Marien Lievaart

Short Fuses & Bad Tempers: Neurocognitive Insights in Trait Anger

Short Fuses & Bad Tempers: Neurocognitive Insights in Trait Anger

Marien Lievaart

Short Fuses & Bad Tempers: Neurocognitive Insights in Trait Anger

Cover design by Robert Kroon
Layout by Marien Lievaart
Printed by Stout Grafische Dienstverlening v.o.f.

ISBN: 978-90-75938-82-1

The research presented in this dissertation was funded by Antes Mental Health Care.

Short Fuses & Bad Tempers: Neurocognitive Insights in Trait Anger

Korte lontjes & vaatjes buskruit: Neurocognitieve inzichten in een dispositie tot woede

Proefschrift

ter verkrijging van de graad van doctor aan de Erasmus Universiteit Rotterdam

op gezag van de rector magnificus
Prof.dr. H.A.P. Pols

en volgens besluit van het College voor Promoties.

De openbare verdediging zal plaatsvinden op
9 september om 11.30

door
Marien Lievaart
geboren te Maassluis

ERASMUS UNIVERSITY ROTTERDAM

Promotiecommissie

Promotoren
Prof. dr. Johannes E. Hovens
Prof. dr. Ingmar H.A. Franken

Overige leden van de commissie
Prof. mr. dr. Eric Rassin
Prof. dr. Jan W. van Strien
Prof. dr. Henk Nijman

Copromotoren
Dr. Frederik M. Van der Veen
Dr. Jorg Huijding

Contents

Chapter One

General introduction

A frustrated father who suddenly snaps and hits his child after several previous warnings. A previous loyal coworker who suddenly remains absent for several days at work due to disagreement with his boss. A married couple giving each other the silent treatment after repeated arguments. A usually reasonable man erratically flipping the bird after being cut off on the road. A war veteran resenting the army and harboring grudges after losing his legs and multiple friends after stepping on a landmine. A once beloved partner that gets killed after suspected infidelity.

These short vignettes described above, illustrate the variety of situations that can lead to and behavioral outcomes that can result from an emotion we are all familiar with, yet understand little about: anger[1]. Anger, in short, can be defined as "an emotional state that varies in intensity from mild irritation to intense fury and rage" (Spielberger, 1999 p. 1). Although little understood, its link with adverse outcomes is evident, making the understanding of anger a valuable enterprise. There is especially a need to better understand the key factors underlying a disposition towards anger, as individuals scoring high on this personality trait have impairments in their day-to-day functioning. To this purpose, the current thesis investigates the (neuro)cognitive aspects underlying anger and a disposition towards anger (i.e., trait anger). Before reviewing existing research on trait anger and its underpinnings in more detail, we first focus on anger as an emotional state. This is followed by a paragraph devoted to describing the clinical relevance of the construct trait anger in more detail. Next, a paragraph in which definitions and distinctions regarding constructs related to trait anger will be reviewed. Finally, cognitive models with respect to trait anger and reactive aggression will be discussed and an overview of the current thesis will be provided.

[1] DiGiuseppe and Trafrate (2010) highlight the scarcity of research around this topic and its neglect in clinical practice as evidenced by an absence of diagnostic categories for anger disorders, making it a challenging construct to define. Note that the intermittent explosive disorder has its main focus on aggressive behavior rather than anger.

1.1. Anger defined: its origins and its consequences

Anger is an emotion that is part of everyday live. Together with happiness, sadness and fear, anger is considered to be a basic emotion that is innate and universal to all humans (Ekman, 1999; Panksepp, 1998; Strongman, 2003)[2]. Diary studies conducted in community samples show that people report to experience and express mild to moderate anger from several times daily to at least several times a week (Averill, 1983; Kassinove, Sukhodolsky, Tsytsarev, & Solovyova, 1997; Meltzer, 1933; Schieman, 2010; Tafrate, Kassinove, & Dundin, 2002). Most of these angry episodes involve significant persons in our live that we love, like or rely on (Averill, 1983; Schieman, 2010). Anger can be elicited by a broad range of triggers of which perceived threats (to the self or to those one feels related to), deliberate harm-doings, disrespect, insults, injustice, unwanted actions of other people, and norm or rule violations are the most common (Potegal & Novaco, 2010). Especially in situations where triggers are appraised as preventable and intentional anger arises. Novaco (2011) also points out the intrinsic role threat perceptions play in the activation of anger. In line with this notion, it is found that the neurobiological threat system (amygdala, hypothalamus, and periaqueductal gray) becomes activated in conditions that elicit anger (Blair, 2012). Like other emotions, the intensity of anger is experienced on a continuous scale, ranging from mild forms of anger (e.g., irritation and annoyance) to severe forms (e.g., fury and rage)[3], and is usually short-lived (Potegal & Qiu, 2010). Typically, anger episodes last around five up till fifteen minutes (Potegal & Qiu, 2010). However, angry rumination and thoughts of revenge can prolong the timecourse of anger substantially (Novaco, 2011). Moreover, angry feelings can be experienced over longer period of times (from several hours up till several days) in less intense forms as well, often referred to as an angry mood (Fernandez, 2013).

---

[2] Although anger is considered to be a basic emotion that is universally expressed and recognized, the behavioural expression of anger at the same time is also evidently culturally modified, sometimes even cultural-specific, and governed by display rules (Matsumoto et al., 2010). Constructivists go as far as to suggest that anger is "a role or a socially constructed, reinforced behavioural script that we learn to play" (Kassinove & Tafrate, 2002, p. 16) and consider anger a fuzzy concept (Russell & Fehr, 1994) that cannot be defined with absolute certainty.

[3] Note that we conceptualize anger here as a single construct that can vary in intensity rather than qualitatively differentiating between forms of anger.

Anger as an emotional state is a multidimensional construct with its own distinct physiological and neurophysiological responses, sensations, feelings, cognitions, and action tendencies (DiGiuseppe & Tafrate, 2010). Phenomenologically, anger is often experienced as a "hot" emotion (Stemmler, 2010), i.e. feeling warm bodily sensations. Its facial expression is characterized by furrowed eyebrows and contraction of corrugator muscles (e.g., raised upper eyelid, tensing of lower eyelid, tightened lips) (Matsumoto, Hee Yoo, & Chung, 2010). Its vocal expression is identified by a fast, loud, and rising melody tone (Green, Whitney, & Gustafson, 2010). In terms of physiology, anger is characterized by the activation of both adrenaline and nor-adrenaline systems combined with strong vagal withdrawal, as reflected by an accelerated heartbeat and respiration rate, increased muscle tension, increased diastolic blood pressure, increased respiration rate, higher skin temperature, increases in skin conductance responses, and decreased total peripheral resistance (Stemmler, 2010). Prototypically, anger is accompanied or elicited by an appraisal of wrongdoing whereby blame is being externalized[4]. The action disposition usually associated with it is to counteract this perceived wrongdoing (c.f. to right the wrong). In that sense, anger is viewed as an approach-related emotion (Carver & Harmon-Jones, 2009). In this regard anger differentiates from other negatively valenced emotions, such as fear, guilt, disgust or sadness, as these emotions typically involve withdrawal motivation. However, we would like to note that under certain conditions during anger withdrawal can occur as well (e.g., psychological distancing, the silent treatment, refusal to cooperate). All in all, the whole constellation of responses associated with anger as described above implies that physical and psychological resources are mobilized during an anger episode to face adversity (e.g., the fight-flight response) and that this action readiness is signalized towards others as well as easily recognized by others (also see, Frijda, 1988). Correspondingly, anger can also be conceptualized as a moral emotion (e.g., Hutcherson & Gross, 2011), a relational emotion (e.g., Hall, 2009), a socially constructed emotion (Schieman, 2010), and even as a bestial passion (see Potegal & Novaco, 2010).

[4] Berkowitz (2010, 2012), however, convincingly argues that non-cognitive factors, such as pain, are important elicitors for the anger experience as well. He views blame placing as a by-product, rather than a cause.

As any other emotion, anger is thought to serve an adaptive function. Some scholars adhere to the idea that anger serves to consolidate social order (Matsumoto et al., 2010), whereas others believe the main function of anger is to prevent transgressions against oneself or to ward off subordination to another individual (Potegal & Stemmler, 2010b), or to initiate a response tendency towards aggressive behavior (Berkowitz, 2012; Fessler, 2010). To my opinion, the best and most extensive description of the functionality of anger comes from Novaco (2010):

> *"it energizes behavior as a high arousal state, increasing the amplitude of responding and serving to override inhibition; it focuses attention on situational elements having threat significance; it expresses or communicates negative sentiment, to convey displeasure and to prompt conflict resolution; it defends the self by social distancing and fear suppression, and it also defends self-worth by externalizing attributions of blame for misfortune; it potentiates a sense of personal control or empowerment among social groups as well as individuals; it instigates aggressive behavior due to its survival relevance, symbolic linkages, and learned connections; it signals information about personal state and situational significance, which is relevant to self-monitoring; and it dramatizes a social role enactment, in the sense of anger expression as dramaturgy played out in accord with social scripts or social rules"* (Novaco, 2010, p. 466).

So far, the image of anger arises as a natural response that serves some adaptive survival functions. In further support of the adaptive and functional nature of anger, Averill (1983) illustrates that beneficial consequences following angry episodes are more frequently reported than baleful consequences. Short-term positive and reinforcing effects of anger are: (amongst others) immediate compliance by others with the angry demands (van Doorn, Zeelenberg, & Breugelmans, 2014), facilitated interpersonal negotiations towards conflict resolution (van Kleef & Côté, 2007), gained respect, a sense of power and control over the situation, and energetic feelings caused by the release of adrenaline and

noradrenaline (Stosny, 1995)[5]. Relatedly, next to feeling angry, participants often report to feel more active, strong, determined, and energized (Harmon-Jones, Peterson, & Harmon-Jones, 2010; Tibubos & Schnell, 2013). Nevertheless, the disruptive effects of anger when experienced or expressed improperly are not to be mitigated. Anger is typically considered to be a negatively valenced emotion in taxonomies of affect that may seem pleasant in prospect, but often becomes unpleasant in hindsight (Fernandez, 2013). Its negative consequences both at the individual level and for society at large are well recognized throughout history in religion, folklores and mythology as well as well-documented in both classical and recent literature (Potegal & Novaco, 2010). Anger easily causes psychological or physical harm and subsequently impairs adaptive functioning in the contexts of family, work, and interpersonal relationships. For instance, anger can lead to interpersonal conflicts, retaliation from others, peer rejection and victimization, breakdowns in negotiations, impaired problem-solving and decision making, diminished work performance, and traffic violations and accidents. Especially, salient is its connection to aggression and violent behavior (DiGiuseppe & Tafrate, 2010; Novaco, 2011)[6]. In severe cases anger drives violent offending (e.g., child abuse, domestic violence, road rage, and rape) causing legal problems. In extreme cases anger can have fatal effects (i.e., murder, serial killings and warfare).

In short, anger as an emotional state can be considered to be both adaptive and maladaptive. However, as will become evident in the upcoming paragraph, anger no longer

[5] In angry clients these short-term reinforcing effects may fuel the angry fire and enhance resistance to acknowledge the detrimental effects their anger has for them in the long run.

[6] Although anger likely precedes aggressive and violent behavior, most anger episodes do not involve aggression. In fact, most anger episodes are solved in prosocial ways (Averill, 1983) and anger is more often shown by verbal and non-confrontational motor behavior (e.g., rolling the eyes) rather than aggressive acts (e.g., hitting and kicking). Hence, anger seems neither sufficient nor necessary for aggressive behavior to occur. Conversely, aggressive and violent acts can also occur without any anger involved (e.g., in some cases of instrumental aggression). Yet, on the other hand, it remains a challenging enterprise to demarcate anger from aggression (Kassinove & Tafrate, 2006). For instance, when do aversive verbalizations change from simple acts of expressing anger into verbal aggression? Given this complexity some suggest anger is only quantitatively different from aggression.

serves adaptive means if it has become a recurring, enduring and pervasive part of someone's personality.

1.2. Trait Anger and its clinical relevance

Unfortunately, for some people their anger can be so excessive in frequency, duration, and intensity that it distorts their daily functioning and becomes dysfunctional (Deffenbacher, Lynch, Oetting, & Yingling, 2001; Deffenbacher, Oetting, Thwaites, et al., 1996; Quinn, Rollock, & Vrana, 2014). For these people, anger has become entrenched in their personality, hence, some scholars refer to these individuals as high trait anger individuals (e.g., Spielberger, 1999; Tafrate et al., 2002). More specifically, trait anger refers to "the disposition to perceive a wide range of situations as annoying or frustrating and by the tendency to respond to such situations with elevations in state anger" (Spielberger, 1999, p. 1). As noted below, trait anger is associated with a variety of adverse outcomes. Anger prone persons are quick in sensing threat (Novaco, 2011) and much more likely to be perceived negatively in work and social relations given their belligerent nature and blaming tendency (Kassinove & Tafrate, 2002). Moreover, with respect to social and occupational functioning, trait anger is associated with marital problems (Baron et al., 2006) and inflicting harm upon coworkers (Douglas & Martinko, 2001). Trait anger also has a serious negative influence on general well-being (Mahon, Yarcheski, & Yarcheski, 2000). Relatedly, anger prone individuals experience severe personal distress (e.g., feeling guilty/ashamed or depressed afterwards), physical distress (e.g., headache, muscle tension), and are more likely to keep dwelling on their anger leading to even greater distress (Tafrate et al., 2002). On the road, trait anger is associated with risky driving behavior as well as verbal and physical aggression towards other drivers (Deffenbacher et al., 2001; Deffenbacher, 2013). Other problematic behavior more often seen in chronically angry persons includes: substance use (Leibsohn, Oetting, & Deffenbacher, 1994; Shopshire & Reilly, 2013; Spielberger, Foreyt, Goodrick, & Reheiser, 1995), pathological gambling (Korman et al., 2008), unhealthy eating (Anton & Miller, 2005; but also see Schneider, Appelhans, Whited, Oleski, & Pagoto, 2010), and suicidal behavior (Daniel, Goldston, Erkanli, Franklin, & Mayfield, 2009; Zhang et al., 2012). Somewhat similar to the longer-term effects of tobacco use, recurrent anger in angry individuals put them at much higher risk for medical problems, such as cardiovascular deceases (Smith, Glazer, Ruiz, & Gallo, 2004).

Trait anger is also highly problematic from a clinical psychology perspective. Clinicians are often faced with anger-prone patients (Cullari, 1994; Hovens, Lievaart, & Rodenburg, 2014; Spielberger, 1999). Moreover, high trait anger individuals are much more likely to present with comorbid psychopathology and have an increased likelihood of inadequate psychosocial functioning compared to low trait anger individuals (McDermut, Fuller, DiGiuseppe, Chelminski, & Zimmerman, 2009). Furthermore, trait anger is predictive of negative treatment outcomes (Erwin, Heimberg, Schneier, & Liebowitz, 2003; Rizvi, Vogt, & Resick, 2009). Most importantly, trait anger is associated with clinically significant anger-related problems (DiGiuseppe & Tafrate, 2004) and has been suggested to be a distinct clinical problem (McDermut et al., 2009). Finally, trait anger is key predictor of aggressive behavior both inside the laboratory (Bettencourt, Talley, Benjamin, & Valentine, 2006; Patton, Stanford, & Barratt, 1995) as well as outside the laboratory (Barbour, Eckhardt, Davison, & Kassinove, 1998; Deffenbacher et al., 2001; Douglas & Martinko, 2001; Nomellini & Katz, 1983). For example, high trait anger individuals report to act out with aggressive behavior at three times the rate of individuals low on trait anger (Kassinove & Tafrate, 2002).

