroticism in daily stress and coping. *Journal of Personality and Social Psychology, 77*, 1087–1100. http://dx.doi.org/10.1037/0022-3514.77.5.1087.
Hahn, S., Buttaccio, D. R., Hahn, J., & Lee, T. (2015). Personality and attention: Levels of neuroticism and extraversion can predict attentional performance during a change detection task. *The Quarterly Journal of Experimental Psychology, 68*, 1041–1048. http://dx.doi.org/10.1080/17470218.2015.1032986.
Hansenne, M. (1999). P300 and personality: An investigation with the Cloninger's model. *Biological Psychology, 50*, 143–155. http://dx.doi.org/10.1016/S0301-0511(99)00008-3.
Hansenne, M., Pinto, E., Scantamburlo, G., Renard, B., Reggers, J., Fuchs, S., et al. (2003). Harm avoidance is related to mismatch negativity (MMN) amplitude in healthy subjects. *Personality and Individual Differences, 34*, 1039–1048. http://dx.doi.org/10.1016/s0191-8869(02)00088-0.
Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. Guilford Press.
Hayes, A. M., & Feldman, G. (2004). Clarifying the construct of mindfulness in the context of emotion regulation and the process of change in therapy. *Clinical Psychology: Science and Practice, 11*, 255–262. http://dx.doi.org/10.1093/clipsy.bph080.
Henderson, L. (2002). Fearfulness predicts self-blame and shame in shyness. *Personality and Individual Differences, 32*, 79–93. http://dx.doi.org/10.1016/S0191-8869(01)00007-1.
Hirshfeld-Becker, D. R., & Biederman, J. (2002). Rationale and principles for early intervention with young children at risk for anxiety disorders. *Clinical Child and Family Psychology Review, 5*, 161–172. http://dx.doi.org/10.1023/A:1019687531040.
Izadpanah, S., Schumacher, M., Bähr, A., Stopsack, M., Grabe, H. J., & Barnow, S. (2016). A 5-year longitudinal study of the adolescent reinforcement sensitivity as a risk factor for anxiety symptoms in adulthood: Investigating the indirect effect of cognitive emotion regulation. *Personality and Individual Differences, 95*, 68–73. http://dx.doi.org/10.1016/j.paid.2016.02.021.
Jha, A., Krompinger, J., & Baime, M. (2007). Mindfulness training modifies subsystems of attention. *Cognitive, Affective, and Behavioral Neuroscience, 7*, 109–119. http://dx.doi.org/10.3758/CABN.7.2.109.
Joormann, J. (2006). Differential effects of rumination and dysphoria on the inhibition of irrelevant emotional material: Evidence from a negative priming task. *Cognitive Therapy and Research, 30*, 149–160. http://dx.doi.org/10.1007/s10608-006-9035-8.
Joormann, J. (2010). Cognitive inhibition and emotion regulation in depression. *Current Directions in Psychological Science, 19*, 161–166. http://dx.doi.org/10.1177/0963721410370293.
Joormann, J., & Gotlib, I. H. (2010). Emotion regulation in depression: Relation to cognitive inhibition. *Cognition and Emotion, 24*, 281–298. http://dx.doi.org/10.1080/02699930903407948.
Josefsson, K., Jokela, M., Cloninger, C. R., Hintsanen, M., Salo, J., Hintsa, T., et al. (2013). Maturity and change in personality: Developmental trends of temperament and character in adulthood. *Development and Psychopathology, 25*, 713–727. http://dx.doi.org/10.1017/S0954579413000126.
Jose, P. E., Wilkins, H., & Spendelow, J. S. (2012). Does social anxiety predict rumination and co-rumination among adolescents? *Journal of Clinical Child and Adolescent Psychology, 41*, 86–91. http://dx.doi.org/10.1080/15374416.2012.632346.
Khng, K. H., & Lee, K. (2014). The relationship between stroop and stop-signal measures of inhibition in adolescents: Influences from variations in context and measure estimation. *Plos One, 9*, e101356. http://dx.doi.org/10.1371/journal.pone.0101356.