In summary, high trait anger individuals seem to suffer a lot from a variety of adverse consequences if their anger remains untreated. As such, it is of critical importance to have a good understanding of the factors underlying trait anger[7]; not only from a clinical perspective, but also from a public-safety- and physical health perspective. Over the years, several studies have been conducted with this purpose in mind. In the following sections, some relevant models as well as the existing body of neurocognitive research on trait anger will be discussed[8]. Yet, before discussing these studies in more detail, important definitions

[7] The emphasis of the current thesis is on trait anger based on its multitude of negative outcomes, although we acknowledge that other clinically relevant anger constructs exist (e.g., Anger Expression-Out).

[8] Even though there are evidently numerous factors eventually determining individual differences in anger, including genetic (e.g., Sluyter et al., 2000), hormonal (e.g., Reuter, 2010), and environmental factors (e.g., Distel et al., 2012; Hoglund & Nicholas, 1995; Muris, Meesters, Morren, & Moorman, 2004), the current thesis will adopt a neurocognitive approach as this was the main focus of our research.

and distinctions in the literature regarding trait anger will be reviewed in order to bare ambiguity to a minimum.

1.3. Trait Anger and Related Concepts

The most evident and empirically validated distinction in the anger literature is between state and trait anger, whereby state anger refers to a psychobiological emotional state that varies in intensity ranging from mild annoyance to intense fury[9] (i.e., how someone may feel at a certain moment in time, having a discrete onset and offset and being situation specific), and trait anger refers to individual differences in the frequency, intensity, and longevity of state anger (i.e., how subjects feel in general, and where it is expected that high trait anger individuals show state anger more frequently and intensively (Fernandez, 2013; Spielberger, 1999). Other well-known distinctions with respect to anger refer to how people deal with their anger (Spielberger, 1999). Anger Expression-Out, for instance, refers to the extent to which individuals tend to express their anger outwardly (e.g., When angry, I slam doors). Anger Expression-In on the other hand refers to the extent to which individuals tend to suppress or hold their anger in when they are experiencing anger (e.g., When angry, I boil inside but don't show it). Note, that these tendencies are not necessarily opposite sides of the same coin, as frequent attempts to hold anger in may also lead to frequently expressing anger outwardly (DiGiuseppe & Tafrate, 2010). Finally, Anger Control-In describes the tendency to reduce arousal generated by anger (e.g., cooling down; when angry, I try to relax), whereas Anger Control-Out refers to the extent to which individuals attempt to

[9] A more extended and integrated definition of state anger comes from DiGiuseppe and Tafrate (2010) who defined anger as "a subjectively experienced emotional state with high sympathetic autonomic arousal. It is initially elicited by a perception of a threat (to one's well-being, property, present of future resources, self-image, social status or projected image to one's group, maintenance of social rules that regulate daily life, or comfort), although it may persist even after the threat has passed. Anger is associated with attributional, informational, and evaluative cognitions that emphasize the misdeeds of others and motivate a response of antagonism to thwart, drive off, retaliate against, or attack the source of the perceived threat. Anger is communicated through facial or postural gestures or vocal inflections, aversive verbalizations, and aggressive behavior. One's choice of strategies to communicate anger varies with social roles, learning history, and environmental contingencies" (p. 21).

prevent the outward expression of anger (e.g., I control my temper). Whilst high trait anger individuals are often more inclined to express their anger outwardly and less inclined to regulate their anger, other subtypes of angry individuals exist that do not experience anger frequently but may act extremely aggressively when they do (DiGiuseppe & Tafrate, 2004). As such, it is important to distinguish between the anger experience, and the expression and regulation of anger. Some consider trait anger in terms of low levels of agreeableness (e.g., Ahadi & Rothbart, 1994). Although trait anger and agreeableness are inversely related constructs (see chapter 2), agreeableness is a broader construct tapping a wider range of facets than trait anger. Other concepts related to anger, that should be avoided to be used interchangeably to describe anger, are irritability, (reactive) aggression, violence, hostility, and hate (see table 1 for the definitions of these constructs). Importantly, the distinctions between these constructs hold at both the state and the trait level. That is, even though trait irritability, trait aggression, and trait anger are closely related and highly correlated (Martin, Watson, & Wan, 2000)[10], I consider them to be separate constructs in the current thesis, as I think it is important to treat anger (emotional component) and aggression (behavioral component) as separate constructs where possible. Yet, given that the underlying cognitive patterns may at times parallel and similarities may be more apparent than the differences, we will also describe some cognitive models that focus more upon reactive aggression than trait anger.

[10] Spielberger (2010) suggested the anger, hostility, and aggression (AHA) syndrome based on the frequent co-occurrence of these concepts.

Table 1. Important definitions of constructs related to, but distinct from, anger.

Construct	Definition	Distinction
Aggression[11]	Refers to "overt motor behavior enacted with the intent to do harm or injury to a person or object, with the expectation that harm will occur" (DiGiuseppe & Tafrate, 2010, p. 23).	Anger refers to an internal feeling, whereas aggression refers to actual behavior. Although anger may increase the likelihood of aggression, other factors may intervene to disentangle this relationship (e.g., Denson, DeWall, & Finkel, 2012).
Violence	Refers to "Aggression that has extreme harm as its goal" (Anderson & Bushman, 2002, p. 29).	See aggression.
Hostility	"Refers to a set of negative attitudes that set the stage for anger and aggression. They represent predispositions about individuals (e.g., "You can't trust adolescents. They are all crazy). Such attitudes, or cognitive sets, increase the probability that neutral actions by the person or by members of these groups will be interpreted as wrong, unjust, purposeful, and preventable or that negative triggers will be seen to represent fundamental characteristics of the individual or group" (Kassinove & Tafrate, 2006, p. 6) .	Hostility refers more to a cognitive attitude than to an emotional felt state.
Irritability	Refers to "a physiological state characterized by a lowered threshold for responding with anger or aggression to stimuli. It is a partially aroused physiological state without the thoughts that usually occur with anger" (DiGiuseppe & Tafrate, 2010, p. 31)	Irritations seems to be a milder form of anger that appears not to be mediated by cognitions, but rather physiologically or neurologically mediated.
Hate	Refers to "an enduring negative affect of antagonism with a strong desire to effect revenge or hurt an opponent, without the physiological arousal normally associated with anger. Hate can turn to anger when the person actually confronts the objects of his or her hate" (DiGiuseppe & Tafrate, 2010, p. 31).	Whereas anger is typically described as a hot emotion, hate is rather experienced as a cold emotion (i.e. without warm bodily sensations).

[11] Note that in the literature there is a further distinction between reactive and proactive aggression (e.g., Buss, 1966; Hubbard, Romano, McAuliffe, & Morrow, 2010), whereby reactive aggression describes aggressive behavior in reaction to provocation and perceived threat, whereas instrumental aggression describes aggressive behavior aiding in goal pursuit beyond physical violence (e.g., robbery). Similar distinctions have found to be present in animal aggressive behavior (e.g., Lorenz, 1966). However, several scholars suggest that the proposed distinction is flawed as multiple motives (e.g., revenge and instrumental motives) may underlie aggression (DiGiuseppe & Tafrate, 2010). For instance, anger may precede deliberate, planned instrumental aggressive behavior. Moreover, reactive aggressive behavior may be rewarding (e.g., compliance with demands).

1.4. Cognitive models of (trait) anger and reactive aggression

Cognitive models of anger and reactive aggression have received widespread attention. Central to these models is the idea that differences in trait anger and reactive aggression can be explained through individual information processing differences. That is, it is assumed that whether or not people are inclined to react with anger or aggression in response to situations is (partly) mediated by cognitive processes, such as attention, cognitive control, and interpretation. Some influential models and theories in this field will now be described.

Based on work of Beck (2000) and Ellis (1977), cognitive-behavioral conceptual models of anger (e.g., Kassinove & Tafrate, 2002, 2011) have postulated that specific cognitions play a role in the experience of anger, such as hostile attributions and externalizing blame (misinterpreting neutral or ambiguous situations as hostile), horrifying and catastrophizing (i.e., exaggerating unpleasantness of a situation), low frustration tolerance (i.e., underestimating ability to deal with the situation), self-centered demandingness (i.e., elevating personal desires into dictates), and global ratings (i.e., overgeneralizing specific unwanted behavior of others as internal personality dispositions). Studies have indeed confirmed that high trait anger individuals tend to show these cognitive distortions more frequently (Martin & Vieaux, 2013; Tafrate et al., 2002). Moreover, treatments based on these models have shown to be effective (Beck & Fernandez, 1998; Deffenbacher, Dahlen, Lynch, Morris, & Gowensmith, 2000; Deffenbacher, 1999; Del Vecchio & O'Leary, 2004; DiGiuseppe & Tafrate, 2003). Whereas these earlier studies had the main focus on identifying the knowledge structures (e.g., beliefs and rules) that were assumed to underlie biased information-processing deficits in dysfunctional angry populations, current studies are more focused on examining the role of information processing mechanisms themselves as underlying aspects of dysfunctional anger.

Pointing out the mediating role information processing plays, appraisal theorists (Clore & Centerbar, 2004; Lazarus, 1991; Roseman, 2004; Smith & Kirby, 2004; Wranik & Scherer, 2010) argue that the way people evaluate occurring events influences which emotions will be elicited, whereby only certain appraisals lead to the generation of anger. Although there is no complete agreement on which kind of appraisals are necessary for anger to occur, most scholars agree that anger is typically caused when personally

significant goals are blocked by improper, blameful acts of someone external or something external in situations where the person feels powerful enough to deal with the provoking source successfully. Although these theories account for the occurrence of state anger rather than trait anger, we could imply from this literature that individuals who appraise more situations as frustrating are more likely to be high on trait anger. This is indeed what has been found in several studies (Hazebroek, Howells, & Day, 2001; Kuppens & Tuerlinckx, 2007; Kuppens, van Mechelen, Smits, De Boeck, & Ceulemans, 2007).

In a somewhat similar vein, Dodge and Crick's Social Information Processing (SIP) theory (Dodge & Crick, 1990) puts emphasis on how information is encoded and interpreted in explaining angry reactivity. In fact, it is considered to be one of the most influential models highlighting information processing as a key element in understanding angry reactivity. According to this model (see Figure 1), social cues have to be skillfully processed in order to react properly to the situation: (1) information has to be encoded, (2) information has to be interpreted, (3) interaction goals need to be clarified, (4) alternative strategies have to be hypothesized, (5) a response has to be selected from the hypothesized strategies, (6) and finally the selected response has to be carried out. Importantly, biases and deficiencies in processing any of these steps may, according to this model, increase the likelihood of aggressive behavior[12]. Based on this model, numerous empirical studies have been conducted that have confirmed the link between deviations in these processes on the one hand and aggressive behavior on the other hand, including the link between hostile attributions of intent and reactive aggression (for reviews, see Akhtar & Bradley, 1991; De Castro, Veerman, Koops, Bosch, & Monshouwer, 2002; Dodge, 2006). With respect to trait anger, Wilkowski and Robinson (2008b) propose that hostile interpretation biases at the first two stages of this model (encoding and interpreting information) can be seen as predisposing individuals towards increased anger and reactive aggression[13]. Indeed, several

[12] For instance, the failure to interpret a peer's intention as benign (i.e., making a hostile attribution), that is proposed to be partly dependent on encoding malicious information, is thought to increase the likelihood of retaliatory aggressive behavior. Additionally, aggressive children may come up with less effective solutions to problematic situations they encounter.
[13] Some may argue that the encoding of information has more to do with selective attention than with interpretation.

studies have established these information processing biases in high trait anger individuals specifically (Epps & Kendall, 1995; Hazebroek et al., 2001; Wingrove & Bond, 2005).

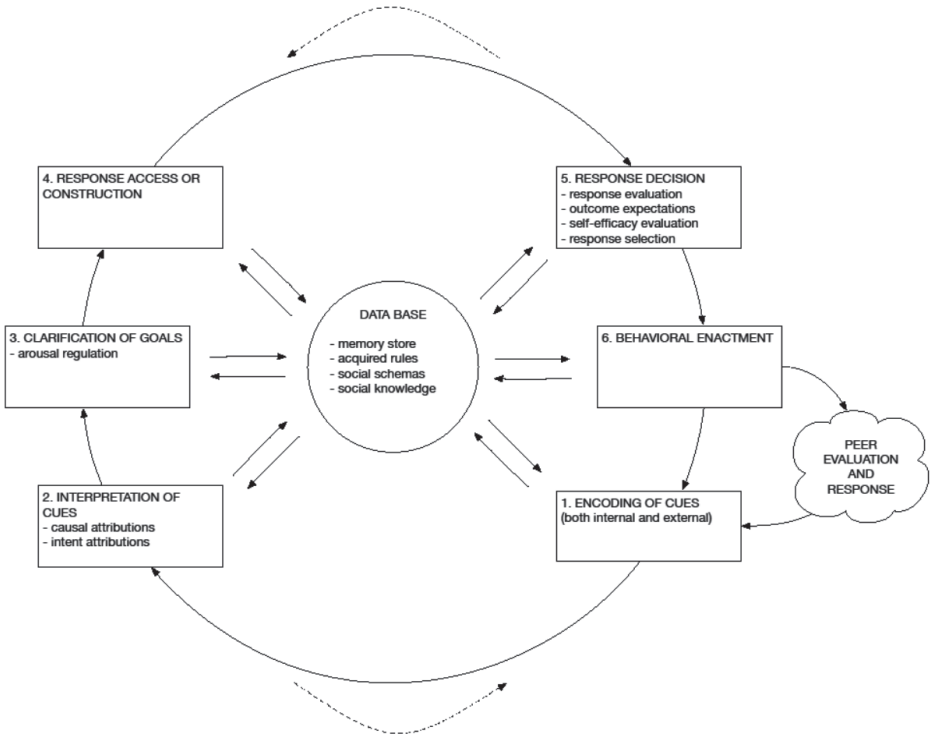


Figure 1. Dodge and Crick's Social Information Processing (SIP) theory (Dodge & Crick, 1990; Dodge, 2006).

Another highly influential model in the field of anger and reactive aggression is Berkowitz's Cognitive Neo-Associationistic theory (Berkowitz & Harmon-Jones, 2004; Berkowitz, 1990, 2010, 2012), which bears on the frustration-aggression hypothesis (Dollard, Miller, Doob, Mowrer, & Sears, 1939) that suggests that goal obstruction (c.f. frustration) leads to aggressive inclinations. Whereas most scholars generally agree that anger is an activator of aggression and view illegitimate frustrations and appraisals of blameworthiness as necessary generators of anger, Berkowitz's contrasting view is that aggressive behavior is produced by negative affect following aversive events rather than

anger per se[14] (see Figure 2). Importantly, Berkowitz puts emphasis on the fact that anger and aggressive actions can arise automatically even in the absence of (conscious or non-conscious) appraisals as a result of associative networks linking negative affects with anger-related feelings, thoughts, and aggressive behavior[15]. Hence, according to this model anger and aggressive inclinations result from (1) initial negative effect/ displeasure caused by decidedly unpleasant events and (2) from internal or external stimuli associated with negative effects (e.g., the mere presence of weapons or adopting an angry pose). The Neo-Associationistic model further implies that simultaneously other associations more closely related to freeze or flight behavior than to fight behavior may co-occur as a result of the negative affect as well (for instance, fear and avoidance behavior can become dominant of anger and aggressive behavior in situations where overwhelming danger is sensed). Another assumption from this model is that following the initial negative affect, the relative dominance of the eventual emotions and behavior eventually shown is dependent on a host of factors, including genetic, learned, and situational factors. As such, following the initial automatic associative processes, cognitive processes, including appraisals, may result into more fully developed emotional experiences. Finally, another important notion of this theory is that through spreading activation one anger component (e.g., hostile thoughts) will activate other anger components (e.g., physiological arousal) based on the strength of their associative connections. Following this theory, individual differences in trait anger may be explained by the functioning of associative networks, for example by chronically accessible hostile thoughts, stronger interconnections between hostile thoughts, and/or wider connections between hostile concepts and non-hostile concepts in high trait anger individuals (Wilkowski & Robinson, 2010). Yet, inconsistent results have been found with

[14] For instance, Berkowitz (2012) notes that "frustrations generate anger and aggressive inclinations only to the extent that they are decidedly unpleasant" (p.324). Anger in this models is viewed as occurring next to aggression.

[15] For example, anger and aggression elicited by priming effects and pain-induced aggression. However, there is still controversy about what actually is involved in the concept cognition. Berkowitz holds that if we adhere to the definition favored by Izard (1993) and Zajonc (1998, 2000), that is when "the term should have to do with the transformation of sensory input into a new mental construction", cognition is not always involved in eliciting anger and aggression (p.108).

respect to memory processes and the accessibility of hostile thoughts in anger disposed individuals (for reviews, see Owen, 2011; Wilkowski & Robinson, 2010). Another point of criticism is that the definition of the term unpleasant stimulus results in a tautology: one can only refer to a trigger as unpleasant based on the behavioral outcome.

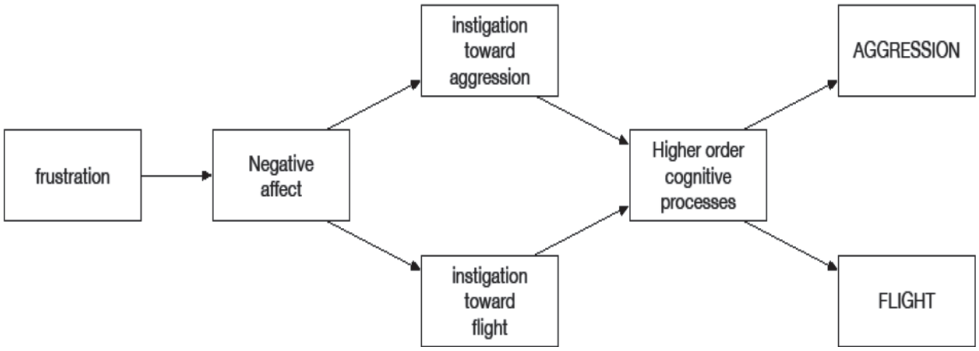


Figure 2. Berkowitz's Cognitive Neo-Associationistic theory (Berkowitz & Harmon-Jones, 2004; Berkowitz, 1990, 2010, 2012).

Two relevant cognitive theories with respect to aggression in general are the General Aggression Model (GAM; Anderson & Bushman, 2002; DeWall, Anderson, & Bushman, 2011) and the I^3 theory (Finkel, 2007). The General Aggression Model is a broad model that integrated several theories into a unifying framework in order to provide a general theory of human aggression (see Figure 3). In short, the GAM posits that internal states (cognition, arousal, and affect) mediate the effects of situational (e.g., provocation, pain, drugs) and personality variables (e.g., personality traits, sex, beliefs, values) on aggression. In line with the Neo-associationistic account of Berkowitz, GAM assumes that spreading activation processes may make hostile cognitions and feelings more accessible. Another important tenet of GAM is that appraisal and decision processes can be automatic or deliberate[16]. The causal role anger plays in aggressive behavior is also mentioned in the GAM. More specifically, GAM notes five reasons why anger can increase the probability of responding aggressively. According to the model, anger (1) activates high emotional arousal, (2) brings

[16] These automatic processes explain why angry clients may be unaware of their triggers causing the sensation of feeling taken by surprise and out of control.

aggression into the person's mindset, (3) provides a feeling of threat and retaliation, (4) keeps the person primed to aggression over time (e.g., increased attention to provoking cues), and (5) reduces inhibitions to aggression (e.g., anger provides a justification for aggressive behavior and interferes with higher-level cognitive processes usually recruited in moral reasoning and judgement). Interestingly, Digiuseppe and Tafrate (2010, p. 67) note that GAM can be seen as a general model of anger as well given that "most of the personal, situational, biological, and psychological factors that affect aggression also influence anger arousal".

The I^3 theory (Finkel, 2007) is a meta-theory which proposes that three interacting orthogonal processes lie at the heart of all behavior (in this case aggression): instigation, impellance, and inhibition (see Figure 4). Instigating factors include factors to which someone is exposed in particular contexts that normatively evoke a certain behavior. For example, provocation is thought to be an instigating factor which normatively evokes an aggressive response (Anderson & Bushman, 2002). Impelling factors include situational or stable factors that heighten the chance that individuals will enact certain behavior when encountering instigating factors in specific contexts. For instance, trait aggressiveness and provocation-focused rumination are thought to be impelling factors that increase the likelihood of aggressive behavior in response to provocation (Denson, DeWall, et al., 2012). Lastly, inhibiting factors encompass dispositional or situational factors that heighten the chance that individuals will override instigating and impelling forces, thereby overriding or reducing the intensity of the proclivity to enact the behavior. For instance, state and trait self-control can be taken as inhibiting factors reducing the likelihood or intensity of aggressive behavior (Denson, DeWall, et al., 2012; DeWall, Baumeister, Stillman, & Gailliot, 2007). In essence, aggression is thought to occur when instigating and impelling forces exceed inhibitory forces.

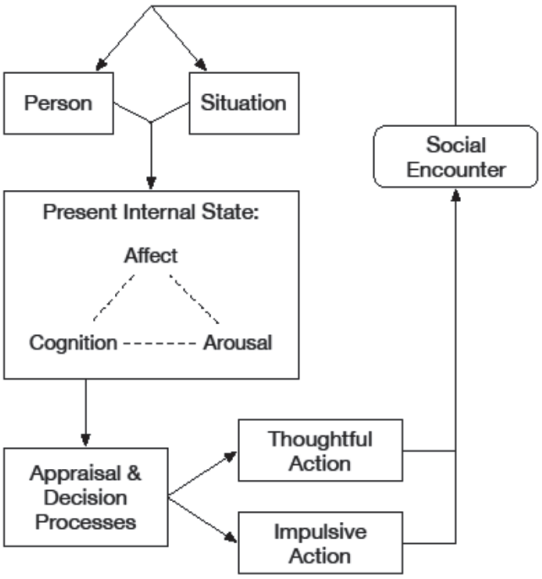


Figure 3. The General Aggression Model (Anderson & Bushman, 2002; DeWall, Anderson, & Bushman, 2011).

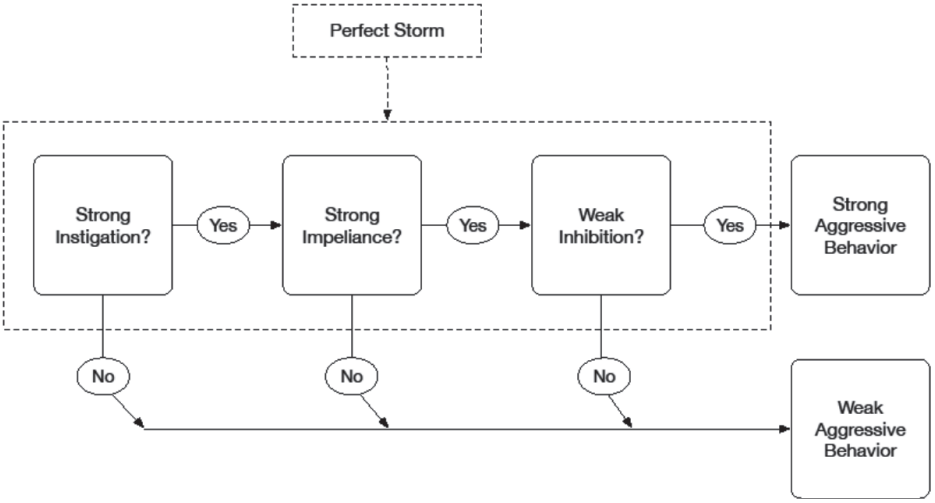


Figure 4. The I^3 theory (Finkel, 2007).

In both of the above mentioned meta-theories, trait anger can be seen as a personality variable that increases the chance of aggressive behavior. GAM further provides clues that individuals disposed towards anger and aggressive behavior are more likely to suffer from impaired cognitive processes, such as the tendency to interpret ambiguous situations as hostile as well as a tendency to focus attention more often towards hostile cues. In addition,

the I^3 model provides clues that anger disposed individuals might lack sufficient inhibitory model. Wilkowski and Robinson (2008b, 2010) who focus more specially on the cognitive underpinnings of trait anger in their integrative review, put emphasis on exact these three processes: automatic hostile interpretations, ruminative attention, and effortful control (cf. cognitive control). Given this model most exclusively focusses on trait anger, and hence forms the basis of the current thesis, it will be described in more detail in the following paragraph.

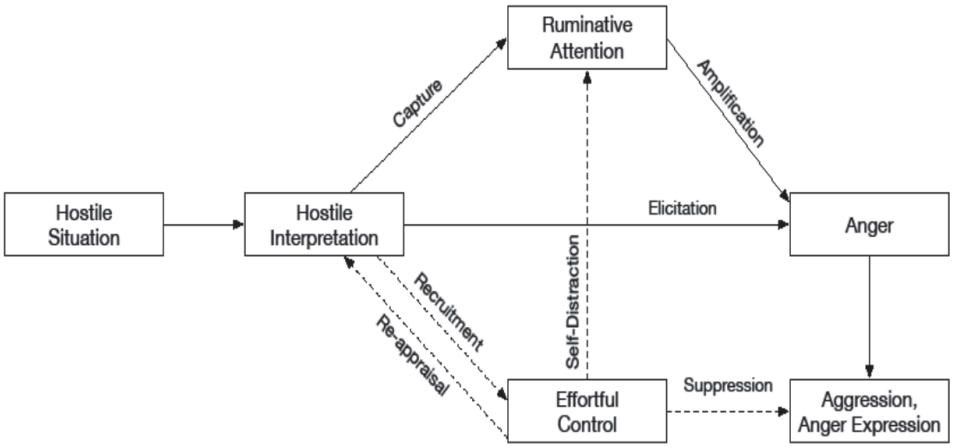


Figure 5. Wilkowski and Robinson's (Wilkowski & Robinson, 2008b, 2010) integrative cognitive model of trait anger and reactive aggression.

Cognitive processes in the model of Robinson and Wilkowski (2008b, 2010) are seen as mediating and moderating the effects of hostile situational influences onto subsequent outputs related to anger and aggression. More specifically, they postulate that anger disposed individuals are inclined to interpret ambiguous information as hostile, which is an automatic process that precedes attentional and effortful control processes and leads to the more frequent elicitation of anger[17]. Within this model, subsequent cognitive processes may mitigate or exacerbate inclinations towards anger following hostile interpretations (see Figure 5). For instance, when a situation is considered or interpreted to be hostile, according to this model, it will automatically capture attention which leads to angry rumination and in

[17] Alternatively, it could well be the case that selective attention precedes interpretation.

turn amplifies anger and the likelihood of enacting reactive aggressive behavior. High trait anger individuals are thought to have difficulties disengaging attention from the hostile information, explaining their more intense and prolonged anger episodes. Indeed, several studies have shown that high trait anger individuals have difficulties in disengaging from hostile stimuli (Eckhardt & Cohen, 1997; Honk et al., 2001; Putman, Hermans, & van Honk, 2004; Smith & Waterman, 2004), although it is unclear to what extent these results are due to state anger (Owen, 2011). Perhaps state anger influences information processing, whereby most reliable effects occur in high trait anger individuals who are experiencing state anger (Schultz, Grodack, & Izard, 2010). Moreover, experimental studies have shown that angry rumination maintains or intensifies state angry feelings (Bushman, 2002; Denson, Moulds, & Grisham, 2012; Ray, Wilhelm, & Gross, 2008; Rusting & Nolen-Hoeksema, 1998), whereas distraction results in decreases of state anger (Konecni, 1974; Mischkowski, Kross, & Bushman, 2012). Finally, effortful control processes may interfere with these automatic inclinations towards anger and aggression. Importantly, high-trait-anger individuals are proposed to self-regulate their automatic inclinations towards anger and aggression less effectively. That is, whereas individuals low on trait anger are proposed to successfully recruit effortful control resources in potentially hostile situations, high trait anger individuals are thought to allow these resources to remain dormant. More specifically, effortful control processes may mitigate angry and aggressive inclinations in the following ways in this model: (1) by reinterpreting hostile interpretations in favor of non-hostile interpretation, (2) by disengaging attention from hostile information, and (3) by suppressing angry and aggressive behavioral tendencies. With regard to effortful control, abundant cognitive behavioral experiments has shown that cognitive control deficits become most apparent under conditions where anger is primed (Wilkowski, Robinson, & Troop-Gordon, 2010; Wilkowski & Robinson, 2007, 2008a; Wilkowski, 2011). Further confirming the causal link between cognitive control and reactive aggression, a different line of research has demonstrated that reductions in state self-control following 'ego depletion' manipulations[18] result in less anger control following provocation (DeWall et al., 2007; Finkel & Campbell,

[18] Ego depletion refers to a state of temporary weakened self-control as a result of previous acts of exercising self-control (e.g., attempts to quit smoking) (Baumeister et al., 2007).

2001; Finkel, DeWall, Slotter, Oaten, & Foshee, 2009; Stucke & Baumeister, 2006), whereas boosts in self-control lead to decreases in reactive aggressive behaviours (Denson, 2015; Wilkowski, Crowe, & Ferguson, 2015). Yet, there is a need to better understand the neurocognitive mechanisms associated with trait anger. For instance, only a few electroencephalographic (EEG) studies have been conducted in high trait anger individuals (Jaworska et al., 2012; Liu et al., 2014), while electrophysiological measures can provide more objective and sensitive assessments of cognitive processes involved (Fabiani, Gratton, & Coles, 2000).

To recapitulate, all of the theoretical models described above postulate that neurocognitive processes play a role in explaining individual differences in anger and reactive aggression, which is soundly supported by empirical studies. Moreover, most of the theories point out that these processes interact with each other as well as with other situational and dispositional factors. Finally, most of the above theories highlight the distinction between initial automatic processes and later more controlled processes that play a role in trait anger and reactive aggression.