Kusunoki, K., Sato, T., Taga, C., Yoshida, T., Komori, K., Narita, T., et al. (2000). Low novelty-seeking differentiates obsessive-compulsive disorder from major depression. *Acta Psychiatrica Scandinavica, 101*, 403–405. http://dx.doi.org/10.1034/j.1600-0447.2000.101005403.x.
Lattimore, P., Fisher, N., & Malinowski, P. (2011). A cross-sectional investigation of trait disinhibition and its association with mindfulness and impulsivity. *Appetite, 56*, 241–248. http://dx.doi.org/10.1016/j.appet.2010.12.007.
Liao, K. Y. H., & Wei, M. F. (2011). Intolerance of uncertainty, depression, and anxiety: The moderating and mediating roles of rumination. *Journal of Clinical Psychology, 67*, 1220–1239. http://dx.doi.org/10.1002/Jclp.20846.
Li, Y., Qin, W., Jiang, T. Z., Zhang, Y. T., & Yu, C. S. (2012). Sex-dependent correlations between the personality dimension of harm avoidance and the resting-state functional connectivity of amygdala subregions. *Plos One, 7*. http://dx.doi.org/10.1371/journal.pone.0035925.
Loch, N., Hiller, W., & Witthöft, M. (2011). Der cognitive emotion regulation questionnaire (CERQ). *Zeitschrift für Klinische Psychologie und Psychotherapie, 40*, 94–106. http://dx.doi.org/10.1026/1616-3443/a000079.
Logan, G. D. (1994). On the ability to inhibit thought and action. In D. Dagenbach, & T. H. Carr (Eds.), *Inhibitory processes in attention* (pp. 189–239). London: Academic Press.
Logan, G. D., Schachar, R. J., & Tannock, R. (1997). Impulsivity and inhibitory control. *Psychological Science, 8*, 60–64. http://dx.doi.org/10.1111/j.1467-9280.1997.tb00545.x.
Luby, J. L., Svrakic, D. M., McCallum, K., Przybeck, T. R., & Cloninger, C. R. (1999). The junior temperament and character inventory: Preliminary validation of a child self-report measure. *Psychological Reports, 84*, 1127–1138. http://dx.doi.org/10.2466/pr0.1999.84.3c.1127.
Lyoo, I. K., Lee, D. W., Kim, Y. S., Kong, S. W., & Kwon, J. S. (2001). Patterns of temperament and character in subjects with obsessive-compulsive disorder. *The Journal of Clinical Psychiatry, 62*, 637–641. http://dx.doi.org/10.4088/JCP.v62n0811.
Mackinnon, D. P., Lockwood, C. M., & Williams, J. (2004). Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioral Research, 39*, 99. http://dx.doi.org/10.1207/s15327906mbr3901_4.
Manfredi, C., Caselli, G., Rovetto, F., Rebecchi, D., Ruggiero, G. M., Sassaroli, S., et al. (2011). Temperament and parental styles as predictors of ruminative brooding and worry. *Personality and Individual Differences, 50*, 186–191. http://dx.doi.org/10.1016/j.paid.2010.09.023.
Mardaga, S., & Hansenne, M. (2009). Autonomic aspect of emotional response in depressed patients: Relationships with personality. *Clinical Neurophysiology, 39*, 209–216. http://dx.doi.org/10.1016/j.neucli.2009.06.002.
Martin, R. C., & Dahlen, E. R. (2005). Cognitive emotion regulation in the prediction of depression, anxiety, stress, and anger. *Personality and Individual Differences, 39*, 1249–1260. http://dx.doi.org/10.1016/j.paid.2005.06.004.
Matthews, G., & Deary, I. J. (2000a). Personality: Performance and information processing. In *Personality traits* (pp. 220–242). Cambridge: Cambridge University Press.
Matthews, G., & Deary, I. J. (2000b). Stable traits and transient states. In *Personality traits* (pp. 70–90). Cambridge: Cambridge University Press.