1.5. A neurocognitive perspective on Trait Anger

By including EEG measurements or neuroimaging during the performance of cognitive tasks (e.g., Go/NoGo task) that require the recruitment of specific cognitive functions (e.g., response inhibition and error-monitoring), more insight can be gained about the timing and the neural substrates of the cognitive processes involved in trait anger. Importantly, cognitive control encompasses several separate but interacting neurocognitive subcomponents (Luna, Garver, Urban, Lazar, & Sweeney, 2004), and only a few of these subcomponents have been given sufficient attention with regard to trait anger. For instance, EEG studies focusing on error-processing deficits in relation to trait anger were at the start of the current PhD Project non-existent, although neural error-processing deficits were to be expected in high trait anger individuals (Robinson, Wilkowski, Meier, Moeller, & Fetterman, 2012). Similarly, little was known about response inhibition on the neurophysiological level, especially under circumstances where anger is primed, even though previous studies suggest impulse control in hostility-primed contexts may be impaired in high trait anger individuals (e.g., Wilkowski, 2011). The results of the EEG studies that were conducted had produced mixed results. That is, whereas one study found support

for reduced attentional control and diminished response inhibition in HTA individuals on a continuous performance task (Jaworska et al., 2012), another study could not find support for reduced inhibitory control on a Go/NoGo task in HTA individuals (Liu et al., 2014). Given that both studies used a neutral task and it is suggested that especially in hostile contexts HTA individuals allow cognitive control processes to lay dormant (Wilkowski & Robinson, 2008b, 2010), inhibitory control deficits at the neural level may have remained undetected. One goal of this thesis was therefore to investigate inhibitory control on a neurophysiological level in a context where anger was primed. Additionally, we examined error-processing on the neurophysiological level in relation to trait, as there were indications for distorted error-processing at the behavioral level (Robinson et al., 2012). Greater insight into the dysfunction of neural networks associated with cognitive control with regard to trait anger is of essential importance, as it could provide valuable information about the aetiology and maintenance of this harmful personality trait. For instance, error-processing and response inhibition deficits may explain the existence of inappropriate (aggressive) behaviour in high trait anger individuals despite its negative consequences.

1.6. Causal links between cognitive processes and anger: The impact of angry rumination and mental fatigue on anger and self-control

To date, most studies on cognitive risk factors for trait anger have investigated such factors in isolation. However, it seems unlikely that cognitive risk factors would operate in isolation. Instead, cognitive factors probably influence each other or interact in such a way that the impact of one factor is influenced by the other. In line with this idea, researchers in the field of anxiety and depression proposed the "combined cognitive bias hypothesis" (Everaert, Koster, & Derakshan, 2012; Hirsch, Clark, & Mathews, 2006), and indeed found evidence to support the idea that cognitive risk factors interact (e.g., Everaert, Tierens, Uzieblo, & Koster, 2013). A similar approach may be highly informative for understanding cognitive risk factors for a disposition towards anger. Several questions seem of particular importance in this respect: (a) To what extent are cognitive risk factors for trait anger interrelated ("association questions")?, (b) To what extent do cognitive risk factors for trait anger causally influence each other ("causal questions')?, and (c) To what extent do cognitive risk factors for trait anger jointly predict someone's disposition towards anger ("predictive magnitude questions")? Answering these questions will help to gain a broader

understanding of the interrelations and interactions among risk factors for trait anger, and may inform us on which risk factors are essential to target during treatment.

The integrative cognitive model of trait anger and reactive aggression (Wilkowski & Robinson, 2008b, 2010) provides an important first step in informing about the possible interrelations and interactions of the involved cognitive processes. Clear hypotheses based on the model can be made. Yet, some of the relationships specified in this model have not been thoroughly tested and new relationships between these cognitive processes may be established. The second aim of the present thesis is therefore to gain more insight in the causal links between the cognitive processes and anger as well as their impact on each other using experimental designs. For instance, whereas the model of Wilkowski and Robinson (2008b, 2010) predicts that cognitive control has an impact on ruminative attention, recent research suggests that the relationship between cognitive control and ruminative attention may also be the other way around. More specifically, according to the multiple systems models of angry rumination (Denson, 2013), angry rumination should result in reductions in cognitive control leading to subsequent aggressive behaviour (Denson, 2009, 2013). However, to our knowledge direct investigations of the impact of angry rumination on cognitive control are scarce (Denson, Pedersen, Friese, Hahm, & Roberts, 2011). As information about the impact of angry rumination on cognitive control may advance our understanding of rumination-induced aggression, we sought to investigate this relation in this thesis.

Interestingly, the hypothesis put forward in the multiple systems model of angry rumination, i.e. that angry rumination should result in reduced self-control, is heavily reliant on resource depletion accounts (DeWall et al., 2007; Finkel & Campbell, 2001; Stucke & Baumeister, 2006), which are in turn based on the Strength Model of Self-Control (Baumeister, Vohs, & Tice, 2007). More specifically, the multiple systems model of angry rumination predicts that angry rumination may result in reduced self-control, as a result of ego depletion following attempts to (a) reduce anger intensity, (b) inhibit angry thoughts, and (c) to inhibit aggressive inclinations, subsequently heightening the likelihood of aggressive behaviour. Yet, in the literature much debate exists, however, as to whether ego depletion (cf. resource depletion) is a real phenomenon warranting explanation (Carter, Kofler, Forster, & McCullough, 2015) as well as about what processes underlie the effects observed in ego depletion experiments. For instance, it is not clear to what extent

motivation and mental fatigue may moderate the effects observed (Hagger, Wood, Stiff, & Chatzisarantis, 2010). An important question therefore is whether effects of ego depletion manipulations on aggressive behavior are caused specifically by engaging in acts of self-control or may also be caused by a more general state of mental fatigue. Another goal of this thesis was to examine this question by investigating the impact of a mentally fatiguing task, unrelated to ego depletion, on experienced anger and anger control following provocation by the experimenter.

1.7. Overview of the current dissertation

The general aim of the current thesis was to examine (neuro)cognitive factors that contribute to the aetiology and maintenance of a disposition towards anger. To this end, correlational and experimental designs in both clinical and nonclinical samples were used. Five studies, described in more detail in the upcoming chapters (chapter 2 to 6), are included in this thesis. Below we provide a short overview.

One of the first challenges we encountered during our research was that the Dutch version of the most often used instrument for measuring trait anger, the State-Trait Anger Expression Inventory-2 (STAXI-2)(Spielberger, 1999), was lacking. Therefore, we translated and backtranslated the STAXI-2 from English to Dutch and subjected it to a thorough psychometric evaluation, as described in **Chapter Two**.

In **Chapters Three and Four**, the neural substrate of response inhibition and error-processing in relation to trait anger was investigated in order to build upon the integrative cognitive model of trait anger and reactive aggression. Importantly, based on the assumption that HTA individuals allow cognitive control processes to lay dormant under potentially hostile situations (Wilkowski & Robinson, 2008b, 2010), we employed an affective Go/NoGo task in order to examine whether response inhibition deficits would be more pronounced if anger was primed. In both studies, response inhibition was measured by behavioural performance (accuracy) as well as Event-Related Potentials (ERPs; N2 and P3). Numerous event-related potential studies have shown increased N2 and P3 amplitudes on NoGo trials compared to Go trials and these ERPs are generally assumed to reflect

inhibitory processing (Falkenstein, Hoormann, & Hohnsbein, 1999; Luijten et al., 2014)[19]. Similarly, in both studies ERPs were used to assess error-processing. More specifically, error-processing was indexed by the Error Related Negativity and the Positivity error (Bernstein, Scheffers, & Coles, 1995; Overbeek, Nieuwenhuis, & Ridderinkhof, 2005; Yeung, Botvinick, & Cohen, 2004)[20].

In the study described in **Chapter Three,** we compared anger-primed inhibitory control and error-processing between high trait anger (HTA) and low trait anger (LTA) individuals using an "extreme groups" design. Here, we expected reduced inhibitory control in HTA compared to LTA as evidenced by reduced accuracy and N2 and P3 amplitudes on NoGo trials, especially for anger-related pictures (Wilkowski & Robinson, 2008b, 2010). With respect to error-processing, we expected to find either reduced ERN or Pe amplitudes in HTA compared to LTA (Robinson et al., 2012). In **Chapter Four,** we examined whether scores on trait anger in forensic psychiatric patients were associated with individual differences in anger-primed inhibitory control and error-processing using a correlational design. Here, we expected that higher scores on trait anger would be associated with more commission errors (i.e., lower accuracy) and reduced N2 and P3 amplitudes on NoGo trials. Furthermore, we expected the inverse relation between trait anger and inhibitory control to be more pronounced for anger-related pictures in support of the integrative cognitive model of trait anger and reactive aggression (Wilkowski & Robinson, 2008b, 2010). Finally, we expected higher scores on trait anger to be accompanied by reduced ERN and Pe amplitudes (Robinson et al., 2012).

---

[19] The N2 signal is a negative-going stimulus locked component that arises shortly after stimulus presentation (200-350ms) and is proposed to measure response conflict (Nieuwenhuis et al., 2003) or the signaling of deviant stimulus features (Fox et al., 2000). The P3 signal is a positive-going stimulus-locked component that arises after stimulus presentation (roughly 250 to 500 ms) and is proposed to reflect the actual inhibitory process with respect to the Go/NoGo task (Kok et al., 2004; Verleger et al., 2006).

[20] The ERN arises shortly after making error in reaction time tasks, such as the Go/NoGo task, and is thought to reflect action monitoring either through conflict monitoring (Yeung et al., 2004) or through automatic error detection (Bernstein et al., 1995). The Pe follows the ERN and is assumed to reflect the more conscious processing of an error (Luijten et al., 2014; Overbeek et al., 2005).

In **Chapter Five** we studied the impact of angry rumination, relative to distraction, on experienced anger and inhibitory control as measured with an affective Go/NoGo task. According to the multiple systems models of angry rumination (Denson, 2013), angry rumination results in reduced effortful control as a result of ego depletion. Hence, based on this model, we expected increased anger and reduced anger-primed inhibitory control (as evidenced by more commission errors on NoGo trials) following angry rumination as compared to distraction. If reduced effortful control is demonstrated following angry rumination, this would suggest that the relation put forward in the integrative cognitive model of trait anger and reactive aggression between rumination and effortful control is reciprocal rather than unidirectional.

In **Chapter Six** we examined the impact of a mentally fatiguing task (that should be unrelated to ego depletion) on experienced anger and anger control (cf. refusal to cooperate) following provocation by the experimenter. We expected that mentally fatigued participants would experience more intense anger following provocation compared to non-fatigued individuals. Moreover, we expected that mentally fatigued individuals would more likely refuse to cooperate with the experimenter than the non-fatigued individuals. These hypothesis are in line with the integrative cognitive model of trait anger and reactive aggression, in which a causal relation between effortful control and anger expression is suggested.

Finally, in **Chapter Seven**, the main findings of this dissertation are summarized and discussed.

Preparatory Work

Validation of the Dutch Version of the STAXI-2

Chapter Two

Anger Assessment in Clinical and Nonclinical Populations: Further Validation of the State–Trait Anger Expression Inventory-2

This chapter has been published as:
Lievaart, M., Franken, I.H.A., & Hovens, J.E. (2016). Anger Assessment in Clinical and Nonclinical Populations: Further Validation of the State–Trait Anger Expression Inventory-2. *Journal of Clinical Psychology, 72 (3)*, 263–278. DOI: 10.1002/jclp.22253

Abstract

Objective: The most commonly used instrument for measuring anger is the State-Trait Anger Expression Inventory-2 (STAXI-2; Spielberger, 1999). This study further examines the validity of the STAXI-2 and compares anger scores between several clinical and nonclinical samples.

Method: Reliability, concurrent, and construct validity were investigated in Dutch undergraduate students (N = 764), a general population sample (N = 1211), and psychiatric outpatients (N = 226).

Results: The results support the reliability and validity of the STAXI-2. Concurrent validity was strong, with meaningful correlations between the STAXI-2 scales and anger-related constructs in both clinical and nonclinical samples. Importantly, patients showed higher experience and expression of anger than the general population sample. Additionally, forensic outpatients with addiction problems reported higher Anger Expression-Out than general psychiatric outpatients.

Conclusion: Our conclusion is that the STAXI-2 is a suitable instrument to measure both the experience and the expression of anger in both general and clinical populations.

2.1. Introduction

Spielberger (1988) pointed out that anger can be both understood as a momentarily state, i.e., how subjects feel at the moment, and as a trait, i.e., how subjects feel in general, and where it is expected that subjects with high trait anger show state anger more frequently and intensely. This distinction between state and trait anger, sometimes referred to as the State–Trait Anger Theory, has been repeatedly empirically validated (Deffenbacher, Oetting, Lynch, & Morris, 1996; Quinn et al., 2014). Moreover, Spielberger (1988) recognized the importance of how these angry feelings are expressed and controlled. For example, when angered, people may express their anger outwardly by physical or verbal assault or inwardly by suppressing anger. Knowing how people deal with their anger is important for understanding both the nature of anger and client problems and concerns (Deffenbacher, Oetting, Lynch, et al., 1996).

Importantly, anger is a clinically relevant emotion. Elevated levels of anger occur in a range of psychiatric disorders (Novaco, 2010) and a high number of anger-disordered clients present for treatment in private clinical settings (Lachmund, DiGiuseppe, & Fuller, 2005). More specifically, several disorders, including bipolar disorder, oppositional defiant disorder, generalized anxiety disorder, posttraumatic stress disorder (PTSD), borderline, antisocial personality disorder, and paranoid personality disorder, mention anger or irritation as a possible symptom or criterion in their diagnostic criteria (DiGiuseppe & Tafrate, 2010). Notably, elevated anger is also present in people with neurological impairments (DiGiuseppe & Tafrate, 2010) and intellectual disabilities (Willner, Jahoda, & Larkin, 2013) and in patients with other psychiatric disorders, such as depression (Koh, Kim, & Park, 2002), substance dependence (Shopshire & Reilly, 2013; Walfish, Massey, & Krone, 1990), and psychotic disorders (Nederlof, Muris, & Hovens, 2011; Novaco & Whittington, 2013).

At least a third to approximately half of the psychiatric outpatients show considerable feelings of anger (McDermut et al., 2009; Posternak & Zimmerman, 2002), and these feeling are associated with several adverse treatment outcomes. In individuals with social anxiety disorder, anger is associated with less satisfaction about their cognitive behavioral treatment and with premature termination from treatment (Erwin et al., 2003). Similarly, patients meeting criteria for PTSD with high levels of anger before treatment seem to benefit less from exposure therapy (Rizvi et al., 2009). Negative treatment effects due to

anger are not limited to anxiety and stress disorders and apply to other disorders as well (also see, Fassino, Abbate-Daga, Pierò, Leombruni, & Rovera, 2003; Fassino, Amianto, Abbate, & Leombruni, 2007; Mammen, Shear, Greeno, Wheeler, & Hughes, 1997; Rüsch et al., 2008). Finally, anger presumably interferes with common therapy factors, such as a strong therapeutic alliance (DiGiuseppe & Tafrate, 2010), motivation for treatment, resistance to change, and less collaboration on goal setting (Hubble, Duncan, & Miller, 2004). Therefore, it may be valuable to enquire about anger difficulties in patients, and this requires validated anger measures.

The most commonly used instrument for measuring anger is Spielberger's State–Trait Anger Expression Inventory second edition (STAXI-2; 1999). The STAXI-2 has proven to be a reliable and valid instrument measuring the experience and management of anger (Eckhardt, Norlander, & Deffenbacher, 2004). Currently, the Dutch version of the STAXI-2 has not been subjected to a thorough psychometric evaluation. Moreover, despite its widespread use, research about differences between several groups of clinical populations and nonclinical populations in the experience and management of anger using the STAXI-2 has received little attention. One study of Cullari (1994) showed that psychiatric outpatients scored higher on the State and Trait anger scales compared with normal subjects, but it did not include the expression scales. Another study, by Etzler, Rohrmann, and Brandt (2014), showed higher scores on Anger Expression-In for inmates compared with the mean of the German standardization sample. Yet here it remains unclear how these inmates would score compared with other clinical samples. Finally, De-Moja and Spielberger (1997) showed higher scores on State Anger, Trait Anger, and ´ Anger Out and lower scores on Anger Control for heroin-dependent drug users compared with a nondrug-using matched control group using the STAXI. Although there are several studies showing higher experience and expression of anger for clinical patients compared with healthy nonclinical subjects (Barbour et al., 1998; Cullari, 1994; De-Moja & Spielberger, 1997; Etzler et al., 2014), most of them used the STAXI instead of the STAXI-2. Because the STAXI-2 is more detailed in measuring the control of anger, using the STAXI-2 might provide extra information about the differences in the control over anger between clinical populations and nonclinical populations.

Moreover, to our knowledge, no studies have yet been conducted in which the experience and management of anger of several patient groups are directly compared with

each other, which could provide important information about the usefulness of the STAXI-2 in clinical settings. It would, for example, be interesting to compare general psychiatric patients with forensic patients regarding their experience and management of anger because we would expect forensic patients to deal with their anger in a less constructive way.

The present study was designed to further validate the STAXI-2. First, we provide initial evidence regarding the reliability and validity of the Dutch Translation of the STAXI-2. Second, we present information about the convergent and divergent validity in both nonclinical and clinical samples, by studying the associations with anger-related symptomatology and the personality traits of the big five. Finally, we provide information about the differences between several clinical patient samples and nonclinical populations regarding anger experience and anger management strategies.

We expected the STAXI-2 to be reliable (regarding both internal consistency and test-retest reliability). In addition, we expected the trait scales to be somewhat more stable than the State Anger Scale. Positive relationships were expected among the STAXI-2 experience and anger expression scales with alternative measures of anger and anger-related symptomatology (i.e., hostility, impulsiveness, and aggression), whereas negative associations were expected for the anger control scales.

Additionally, we expected the STAXI-2 scales to be associated with three of the big five personality traits: Neuroticism, Conscientiousness, and Agreeableness. More specifically, we expected positive correlations between the Trait Anger Scale and the anger expression scales with Neuroticism, and negative correlations with Agreeableness and Conscientiousness (Kuppens, 2005; Martin et al., 1999; Pease & Lewis, 2015; Sanz, García-Vera, & Magán, 2010; Whiteman, Bedford, Grant, Fowkes, & Deary, 2001). Because the links between the Trait Anger and anger expression scales on the one hand and Extraversion and Openness on the other hand are less well established, no specific hypotheses were formulated for these scales and the analyses can be regarded as exploratory. Importantly, we expected clinical populations to experience and express anger more frequently and to control anger less frequently than the general population sample. Finally, we expected forensic patients to experience and express anger more frequently compared with general psychiatric patients.

2.2. Method

2.2.1. *Ethics Statement*

This study was conducted according to the rules of the Helsinki Declaration on informed consent and confidentiality (World Medical Association, 2001). Approval for this study was obtained from the Ethical Committee of the department of psychology of the Erasmus University Rotterdam. For the psychiatric patients, we obtained approval from the Ethical Committee of Delta Psychiatric Hospital. Participants were informed about the study, after which they gave informed consent. Participation was based on a voluntary basis and participants were free to refrain from participation at any point in time. Information about the research was provided on the questionnaires or via a recruitment letter.

2.2.2. *Participants*

The data reported here were collected among both nonclinical and clinical samples. The nonclinical samples comprised samples from both undergraduate students (*N* = 764) and the general population (*N* = 1211). The group of students comprised psychology students at the Erasmus University Rotterdam (*N* = 564) and the Amsterdam University of Applied Sciences (*N* = 200), who took part in the annual screening for research purposes, for which they were rewarded with study credits. Of the students, 25% were men and 75% were women. Mean age was 20.4 years (standard deviation [*SD*] = 2.9), with an age range from 18 to 46 years. The general population sample was a random representative sample drawn from the general population and the gender distribution was 49% men and 51% women. Mean age was 32.2 years (*SD* = 9.4), with an age range from 18 to 50 years. Both the students and the people from the general population completed the questionnaires online using Qualtrics Survey Software (Qualtrics Labs, Provo, Utah). Test-retest reliability was estimated in a subgroup of psychology students (*N* = 217) and the general population (*N* = 99).

The clinical samples were derived from several psychiatric treatment facilities in the Netherlands. The total group of patients comprised psychiatric outpatients from a general psychiatric hospital (*N* = 80), outpatients with addiction problems (*N* = 88) from an addiction clinic, and forensic outpatients with addiction problems (*N* = 58) from the forensic department of the same addiction clinic. Of the total patient group, 63% were men and 37%

were women. Their mean age was 42.9 years (*SD* = 11.6), with an age range from 19 to 79 years.

Patients from the general psychiatric hospital were mostly diagnosed with major depression (*n* = 34) or bipolar disorder (*n* = 11), but other diagnoses were present as well, such as anxiety disorders (*n* = 2), alcohol dependency (*n* = 4), schizophrenia (*n* = 2), borderline (*n* = 3), and/or PTSD (*n* = 2). These diagnoses were based on clinical records. Gender distribution in this outpatient sample was 34.6% men. Mean age was 44.45 years (*SD* = 12.06), with an age range from 19 to 68 years.

The outpatients with addiction problems were mostly polydrug users primarily diagnosed with a substance use disorder, ranging from alcohol, amphetamine, cannabis, cocaine, and heroin use disorders. Some of the patients with addiction problems had a gambling disorder. Comorbid psychopathology was not an exclusion criterion and was not recorded. Typically, this population has high levels of comorbidity, including major depression, PTSD, attention deficit hyperactivity disorder, borderline personality disorder, and/or antisocial personality disorder. The outpatient sample with addiction problems mostly comprised male participants (68.2%). Mean age was 43.65 years (*SD* = 11.71) with an age range of 23 to 79 years.

The forensic outpatients with addiction problems were also mostly polydrug users and additionally convicted for violent and/or nonviolent offenses, such as theft, fraud, drug and alcohol-related crimes, assault and battery, domestic violence, and/or robbery. The forensic outpatient sample with addiction problems mainly comprised male participants (93.1%). Mean age was 39.79 years (*SD* = 10.39), with an age range from 22 to 61 years. All patients were individually tested in the presence of the research assistant and completed the questionnaires as paper-and-pencil tests.

2.2.3. Materials

STAXI-2 (Spielberger, 1999). The STAXI-2 measures the experience of anger, the tendency to express anger, and the tendency to control anger. Two bilingual translators independently translated all 57 items of the STAXI-2 from English into Dutch. Inconsistencies were discussed with the authors (ML and JEH) until consensus was reached and finally this version was back translated to English. Spielberger authorized this translation. The STAXI-2 is scored

on a 4-point Likert scale and comprises 57 items and six scales: State Anger, Trait Anger, Anger Expression-In, Anger Expression-Out, Anger Control-In, and Anger Control-Out.

The 15-item State Anger (S-Ang; range 15–60) scale assesses three distinctive components of the intensity of anger as an emotional state: Feeling Angry (S-Ang/F), Feel Like Expressing Anger Verbally (S-Ang/V), and Feel like Expressing Anger Physically (S-Ang/P). Each subscale comprises five items.

The 10-item Trait Anger scale (T-Ang; range 10–40) assesses two distinctive components of the general tendency to experience more frequent, more intense, and longer episodes of anger: Angry Temperament (T-Ang/T), measuring the disposition to experience anger without provocation, and Angry Reaction (T-Ang/R), measuring the frequency that angry feelings are experienced in situations that involve frustration and/ or negative evaluations. Each subscale comprises four items.

Anger Expression-Out (AX-O) measures the outward expression of anger toward other people or objects either verbally or physically, while Anger Expression-In (AX-I) measures how often the participant experiences an angry feeling and suppresses it or holds it in instead of expressing it. Anger Control-Out (AC-O) measures how often a person controls the outward expression of angry feelings, while Anger Control-In (AC-I) measures how often a person controls his or her angry feeling by calming down or cooling off. All four expression scales comprise eight items.

The Dutch version of the Aggression Questionnaire (AVL; Buss & Perry, 1992; Meesters, Muris, Bosma, Schouten, & Beuving, 1996). The AVL measures self-reported aggression. The AVL comprises 29 items that are scored on a 5-point Likert scale ranging from 1 (entirely disagree) to 5 (entirely agree) (range = 29–145). The AVL comprises four subscales, i.e., Physical Aggression, Verbal Aggression, Anger, and Hostility, which have been confirmed by factor analyses (Meesters et al., 1996). Moreover, test-retest reliability for all four subscales is between .76 and .79 with a 6-week interval (Meesters et al., 1996). Cronbach's alpha coefficients in the current samples indicate acceptable to good reliability according to Kline (2005), with values ranging from .63 to .85.

The Dutch Version of the Barratt Impulsiveness Scale 11 (BIS11; Lijffijt & Barratt, 2005; Patton et al., 1995). The BIS11 measures trait impulsivity. The BIS11 comprises three subscales: Attentional Impulsiveness, Motor Impulsiveness, and Non-planning Impulsiveness. The BIS11 comprises 30 items and is scored on a 4-point Likert scale ranging

from 1 (rarely/never) to 4 (almost always/always) (range 30–120). The original BIS11 shows good psychometric properties: the three-factor structure has consistently been replicated, the questionnaire is associated with other self-report measures of impulsivity, and test-retest reliability for the total score is ρ = .83 (Patton et al., 1995; Stanford et al., 2009). Cronbach's alpha coefficients in the current samples indicate acceptable to good reliability, with values ranging from .65 to .80.

The Symptom Checklist (SCL-90; Arrindell & Ettema, 1986). The SCL-90 comprises 90 items and measures nine primary distress dimensions. For this study, we used only the Hostility scale, which comprises six items. Cronbach's alpha in our patient sample was .81, indicating good reliability according to Kline (2005). The SCL-90 is scored on a 5-point Likert scale ranging from 1 (not at all) to 5 (extremely). Test-retest reliabilities range from r = .68 to .85 for the SCL-90 subscales. Additionally, psychiatric patients score higher on the SCL-90 than people from the general population. Finally, construct validity was supported by factor analyses (Arrindell & Ettema, 1986; Evers, Vliet-Mulder, & Groot, 2000).

The NEO Five Factor Inventory (NEO-FFI; Costa & McCrae, 1992; Hoekstra, Ormel, & De Fruyt, 1996). The NEO-FFI measures the five major dimensions of personality: Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness. All subscales include 12 items measured on a 5-point Likert scale ranging from 1 (entirely disagree) to 5 (entirely agree). The NEO-FFI shows good psychometric properties (Hoekstra et al., 1996). Cronbach's alpha coefficients in our patient sample indicated good reliability for Neuroticism, Agreeableness, and Conscientiousness, with values above .76. The Extraversion and Openness scales had lower reliability with Cronbach's alpha coefficients of .59 for Openness and .63 for Extraversion.

The NEO-FFI-3 that was administered in the general population sample is similar to the NEOFFI. The NEO-FFI-3 comprises 60 items as well and measures the same big five personality constructs. However, 15 items have been rewritten or replaced to improve readability and the psychometric properties (Hoekstra & De Fruyt, 2014). For the English version, Cronbach's alpha coefficients ranged from .72 to .88 in adolescent and adult samples (McCrae & Costa, 2007). Moreover, the original five-factor structure for the NEO-FFI-R was retained. Cronbach's alpha coefficients in the population sample of the current study indicate good reliability, with all values above .70.

2.2.4. *Procedure*

Each group completed the STAXI-2 along with several additional questionnaires. Students at the Erasmus University additionally completed the Aggression Questionnaire and a part of these students also completed the Barratt Impulsiveness Scale. The psychiatric outpatients completed the AVL, the SCL-90, the BIS, and the NEO-FFI in addition to the STAXI-2. People from the general population completed only the NEO-FFI-3 in addition to the STAXI-2.

2.3. Results

2.3.1. *Reliability, Stability, and Factor Structure of the Dutch Version of the STAXI-2*

Internal consistencies (Cronbach's alpha) were satisfactory for all scales and subscales of the Dutch version of the STAXI-2 in the student, general population, and psychiatric outpatient samples (see Table 1).

The test-retest stability was computed in a subsample of students (retest after 7 to 44 days; mean [*M*] = 23 days, *SD* = 6 days) and in a selection of the population responders (retest after 21 to 28 days; *M* = 24 days, *SD* = one day). All STAXI-2 scales showed a relative high stability, except for the State Anger Scale, as was expected (see Table 2).

There was a high degree of (sub)scale intercorrelation that was generally in the expected direction for the general population sample (see Table 3). According to our expectation, the correlations between S-Ang with S-Ang/F, S-Ang/V, and S-Ang/P were high, with *r*s > .90. The intercorrelations between the three subscales were also high (all *r*s > .80). Although the intercorrelations are very high, it might still be clinically relevant to differentiate between the three subscales. T-Ang was highly correlated with both its subscales as well (both *r*s > .80). The intercorrelation among T-Ang/T and T-Ang/R was much lower, with *r* = .55, indicating that both subscales measure different aspects of T-Ang. AX-O showed a substantial correlation with T-Ang, T-Ang/T, and T-Ang/R, which suggests that Anger Expression-Out is more related to Trait Anger than initially expected. As expected, both AC-O and AC-I correlated negatively with T-Ang, T-Ang/T, T-Ang/R, and AX-O, and positively with AX-I, supporting the construct validity. However, AC-O and AC-I were also highly positively correlated with each other, *r* = .77, suggesting that both scales have much in common.

Table 1. Internal consistencies using Cronbach's alpha among students, psychiatric patients, and a sample of the general population.

	Students (*N* = 764)	**Psychiatric patients (*N* = 226)**	**General population (*N* = 1211)**
S-Ang	.91	.96	.96
S-Ang/F	.81	.93	.90
S-Ang/V	.84	.93	.91
S-Ang/P	.79	.93	.91
T-Ang	.83	.92	.88
T-Ang/T	.83	.92	.88
T-Ang/R	.73	.83	.77
AX-O	.77	.84	.80
AX-I	.71	.72	.71
AC-O	.84	.83	.81
AC-I	.90	.82	.89

Note: S-Ang = State Anger; *S-Ang/F* = Feeling Angry; *S-Ang/V* = Feel Like Expressing Anger verbally; *S-Ang/P* = Feel like Expressing Anger Physically; *T-Ang* = Trait Anger; *T-Ang/T* = Angry Temperament; *T-Ang/R* = Angry Reaction; *AX-O* = Anger Expression-Out; *AX-I* = Anger Expression-In; *AC-O* = Anger Control-Out; *AC-I* = Anger Control-In.

Table 2. Consistency of the STAXI-2 over time in students and a subsample of the general population.