Matthews, G., Joyner, L., Gilliland, K., Huggins, J., & Falconer, S. (1999). Validation of a comprehensive stress state questionnaire: Towards a state big three? In I. Merville, I. J. Deary, a. F. DeFruyt, & F. Ostendorf (Eds.), *Personality psychology in Europe* (pp. 335–350). Tilburg: Tilburg University.
Matud, M. P. (2004). Gender differences in stress and coping styles. *Personality and Individual Differences, 37*, 1401–1415. http://dx.doi.org/10.1016/j.paid.2004.01.010.
Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*, 49–100. http://dx.doi.org/10.1006/cogp.1999.0734.
Most, S. B., Chun, M. M., Johnson, M. R., & Kiehl, K. A. (2006). Attentional modulation of the amygdala varies with personality. *Neuroimage, 31*, 934–944. http://dx.doi.org/10.1016/j.neuroimage.2005.12.031.

Most, S. B., Chun, M. M., Widders, D. M., & Zald, D. H. (2005). Attentional rubbernecking: Cognitive control and personality in emotion-induced blindness. *Psychonomic Bulletin and Review, 12*, 654–661. http://dx.doi.org/10.3758/bf03196754.
Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology. *Psychological Bulletin, 126*, 220–246. http://dx.doi.org/10.1037/0033-2909.126.2.220.
Nolen-Hoeksema, S., & Aldao, A. (2011). Gender and age differences in emotion regulation strategies and their relationship to depressive symptoms. *Personality and Individual Differences, 51*, 704–708. http://dx.doi.org/10.1016/j.paid.2011.06.012.
Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. *Journal of Clinical Psychology, 51*, 768–774. http://dx.doi.org/10.1002/1097-4679(199511)51, 6<768::Aid-Jclp2270510607>3.0.Co;2–1.
Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*, 879–891. http://dx.doi.org/10.3758/BRM.40.3.879.
Preacher, K. J., Rucker, D. D., & Hayes, A. F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research, 42*, 185–227. http://dx.doi.org/10.1080/00273170701341316.
Reynolds, B., Ortengren, A., Richards, J. B., & de Wit, H. (2006). Dimensions of impulsive behavior: Personality and behavioral measures. *Personality and Individual Differences, 40*, 305–315. http://dx.doi.org/10.1016/j.paid.2005.03.024.
Richter, J., Eisemann, M., & Richter, G. (2000). Zur deutschsprachigen Version des Temperament- und Charakterinventars [A German version of temperamnt and character inventory]. *Zeitschrift für Klinische Psychologie und Psychotherapie, 29*, 117–126. http://dx.doi.org/10.1026//0084-5345.29.2.117.
Roisman, G. I., Masten, A. S., Coatsworth, J. D., & Tellegen, A. (2004). Salient and emerging developmental tasks in the transition to adulthood. *Child Development, 75*, 123–133. http://dx.doi.org/10.1111/j.1467-8624.2004.00658.x.
Schmeck, K., Goth, K., Poustka, F., & Cloninger, R. C. (2001). Reliability and validity of the junior temperament and character inventory. *International Journal of Methods in Psychiatric Research, 10*, 172–182. http://dx.doi.org/10.1002/mpr.113.
Schreiber, L. R., Grant, J. E., & Odlaug, B. L. (2012). Emotion regulation and impulsivity in young adults. *Journal of Psychiatry Research, 46*, 651–658. http://dx.doi.org/10.1016/j.jpsychires.2012.02.005.
Smallwood, J., Fitzgerald, A., Miles, L. K., & Phillips, L. H. (2009). Shifting moods, wandering minds: Negative moods lead the mind to wander. *Emotion, 9*, 271–276. http://dx.doi.org/10.1037/a0014855.
Southam-Gerow, M. A., & Kendall, P. C. (2002). Emotion regulation and understanding: Implications for child psychopathology and therapy. *Clinical Psychology Review, 22*, 189–222. http://dx.doi.org/10.1016/S0272-7358(01)00087-3.