	Students (*N* = 217)	**Population (*N* = 99)**
S-Ang	.32	.59
S-Ang/F	.22	.41
S-Ang/V	.34	.55
S-Ang/P	.37	.53
T-Ang	.78	.82
T-Ang/T	.72	.81
T-Ang/R	.65	.76
AX-O	.76	.76
AX-I	.66	.70
AC-O	.70	.73
AC-I	.71	.66

Note: S-Ang = State Anger; *S-Ang/F* = Feeling Angry; *S-Ang/V* = Feel Like Expressing Anger verbally; *S-Ang/P* = Feel like Expressing Anger Physically; *T-Ang* = Trait Anger; *T-Ang/T* = Angry Temperament; *T-Ang/R* = Angry Reaction; *AX-O* = Anger Expression-Out; *AX-I* = Anger Expression-In; *AC-O* = Anger Control-Out; *AC-I* = Anger Control-In.

Table 3. Correlations between *T-Ang*, *AX-O*, *AX-I*, *AC-O*, and *AC-I* in the population sample (*N* = 1211).

	T-Ang	***T-Ang*/T**	***T-Ang*/R**	***AX-O***	***AX-I***	***AC-O***	***AC-I***
T-Ang							
T-Ang/T	.85						
T-Ang/R	.87	.55					
AX-O	.58	.53	.43				
AX-I	.28	.17	.32	.31			
AC-O	-.26	-.28	-.14	-.31	.32		
AC-I	-.21	-.23	-.11	-.25	.27	.77	

Note: All correlations: $p < .001$; *T-Ang* = Trait Anger; *T-Ang*/*T* = Angry Temperament; *T-Ang*/*R* = Angry Reaction; *AX-O* = Anger Expression-Out; *AX-I* = Anger Expression-In; *AC-O* = Anger Control-Out; *AC-I* = Anger Control-In.

Because Spielberger (1999) viewed the STAXI-2 as three questionnaires, i.e., a state (15 items), a trait (10 items), and an expression control questionnaire (32 items), we tried to replicate his findings in our general population sample with separate exploratory principal factor analyses on these scales using obligue rotations (PROMAX). We used factors with an initial eigenvalue >1.00 and Cattell's' scree plot inflexion point to determine the number of factors to retain. In these analyses, items with salient factor loadings equal to or greater than .40 are reported. In general, the construct validity of the Dutch STAXI-2 was supported by a factor structure close to Spielberger's (1999) original conceptualization (see Tables 4 and 5). The most striking difference between our results and those reported by Spielberger is that our factor analysis extracted one general anger control scale instead of two distinct anger control scales. As we considered it to be clinically and theoretically relevant to differentiate between the proposed expression and control scales of the STAXI-2, and for international comparisons, we adhered to Spielberger's original conceptualization in our subsequent analyses.

Table 4. Principal factor analysis with promax rotation on *T-Ang* in the general population sample (*N* = 1211).

	Factor 1	**Factor 2**
T-Ang/T		
Am quick tempered	.79	
Have a fiery temper	.84	
Am a hotheaded person	.90	
Fly off the handle	.66	
T-Ang/R		
Get angry when slowed down by others' mistakes		.52
Feel annoyed when not given recognition for doing good work		.66
Furious when criticized in front of others		.63
Feel infuriated when do a good job and get poor evaluation		.81
Say nasty things when mad		.44
% Variance	44.43	7.67
Eigenvalue	4.44	.77

Table 5. Principal factor analysis with promax rotation on *AX-O*, *AX-I*, *AC-O*, and *AC-I* in the general population sample (*N* = 1211).

	Factor 1	Factor 2	Factor 3
AX-O			
Express anger			.72
Is someone is annoying, apt to tell him or her			.64
Lose temper			
Make sarcastic remarks to others		.40	
Do things like slam doors			
Argue with others			.49
Strike out at whatever is infuriating		.40	.42
Say nasty things			.45
AX-I			
Keep things in			
Pout or sulk			.48
Withdraw from people		.43	
Boil inside, but don't show it		.49	
Tend to harbor grudges that don't tell anyone about		.54	
Am secretly quite critical of others		.49	
Am angrier than willing to admit		.53	
Irritated a great deal more than people are aware of		.60	
AC-O			
Control temper	.50		
Am patient with others	.50		
Control urge to express angry feelings		.41	
Keep cool	.65		
Control behavior	.67		
Can stop from losing temper			
Try to be tolerant and understanding	.62		
Control my angry feelings	.60		
AC-I			
Take a deep breath and relax	.73		
Try to calm down as soon as possible	.80		
Try to simmer down	.85		
Try to soothe angry feelings	.77		
Endeavor to become calm again	.82		
Reduce anger as soon as possible	.65		
Do something relaxing to calm down	.67		
Try to relax	.84		
% Variance	24.63	11.33	3.74
Eigenvalue	7.88	3.62	1.20

2.3.2. Correlates of Anger in Nonclinical Samples and a Psychiatric Outpatient Sample

Looking at Table 6, we find that the STAXI-2 demonstrates strong convergent and divergent validity, with statistically significant correlations in the expected direction for the normal samples as well as the total psychiatric outpatient sample. In general, the anger experience and anger expression scales were positively associated with self-reported anger, aggression, and hostility. Moreover, in general, both anger control scales were negatively related to self-reported anger, aggression, and hostility. Importantly, the strongest correlations were found among the STAXI-2 subscales and the Anger Scale of the AVL. Moreover, the Anger Expression-In subscale was not associated with Verbal and Physical Aggression, whereas the Anger Expression-Out scale was moderately to strongly positively associated with Verbal and Physical Aggression, supporting the concurrent validity.

The correlations with the STAXI-2 subscales and the BIS11 scales were also as expected (see Table 6). Attentional Impulsiveness was weakly to moderately positively correlated with anger experience and expression in both students and psychiatric outpatients. Moreover, Attentional Impulsiveness was weakly to moderately negatively correlated with both anger control scales in students. In psychiatric patients, we found only a moderate negative correlation for AC-I with Attentional Impulsiveness. Motor Impulsiveness did not correlate with the STAXI-2 scales in students, whereas in psychiatric patients Motor Impulsiveness was weakly positively correlated with T-Ang and AX-O, and moderately, negatively correlated with AC-O. Nonplanning Impulsiveness had weak to moderate negative correlations, with both anger control scales in both students and psychiatric outpatients. Further, Nonplanning Impulsiveness was also weakly, positively associated with anger experience, and AX-O in psychiatric patients. Finally, Nonplanning Impulsiveness was positively related only to T-Ang in students.

Last, the STAXI-2 scales showed meaningful correlations in the expected directions with the big five personality constructs of the NEO-FFI. The correlations are presented in Table 7. As expected, positive correlations were found between T-Ang and Neuroticism and negative correlations were found among T-Ang with Agreeableness and Conscientiousness. AX-O was negatively associated with Agreeableness and Conscientiousness as well, but it was positively associated only with Neuroticism in the general population sample. Further, AX-I was positively correlated with Neuroticism and negatively correlated with Conscientiousness and Extraversion. Moreover, both anger control scales correlated

positively with Agreeableness and Conscientiousness in both samples, whereas anger control was positively associated only with Neuroticism, Extraversion, and Openness in the general population sample. S-Ang correlated positively with Neuroticism and negatively with Extraversion, Openness, Agreeableness, and Conscientiousness in the general population sample. The association between S-Ang with Openness and Conscientiousness, however, was not found in the patient sample.

Table 6. Correlations of the STAXI-2 scales with the Aggression Questionnaire, the Hostility Scale of the SCL-90, and the Barratt Impulsiveness Scale.

	S-Ang		***T-Ang***		***AX-O***		***AX-I***		***AC-O***		**AC-I**	
	Students	Patients	Students	Patients	Students	Patients	Students	Patients	Students	Patients	Students	Patients
N[1]	555	213	555	211	555	208	555	211	555	208	555	211
AQ-Phys	.26***	.28***	.49***	.44***	.45***	.52***	.06	.10	-.34***	-.24***	-.29***	-.12
AQ-verb	.20***	.24***	.46***	.48***	.45***	.54***	-.01	.04	-.31***	-.25***	-.25***	-.18**
AQ-Ang	.31***	.30***	.69***	.60***	.62***	.62***	.13**	.19**	-.54***	-.42***	-.38***	-.27**
AQ-Hos	.33***	.33***	.49***	.51***	.38***	.42***	.40***	.38***	-.23***	-.16*	-.19***	-.13
N		205		207		205		208		208		206
SCL-90-HOS		.51***		.65***		.55***		.22***		-.39***		-.36***
N	125	217	125	215	125	212	125	215	125	215	125	212
BIS-AI	.17*	.27***	.55***	.35***	.46***	.26***	.21*	.27***	-.35***	-.21**	-.38***	-.13
BIS-MI	-.01	.11	.06	.29***	.02	.22***	-.06	.10	.06	-.19**	-.02	-.02
BIS-NonP	-.02	.17**	.18*	.30***	.15	.18**	-.11	.10	-.24**	-.27***	-.31***	-.18**

Notes: *** p <.001; ** p < .01; * p < .05; AQ = Aggression Questionnaire; AQ-Phy =AQ Physical Aggression; AQ-Verb = AQ verbal Aggression; AQ-Ang = AQ Anger Subscale; AQ-Hos = AQ Hostility Subscale; SCL-90-Hos = Symptom Checklist Hostility Subscale; BIS-AI = Barratt Impulsiveness Scale -Attentional Impulsivity; BIS-MI = Barratt Impulsiveness Scale -Motor Impulsivity; BIS-NonP = Barratt Impulsiveness Scale -Non-Planning Impulsivity.

[1]Due to missing values not all *N* are the same.

Table 7. Correlations of the NEO-FFI with the STAXI-2 in the populations and in psychiatric outpatients.

NEO-FFI		*S-Ang*	*T-Ang*	*AX-O*	*AX-I*	*AC-O*	*AC-I*
Neuroticism	Population[1]	.30***	.36***	.27***	.39***	-.19***	-.15***
	Patients[2]	.19**	.31***	.10	.42***	-.08	-.14
Extraversion	Population	-.17***	-.13***	-.06	-.23***	.13***	.18***
	Patients	-.19**	-.11	.08	-.32***	-.01	.05
Openness	Population	-.10***	-.04	-.05	.07*	.17***	.19***
	Patients	-.08	.02	.06	.02	.05	.10
Agreeableness	Population	-.29***	-.36***	-.43***	-.09***	.32***	.34***
	Patients	-.36***	-.47***	-.44***	-.12	.29***	.23***
Conscientiousness	Population	-.27***	-.22***	-.26***	-.18***	.25***	.27***
	Patients	-.09	-.25**	-.19**	-.22***	.21**	.15*

Notes: *** p <.001; ** p < .01; * p < .05; State Anger; *T-Ang* = Trait Anger; *AX-O* = Anger Expression-Out; *AX-I* = Anger Expression-In; *AC-O* = Anger Control-Out; *AC-I* = Anger Control-In.

[1] *N* = 1211.

[2] *N* ranges, due to missing values, from 199-205.

2.3.3. *Differences in Experience and Management of Anger Between Clinical and Nonclinical Samples*

In general, we expected psychiatric outpatients to experience and express anger more frequently and be inclined to control anger less frequently than the respondents from the general population. Therefore, we compared all the mean scale scores of the psychiatric outpatients and the respondents from the population using one-way analyses of variance (ANOVAs). As expected the psychiatric patients experienced and expressed more anger than the population respondents (see Table 8). More specifically, the patients reported to experience anger more intensely and more frequently compared with the population sample. Moreover, they reported to be more inclined to express their anger both outwardly toward individuals or objects through physically or verbally aggressive behavior and inwardly by suppressing anger or holding it in. Contrary to our expectation, we found no group differences on the anger control scales between patients and the general population.

Table 8. Anger, anger expression, and anger control in respondents from the population and psychiatric outpatients.

	Population sample (*N* = 1211)		**Psychiatric sample (*N* = 224)**		***F* (Patients versus Population)**
	M	***SD***	***M***	***SD***	
S-Ang	18.72	7.08	19.99	8.35	7.36**
S-Ang/F	6.48	2.50	7.09	3.11	14.20***
S-Ang/V	6.23	2.61	6.56	3.04	4.15*
S-Ang/P	6.01	2.41	6.34	2.88	3.31
T-Ang	16.37	5.03	18.39	6.21	28.53***
T-Ang/T	5.63	2.18	6.52	2.61	29.61***
T-Ang/R	7.61	2.44	8.17	2.82	11.25***
AX-O	14.76	3.90	15.47	4.83	4.63*
AX-I	17.48	3.89	18.93	4.39	28.04***
AC-O	20.96	4.62	20.67	5.11	.81
AC-I	21.23	5.03	20.88	5.44	1.11

Note: *** $p < .001$; ** $p < .01$; * $p < .05$; *S-Ang* = State Anger; *S-Ang/F* = Feeling Angry; *S-Ang/V* = Feel Like Expressing Anger verbally; *S-Ang/P* = Feel like Expressing Anger Physically; *T-Ang* = Trait Anger; *T-Ang/T* = Angry Temperament; *T-Ang/R* = Angry Reaction; *AX-O* = Anger Expression-Out; *AX-I* = Anger Expression-In; *AC-O* = Anger Control-Out; *AC-I* = Anger Control-In.

In order to investigate differences in the experience and management of anger among the patient groups, we also compared all the mean scale scores of the STAXI-2 using one-way ANOVAs with Patient Group (general psychiatric outpatients, outpatients with addiction problems, forensic outpatients with addiction problems) as the between-subjects factor. Results revealed no differences between the patient groups regarding State Anger, Trait Anger, and Anger Control. Interestingly, we did find main effects of Patient Group on Anger Expression-Out and Anger Expression-In (see Table 9). Post hoc tests using Bonferroni correction revealed that the forensic outpatients with addiction problems, as expected, were more inclined to express their anger outwardly compared with the two other groups (both *p*s < .05). Moreover, patients from the general psychiatric hospital reported higher Anger Expression-In compared with the outpatients with addiction problems, whereas the general psychiatric outpatients did not differ significantly from the forensic outpatients.

Table 9. Anger experience and anger management per patient group.

	General Psychiatric Outpatients (*N* = 80)		**Addicted Outpatients (*N* = 88)**		**Forensic Addicted Outpatients (*N* = 58)**		***F* (Patients versus Population)**
	M	***SD***	***M***	***SD***	***M***	***SD***	
S-Ang	21.30	.96	18.47	.91	21.26	1.15	2.876
S-Ang/F	7.68	.91	6.65	.34	7.22	.43	2.182
S-Ang/V	7.04	.35	5.99	.33	7.02	.42	2.976
S-Ang/P	6.58	.33	5.84	.32	7.02	.42	2.982
T-Ang	19.06	.70	17.52	.67	19.24	.84	1.781
T-Ang/T	6.60	.30	6.33	.28	6.89	.36	.760
T-Ang/R	8.73	.32	7.76	.30	8.22	.38	2.384
AX-O	15.14	.54	14.72	.52	17.33	.65	5.362**
AX-I	20.06	.49	17.95	.47	19.20	.59	4.872**
AC-O	20.49	.59	20.89	.56	20.70	.71	.887
AC-I	21.09	.61	20.52	.58	20.74	.72	.790

Note: *** *p* <.001; ** *p* < .01; * *p* < .05; *S-Ang* = State Anger; *S-Ang/F* = Feeling Angry; *S-Ang/V* = Feel Like Expressing Anger verbally; *S-Ang/P* = Feel like Expressing Anger Physically; *T-Ang* = Trait Anger; *T-Ang/T* = Angry Temperament; *T-Ang/R* = Angry Reaction; *AX-O* = Anger Expression-Out; *AX-I* = Anger Expression-In; *AC-O* = Anger Control-Out; *AC-I* = Anger Control-In.

2.4. Discussion

The main goal of this study was to further validate the STAXI-2 by (a) providing initial information about the reliability and validity of the Dutch version of the STAXI-2, (b) providing additional support for the concurrent validity of the STAXI-2 in general by investigating convergent and divergent validity in both normal and clinical samples, and (c) informing about differences between several clinical patient samples and a nonclinical population sample regarding anger experience and anger management strategies. On the whole, our results indicate adequate psychometric properties of the Dutch version of the STAXI-2.

To begin with, we expected this STAXI-2 to be reliable in terms of both internal consistency and test-retest reliability. In addition, we expected the trait scales to be somewhat more stable than the State Anger Scale, given that state anger is defined as being a transient emotional state, whereas the trait scales are conceptualized as stable personality traits (Spielberger, 1999). As predicted, the internal consistency of the STAXI-2 was adequate, with alpha coefficients for the STAXI-2 scales all above .70. Moreover, the test-retest reliabilities were fairly stable and similar to previous studies using a translation of the STAXI-2 (e.g., Rohrmann et al., 2013). Importantly, lower test-retest stabilities were found for State Anger compared with the trait scales, supporting the State–Trait Anger Theory (Deffenbacher, Oetting, Thwaites, et al., 1996).

Second, the construct validity of the Dutch STAXI-2 was supported by a factor structure close to Spielberger's (1999) original conceptualization. Whereas we found partial evidence for the construct validity of the anger expression and anger control scales, we found sufficient support for the construct validity of the State Anger and Trait Anger scales. The most striking difference between our results and those reported by Spielberger is that our factor analysis extracted one general anger control scale instead of two distinct anger control scales. One possible explanation could be that the cultural difference between Europe and the United States account for these results, as a one-factor solution for both control scales was also found for the German version of the STAXI-2 (Rohrmann et al., 2013). Alternatively, conceptual nuances might as well have been lost in the translation process, despite our fairly stringent translation process. In future research consideration should be given to scoring the anger control items as either one factor or two separate factors.

Although there were some minor differences between our results and Spielbergers data, we still consider it to be clinically and theoretically relevant to differentiate between the proposed expression and control scales of the STAXI-2. For example, clients high on Anger Control-Out frequently monitor their own anger and work to prevent angry expression, which might take a lot of energy and can result in passivity and withdrawal in situations where expressing anger assertively might be better (Kassinove & Tafrate, 2002). On the contrary, clients high on Anger Control-In work to calm down and reduce experienced anger, which might be more adaptive. Our finding that Anger Control-Out was associated with physical aggression and motor impulsiveness, whereas Anger Control-In was not in our total patient sample may be taken as further proof of the potential importance of differentiating between Anger Control-In and Anger Control-Out. In short, these results altogether show that the Dutch version of the STAXI-2 demonstrates good validity.

Importantly, in support of the validity of the STAXI-2 in general, we found support for the concurrent validity in both nonclinical and clinical samples. First, positive correlations were found for the anger experience and anger expression scales with the AVL Anger subscale and with measures of anger-related symptomatology (i.e., hostility, [attentional] impulsiveness, and aggression) in both samples. Interestingly, the Anger Expression-In subscale was not associated with Verbal and Physical Aggression, whereas the Anger Expression-Out scale was moderately to strongly positively associated with Verbal and Physical Aggression. Furthermore, as expected, negative associations were found for the anger control scales with anger and anger-related symptomatology. Finally, in line with the State–Trait Anger theory (Deffenbacher, Oetting, Thwaites, et al., 1996), trait anger was more strongly related to anger constructs than to other constructs.

Moreover, our results were in line with previous research showing positive correlations between Trait Anger and the anger expression scales with Neuroticism (Sanz et al., 2010; Whiteman et al., 2001), and inverse relationships with Agreeableness and Conscientiousness (Kuppens, 2005; R. Martin et al., 1999; Pease & Lewis, 2015; Sanz et al., 2010; Whiteman et al., 2001). Additionally, the anger control scales were positively related to Agreeableness and Conscientiousness in both samples. Finally, we also found small negative associations between Extraversion and the Trait Anger and anger expression scales on the one hand and small positive associations between Extraversion and the anger control scales on the other hand in the population sample. As Extraversion is positively related to

positive affect (Costa & McCrae, 1992) and social commitment, a negative correlation between Extraversion and anger is not unexpected. The finding that these relations were significant in the population sample but not in the patient sample may be explained by differences in the sample size, because the effect sizes found in the population were small. Importantly, the results of our study suggest that the convergent and divergent validity is supported in both nonclinical as well as clinical samples.

Finally, our study provides information about differences between clinical and nonclinical samples regarding the experience and management of anger. First, our study replicates previous studies showing that patients tend to experience anger more intensely and more frequently than people from the general population and are more inclined to express their anger both outwardly by means of physically and verbally aggressive behavior and inwardly by suppressing anger or holding anger in (Barbour et al., 1998; Cullari, 1994; De-Moja & Spielberger, 1997; Etzler et al., 2014; Spielberger, 1999). Notably, no differences were found between patients and the general population sample regarding the control of anger. This finding is in line with the study of Etzler et al. (2014) who have found no differences in anger control between prison inmates and the German standardization sample, but it is in contrast with the results from Spielberger (1999), who found less anger control both inwardly and outwardly for patients compared with normal subjects. Perhaps the patients in our samples try to control anger equally often but are less effective in doing so, resulting in a higher tendency to experience and express anger.

Further, our study showed that forensic outpatients with addiction problems tend to express their anger more outwardly, whereas they do not seem to differ regarding the frequency and intensity of their anger experience compared with general psychiatric patients. These results add further support to the predictive validity of the STAXI-2 because we would expect forensic patients to deal with their anger in a less constructive way. Further, these results imply that forensic patients might benefit from targeted treatments in which they learn how to deal with their anger in more adaptive, non-expressive ways because the way patients deal with their anger seems more important than the frequency at which it occurs. Last, our study showed that the general psychiatric patients were somewhat more inclined to suppress their anger or hold it in compared with the outpatients with addiction problems. This result implies that clinicians should be extra aware of hidden anger in their psychiatric outpatients in order to prevent negative treatment effects.

2.5. Limitations

Although our study benefitted from the use of a large, nationally, representative sample as well as a large psychiatric outpatient sample, specific limitations and recommendations for future work should be noted. First, to better assess the construct validity of the scales and subscales of the STAXI-2, future studies might benefit from including a sample (with subgroups) of patients that primary sought treatment because of anger-related problems. Perhaps, the subscales and scales can be better differentiated from each other when using a diverse sample of angry clients. For example, while some clients might express their anger mostly verbally, other clients might express their anger mostly physically, leading to a better discrimination between the Anger State subscales. Moreover, while people from the general population might control their anger both outwardly and inwardly, leading to one factor, angry clients might use one control strategy more over the other, leading to a better discrimination of the two anger control scales. Second, prospective designs demonstrating that the STAXI-2 predicts the frequency, intensity, and duration of anger and observed aggressive behavior and other negative consequences would further add to the validity of this measure.

2.6. Conclusion

Mindful of these limitations, we can conclude from our study that the STAXI-2, including the Dutch version of the STAXI-2, provides a reliable, valid measure of the experience, expression, and control of anger, making it a valuable assessment tool for clinicians to conduct a proper anger evaluation. Moreover, this study adds further support to the usefulness of the STAXI-2 because we found meaningful differences between clinical and nonclinical samples regarding their anger experience and management.

Part I

Cognitive Control in Relation to Trait Anger

Chapter Three

Trait Anger in Relation to Neural and Behavioral Correlates of Response Inhibition and Error-Processing

This chapter has been published as:
Lievaart, M., Veen, F.M. van der, Huijding, J., Naeije, L, Hovens, J.E. & Franken, I.H.A. (2016). Trait Anger in Relation to Neural and Behavioral Correlates of Response Inhibition and Error-processing. *International Journal of Psychophysiology, 99*, 40-47. DOI: 10.1016/j.ijpsycho.2015.12.001

Abstract

Effortful control is considered to be an important factor in explaining individual differences in trait anger. In the current study, we sought to investigate the relation between anger-primed effortful control (i.e., inhibitory control and error-processing) and trait anger using an affective Go/NoGo task. Individuals low (LTA; n = 45) and high (HTA; n = 49) on trait anger were selected for this study. Behavioral performance (accuracy) and Event-Related Potentials (ERPs; i.e., N2, P3, ERN, Pe) were compared between both groups. Contrary to our predictions, we found no group differences regarding inhibitory control. That is, HTA and LTA individuals made comparable numbers of commission errors on NoGo trials and no significant differences were found on the N2 and P3 amplitudes. With respect to error-processing, we found reduced Pe amplitudes following errors in HTA individuals as compared to LTA individuals, whereas the ERN amplitudes were comparable for both groups. These results indicate that high trait anger individuals show deficits in later stages of error-processing, which may explain the continuation of impulsive behavior in HTA individuals despite its negative consequences.

3.1. Introduction

Anger is a universal, and in general an adaptive emotion that people experience regularly (Averill, 1983; Kassinove et al., 1997). Some individuals, however, experience anger with such an excessive frequency and intensity that their anger starts to interfere with daily life and becomes dysfunctional. For instance, high trait anger (HTA) is associated with a diversity of adverse outcomes, including aggressive behaviour (Bettencourt et al., 2006; Tafrate et al., 2002), domestic violence (Barbour et al., 1998), poor psychosocial functioning (McDermut et al., 2009), health problems (T. Smith et al., 2004), and interpersonal problems (Baron et al., 2006; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). Given the dysfunctional nature of HTA, several studies have been conducted to gain a deeper understanding of the factors underlying this harmful personality trait (for reviews, see Owen, 2011; Wilkowski & Robinson, 2008b, 2010).

3.1.1. Trait anger and effortful control

Studies on individual differences in trait anger have identified reduced effortful control as one of the key cognitive factors involved (Bresin & Robinson, 2013; Denson, 2015; Wilkowski & Robinson, 2008b, 2010). Effortful control (cf. cognitive control; Luna et al., 2004) refers to "the efficiency of executive attention — including the ability to inhibit a dominant response and/or to activate a subdominant response, to plan, and to detect errors" (Rothbart & Bates, 2006, p. 129). Effortful control is proposed to mitigate anger through several means. For example, it allows individuals to reinterpret hostile interpretations in favor of non-hostile interpretations, to disengage from hostile information, and to suppress angry expressions and aggressive behaviour tendencies (Wilkowski & Robinson, 2010). According to the theory of Wilkowski and Robinson (2010) individuals low in trait anger (LTA) recruit effortful control resources in potential hostile situations, whereas individuals high in trait anger allow these resources to lay dormant. Importantly, effortful control is underlain by several separate but interacting subcomponents, including (but not restricted to) inhibitory control, and performance monitoring (Luna et al., 2004). The current study focuses on these two effortful control processes (i.e. response inhibition and performance monitoring; cf. error-processing) with respect to trait anger.

3.1.2. Trait anger and inhibitory control

The first process, inhibitory control, refers to the ability to suppress automatic, inappropriate, goal-incompatible behavior (such as aggression) in favor of adaptive, deliberate, goal-oriented behaviour (Luijten et al., 2014; Luna et al., 2004). A series of studies conducted by Wilkowski et al. (Wilkowski et al., 2010; Wilkowski & Robinson, 2007, 2008a; Wilkowski, 2011) have shown that high trait anger individuals (HTA) demonstrate lower inhibitory control compared to low trait anger individuals (LTA), especially following exposure to hostile stimuli, such as hostile words and angry expressions, on behavioral tasks (e.g., Stop Signal Task and Flanker Task). To our knowledge, there is only one study that assessed inhibitory control in HTA and LTA individuals with the inclusion of recording electroencephalographic (EEG) activity. Including electroencephalographic measures (e.g., Event-Related Brain potentials, ERPs) is important as they may provide information about the underlying neurophysiological mechanisms and can additionally inform about the time course of response inhibition. In this particular study, Liu et al. (2014) found faster reaction times and a decreased P3 component using difference waveforms for HTA individuals compared to LTA individuals, which is consistent with the idea of impaired response inhibition in HTA individuals. Notably, however, no differences were found between LTA and HTA individuals on the N2 component and the P3 component on NoGo trials, which are both event-related potentials often proposed to reflect inhibitory control related changes in brain activity (Luijten et al., 2014). Since effortful control deficits seem to become especially apparent when anger is primed at the behavioural level (Wilkowski & Robinson, 2008b, 2010), perhaps inhibitory deficits on the neurophysiological level for NoGo trials remained undetected as the study of Liu and colleagues included an affectively neutral Go/NoGo task. That is, given that HTA are hypothesized to allow effortful control processes to lay dormant under potentially hostile situations, whereas LTA do not (Wilkowski & Robinson, 2010), inhibitory control deficits for HTA on the neurophysiological level should also become more apparent following the activation of hostile thoughts. To test this idea, we sought to compare anger primed inhibitory control in HTA and LTA individuals using an affective Go/NoGo task with the inclusion of an electrophysiological measure.

3.1.3. Trait anger and error-processing

The second process, error-processing, refers to the optimization of goal-directed behaviour by monitoring and evaluating ongoing behaviour (Luijten et al., 2014). Error-processing is vital in order to adapt behaviour to situational demands and in order to optimize goal-directed behaviour (Botvinick, Braver, Barch, Carter, & Cohen, 2001; Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). As such, deficits in error-processing are hypothesized to lead to the continuation of inappropriate behavior (e.g., outward expressions of anger), despite its negative consequences (Luijten, van Meel, & Franken, 2011). Indeed, several studies have found reduced error-processing, as indexed by both reduced ERN and Pe amplitudes, while using different tasks in populations with externalizing psychopathology that are characterized by impulse control problems. However, mixed results have been obtained as to what component drives these error-processing deficits. Whereas some studies found evidence for both reduced ERN and Pe amplitudes (Franken, van Strien, Franzek, & van de Wetering, 2007; Luijten, van Meel, et al., 2011; Ruchsow, Spitzer, Grön, Grothe, & Kiefer, 2005), other studies either found evidence for reduced ERN amplitudes both in community (Dikman & Allen, 2000; J. Hall, Bernat, & Patrick, 2007; Heritage & Benning, 2012; Potts, George, Martin, & Barratt, 2006) and patient samples (Munro et al., 2007b; Vilà-Balló, Hdez-Lafuente, Rostan, Cunillera, & Rodriguez-Fornells, 2014; Von Borries et al., 2010) or for reduced Pe amplitudes in patient samples (Brazil et al., 2009; Chen, Tien, Juan, Tzeng, & Hung, 2005; Maurer et al., 2015). Interestingly, studies that found evidence for reduced Pe amplitudes have been mostly conducted in impulsive violent patient samples, such as female incarcerated psychopaths (Maurer et al., 2015), violent offenders with psychopathy (Brazil et al., 2009), and impulsive-violent offenders (Chen, Muggleton, & Chang, 2014). Error-processing deficits on the neurophysiological level in HTA individuals have not yet been investigated, even though there are reasons to expect that impaired error-processing may be present in high trait anger individuals as well (Robinson et al., 2012). Hence, to our knowledge this would be the first study to compare error-processing in HTA and LTA individuals on the neurophysiological level.