Tamres, L. K., Janicki, D., & Helgeson, V. S. (2002). Sex differences in coping behavior: A meta-analytic review and an examination of relative coping. *Personality and Social Psychology Review, 6*, 2–30. http://dx.doi.org/10.1207/s15327957pspr0601_1.
Taylor, C. T., Hirshfeld-Becker, D. R., Ostacher, M. J., Chow, C. W., LeBeau, R. T., Pollack, M. H., et al. (2008). Anxiety is associated with impulsivity in bipolar disorder. *Journal of Anxiety Disorders, 22*, 868–876. http://dx.doi.org/10.1016/j.janxdis.2007.09.001.
Tomko, R. L., Trull, T. J., Wood, P. K., & Sher, K. J. (2013). Characteristics of borderline personality disorder in a community sample: Comorbidity, treatment utilization, and general functioning. *Journal of Personality Disorders*, 1–17. http://dx.doi.org/10.1521/pedi_2013_27_093.
Tortella-Feliu, M., Balle, M., & Sesé, A. (2010). Relationships between negative affectivity, emotion regulation, anxiety, and depressive symptoms in adolescents as examined through structural equation modeling. *Journal of Anxiety Disorders, 24*, 686–693. http://dx.doi.org/10.1016/j.janxdis.2010.04.012.
Watson, D., & Clark, L. A. (1992). Affects separable and inseparable: On the hierarchical arrangement of the negative affects. *Journal of Personality and Social Psychology, 62*, 489–505. http://dx.doi.org/10.1037/0022-3514.62.3.489.
Wegner, D. M. (1994). Ironic processes of mental control. *Psychological Review, 101*, 34–52. http://dx.doi.org/10.1037/0033-295x.101.1.34.
Wegner, D. M. (1997). When the antidote is the poison: Ironic mental control processes. *Psychological Science, 8*, 148–150. http://dx.doi.org/10.1111/j.1467-9280.1997.tb00399.x.
Weierich, M. R., Treat, T. A., & Hollingworth, A. (2008). Theories and measurement of visual attentional processing in anxiety. *Cognition and Emotion, 22*, 985–1018. http://dx.doi.org/10.1080/02699930701597601.
Whitmer, A. J., & Banich, M. T. (2007). Inhibition versus switching deficits in different forms of rumination. *Psychological Science, 18*, 546–553. http://dx.doi.org/10.1111/j.1467-9280.2007.01936.x.
Zetsche, U., D'Avanzato, C., & Joormann, J. (2012). Depression and rumination: Relation to components of inhibition. *Cognition and Emotion, 26*, 758–767. http://dx.doi.org/10.1080/02699931.2011.613919.
Zetsche, U., & Joormann, J. (2011). Components of interference control predict depressive symptoms and rumination cross-sectionally and at six months follow-up. *Journal of Behavior Therapy and Experimental Psychiatry, 42*, 65–73. http://dx.doi.org/10.1016/j.jbtep.2010.06.001.
Zhang, W., Lu, J., Ni, Z., Liu, X., Wang, D., & Shen, J. (2013). Harm avoidance in adolescents modulates late positive potentials during affective picture processing. *International Journal of Developmental Neuroscience, 31*, 297–302. http://dx.doi.org/10.1016/j.ijdevneu.2013.03.009.
Zlomke, K. R., & Hahn, K. S. (2010). Cognitive emotion regulation strategies: Gender differences and associations to worry. *Personality and Individual Differences, 48*, 408–413. http://dx.doi.org/10.1016/j.paid.2009.11.007.

Erklärung

Erklärung gemäß § 8 (1) c) der Promotionsordnung der Universität Heidelberg für die Fakultät für Verhaltens- und Empirische Kulturwissenschaften
Declaration in accordance to § 8 (1) c) of the doctoral degree regulation of Heidelberg University, Faculty of Behavioural and Cultural Studies

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