3.1.4. The present study

In short, the goal of the present study was to investigate effortful control (i.e. inhibitory control and error-processing) in HTA and LTA individuals on both the behavioral level and

the neurophysiological level, because there is a need to better understand the (neuro)cognitive processes associated with trait anger. To this end, we recorded EEG activity while participants high or low on trait anger performed an affective Go/NoGo task including both anger-related pictures and neutral pictures. Go/NoGo tasks are frequently used to measure inhibitory control as well as error-processing (Luijten et al., 2014). Regarding inhibitory control, event-related potential studies have repeatedly shown increased N2 amplitudes in the frontal region and increased P3 amplitudes in the frontocentral region on NoGo trials compared to Go trials (Falkenstein et al., 1999; Luijten et al., 2014; Rietdijk, Franken, & Thurik, 2014). With respect to error-processing, two error-related brain waves are of interest, namely the Error-Related Negativity (ERN), and the error positivity (Pe). The ERN arises shortly after making commission errors and reflects initial error detection (Bernstein et al., 1995). The Pe follows the ERN, and reflects the more conscious processing or awareness of the motivational significance of an error (Luijten et al., 2014; Overbeek et al., 2005). Based on the literature, we expected HTA individuals to demonstrate less inhibitory control as reflected in more commission errors on NoGo trials, especially for anger-primed NoGo trials. Moreover, we expected reduced N2 and P3 NoGo amplitudes for HTA compared to LTA individuals; with more pronounced effects on trials including anger-related pictures. Finally, we explored whether HTA individuals showed deficits in error-processing as reflected by reduced amplitudes on the ERN and the Pe compared to LTA individuals.

3.2. Methods and materials

3.2.1. *Participants*

In total, 133 undergraduate students were recruited to take part in our experiment in return for course credits or a financial compensation of 10 euros. From this group, 94 participants were selected as high or low in trait anger. HTA participants scored 21 or higher and LTA scored 15 or lower on the Dutch version of the Trait Anger Subscale (TAS) from the State-Trait Anger-Expression Inventory-2 (Hovens et al., 2014) respectively at the time of testing. The TAS has adequate psychometric qualities (Hovens et al., 2014; Spielberger, 1999) and similar cut-off criteria have been used in previous studies to select subclinical angry samples (e.g., Eckhardt & Cohen, 1997; Honk et al., 2001). The resulting LTA group consisted of 45 participants (71.7% women; *M* age = 20.76, SD = 2.44) with a mean score of 13.13 (*SD* =

1.42) on the TAS. The HTA group (79.6% women; *M* age = 20.88, SD = 3.11) consisted of 49 participants with a mean score of 24.57 (*SD* = 3.23), reflecting high scores (above the 85th percentile) on the TAS. One participant was excluded from the analyses on the Go/NoGo task because he failed to comprehend the instructions. All participants had normal or corrected-to-normal vision. This study was conducted according to the rules of the Helsinki Declaration on informed consent and confidentiality (World Medical Association, 2001). All procedures were carried out with adequate understanding and written informed consent of the participants and with permission of the local ethics committee.

3.2.2. Instruments

The Dutch version of the State Trait Anger Expression Inventory-2 (STAXI-2; Spielberger, 1999; Dutch translation: Hovens et al., 2014) measures the experience, expression, and control of anger. The STAXI-2 contains 57 items that are scored on a 4-point Likert scale (1 = "almost never", 2 = "sometimes", 3 = "often", 4 = "almost always"). The measure comprises six distinct scales, i.e. State Anger, Trait Anger, Anger Expression-In, Anger Expression-Out, Anger Control-In, and Anger Control-Out. In the current study we focused on the Trait Anger scale (T-Ang; range 10–40), which assesses the general tendency to experience frequent, intense, and longer episodes of anger. The STAXI-2 has adequate psychometric properties (Hovens et al., 2014; Spielberger, 1999).

3.2.3. Anger-related Go/NoGo task

An anger-related version of a Go/NoGo task, adapted from Luijten et al. (2011), was used. In this task, participants viewed a series of pictures with an anger-related or neutral content. Each picture was displayed for 200 ms and had a blue or yellow frame. The frame color indicated whether a stimulus was a Go or a NoGo trial. Response assignments were randomized across participants. Each stimulus was followed by a black screen for a randomly varying duration between 1020 ms and 1220 ms. Participants were explicitly instructed to respond as fast and as accurately as possible to the pictures in Go trials by pressing a button with their index finger, and to withhold their response for the NoGo trials. The task consisted of 112 different anger related pictures and 112 neutral pictures selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) and Google Images. Anger-related pictures displayed scenes of angry and/or fighting people,

whereas neutral pictures showed similar scenes of people engaged in non-angry behavior. Both picture types were matched for color, gender and number of people displayed on the pictures. Each picture was presented four times, resulting in a total of 896 trials, of which 25% were NoGo and 75% were Go trials. The amount of NoGo trials was equally divided over picture categories (i.e., 112 NoGo trials and 336 Go trials). We used a blocked design with four blocks consisting of 224 trials each. The first two blocks consisted of neutral pictures and the last two blocks consisted of anger-related pictures. This fixed order was chosen to prevent priming and carry-over effects of the anger-related pictures onto the neutral pictures. After each block, participants were given the opportunity to take a short break. The order of Go and NoGo trials was quasi randomized such that at most two NoGo trials were presented consecutively. Before starting the actual task participants performed 23 practice trials involving additional neutral pictures. Total task duration was about 22 min.

The accuracy rates for Go and NoGo trials as well as the median reaction times (RT) for the Go trials only were used as performance measures for the Go/NoGo task. The reaction time data for the Go trials were calculated after the deletion of incorrect responses and outliers for each individual, i.e., reaction times below 150 ms or above 1500 ms.

3.2.4. Procedure

Each patient was tested individually in a silent, secluded room. Participants were seated in a comfortable chair, and received a brief general introduction on the EEG measurements and the procedures. Next, electrodes were placed and participants were instructed to sit relaxed and to minimize eye-blinks and body movements during the experiment. Following these instructions, participants completed the Go/NoGo task along with two other tasks during EEG recording. E-Prime software (Version 2.0; Psychology Software Tools, Pittsburg, PA) was used to administer the tasks. Task instructions were provided before the start of each task. After completion of the three tasks, electrodes were removed and participants were seated behind a computer to complete the questionnaires[21]. After having completed the

[21] The Aggression Questionnaire (Buss & Perry, 1992), the Barratt Impulsiveness Scale (Patton et al., 1995), the Anger Rumination Scale (Sukhodolsky et al., 2001), and the Ambiguous Hostile Stories Task were also administered for different research purposes, but are not reported here as they go beyond the goals of the present study.

experiment, participants were thanked for their participation. In total, testing lasted approximately 1h and 45 min.

3.2.5. EEG recording and data reduction

The electroencephalogram (EEG) was recorded using the Biosemi Active-Two amplifier system (Biosemi, Amsterdam, The Netherlands) from 34 scalp sites mounted in an elastic cap according to the international 20/10 system (ACNS, 2006), and with two additional electrodes at FCz and CPz. Six additional electrodes were placed for offline referencing and for recording horizontal (HEOG) and vertical electro oculogram (VEOG) to correct for eye-movements and blinks. The reference electrodes were attached to the left and right mastoids. The other electrodes were placed on the outer canthi of both eyes (HEOG), and the infraorbital and supraorbital regions of the right eye (VEOG). BrainVision Analyzer 2 (Brain products GmbH, Munich, Germany) was used to process the offline data. All signals were digitized with a sample rate of 512 Hz and 24-bit A/D conversion with a bandpass of 0–134 Hz, and were filtered offline. Data were offline-referenced to the average of the left and right mastoids. Off-line, EEG and EOG activity was filtered using phase shift-free Butterworth filters (24 dB/Octave roll off) with a bandpass of .15 Hz to 30 Hz. The Go/NoGo task EEG data were segmented in epochs from 200 ms before stimulus presentation to 1450 ms after stimulus presentation and 100 ms before the response onset to 600 ms after the response onset. Ocular correction was applied using the Gratton and Coles algorithm (Gratton, Coles, & Donchin, 1983). The mean 100 ms pre-stimulus period served as baseline. Artifacts were rejected by excluding epochs exceeding ±75 μV from the average.

For the N2 and P3 components the average ERP waves were calculated for artifact free trials for neutral Go, neutral NoGo, anger-related Go, and anger-related NoGo stimuli separately. Moreover, for calculating these components segments with incorrect responses (miss for Go trials or false alarms for NoGo trials) were excluded from the analyses. The N2 was defined as the average activity within the 200–300 ms time interval after stimulus onset and was studied at a cluster of frontocentral electrodes, including FC1, Fz, FC2, FCz, and Cz (Luijten, Littel, et al., 2011) as the N2 is predominantly examined and observed over anterior scalp sites (Falkenstein et al., 1999). The P3 was defined as the average value within the 300–500 ms time interval after stimulus onset and was studied at C3, Cz, C4, FCz, and CPz (Luijten, Littel, et al., 2011; Rietdijk et al., 2014). The mean number of analyzable Go and

NoGo epochs for the N2 and P3 components after removal of the artifacts was 270 and 68 for anger-related pictures and 278 and 73 for neutral pictures respectively. Eight participants (3 LTA and 5 HTA individuals) in total were excluded from these ERP analyses because of less than 20 artifact free ERP epochs in at least one of the task conditions, which is required to obtain a reliable N2 and P3 (Rietdijk et al., 2014).

For the ERN and Pe components the average ERP waves were calculated for artifact free trials for correct Go trials (hits for Go trials) and for incorrect NoGo trials (false alarms for NoGo trials). The ERN was defined as the average value in the 0–100 ms range after response onset (e.g., Ladouceur, Dahl, Birmaher, Axelson, & Ryan, 2006; Vocat, Pourtois, & Vuilleumier, 2008). The Pe was defined as the average value in the 150–350 ms range after response onset (Alexopoulos et al., 2007; Rollnik et al., 2004). Both the ERN and the Pe were studied at FCz, Cz, and CPz as these midline electrodes are typically examined (Luijten, van Meel, et al., 2011; Rietdijk et al., 2014). To obtain reliable information for the ERN and Pe at least 6 trials are needed. In total, five participants were excluded from these ERP analyses because of fewer than 6 artifact free ERP epochs due to too few errors or due to too many artifacts. The mean number of analyzable epochs for the ERN and Pe components after removal of the artifacts was 591 for correct Go trials and 45 for incorrect NoGo trials.

3.2.6. Statistical analyses

Data were analyzed using SPSS 22.0. Repeated Measures Analyses of Variance (RM-ANOVA with Greenhouse–Geisser adjusted *p*-values) were used to analyze the accuracy rates and reaction time data on the Go/NoGo task, as well as the ERP indices of response inhibition (i.e., N2 and P3) and error monitoring (i.e., ERN and Pe). The between-subjects factor in all RM-ANOVAs was Group (HTA versus LTA). Several two-level within-subjects factors were of interest, specifically (a) Inhibition (Go versus NoGo), (b) Picture (Anger-related versus Neutral), and (c) Accuracy (Correct Go versus Incorrect NoGo). An Inhibition RM-ANOVA was conducted to analyze behavioral accuracy on the Go/NoGo task and a Picture RM-ANOVA was performed to analyze reaction time data in order to investigate general performance. Moreover, a Group × Picture RM-ANOVA was chosen to analyze group differences with respect to the behavioral accuracy on the NoGo trials, and a Group × Picture RM-ANOVA was conducted to analyze group differences regarding the reaction time data on Go trials. A Group × Inhibition × Picture × Electrode (FC1, Fz, FC2, FCz, and Cz for N2; C3, Cz, C4, FCz, and

CPz for P3) RM-ANOVA was performed for the stimulus-locked ERP analyses, and a Group × Accuracy × Electrode (FCz, Cz, and CPz) was conducted to analyze the response-locked ERP waves. Picture type was not included as a within subject variable in the analyses of the response-locked ERPs due to too few segments for each category. Post-hoc tests for interactions using Bonferroni correction for multiple comparisons were conducted only for interactions that included the between-subject factor Group.

3.3. Results

3.3.1. *Behavioral data: general performance*

As expected, participants performed less accurate on NoGo trials than on Go trials (75.6% versus 98.3% respectively), $F(1, 92) = 349.97$, $p < .001$, $\eta_p^2 = .79$.

3.3.2. Behavioral data: trait anger and performance on the anger-related Go/NoGo task

Results show that the reaction times did not differ between both groups nor was there a significant Group × Picture interaction, both *F*s < 1 (see Table 1). With regard to accuracy, participants were less accurate on anger-related NoGo trials than on neutral NoGo trials, $F(1,91) = 27.81$, $p < .001$, $\eta_p^2 = .23$. However, in contrast to predictions, no group differences were found on accuracy, $F(1, 91) < 1$, and there was also no significant Group × Picture interaction, $F(1, 91) = 1.35$, $p = .248$, $\eta_p^2 = .02$.

Table 1. Accuracy rates (in proportions) and reaction times (in ms) for both groups on the anger-related Go/NoGo task

	LTA (*N* = 44)		HTA (*N* = 49)	
	M	***SD***	***M***	***SD***
Acc Go Agr	.98	.02	.98	.03
Acc Go Neutr	.98	.02	.99	.02
Acc NoGo Agr	.74	.16	.73	.14
Acc NoGo Neutr	.78	.12	.78	.12
RT (ms) Go Agr	304	66	306	70
RT (ms) Go Neutr	307	63	305	52

3.3.3. N2 amplitudes

Fig. 1 shows the grand average waveforms for neutral and anger-related pictures at Fz and Cz for the HTA and LTA groups. As expected, a robust main effect was found for Inhibition, $F(1, 83) = 17.53$, $p < .001$, $\eta_p^2 = .17$, on the N2 component at the frontocentral electrode cluster, meaning that on average the N2 amplitude elicited by NoGo stimuli was larger than by Go stimuli[22]. Moreover, there was a main effect for Picture, $F(1, 83) = 14.96$, $p < .001$, $\eta_p^2 = .15$, indicating that the N2 amplitudes were larger for neutral pictures than for anger-related pictures. The main effect for Electrode was also significant, $F(4,332) = 31.38$, $p < .001$, $\eta_p^2 = .27$, with the largest N2 (−7.44 μV) at Fz. The Electrode × Inhibition interaction effect was significant as well, $F(4, 322) = 12.58$, $p < .001$, $\eta_p^2 = .13$. Follow-up analyses showed that for each electrode the difference in N2 amplitudes for Go and NoGo trials was significant, all *t*s > 3.08 and all *p*s < .003, although this difference was somewhat smaller for the Electrode FC1. The Picture x Inhibition interaction effect, however, was not significant, $F(1, 83) = 3.64$, $p = .060$, $\eta_p^2 = .04$. Importantly, contrary to expectations, no Group main or interaction effects were found, meaning groups did not differ regarding the N2 amplitude, all *F*s < 3.36 and all *p*s > .070.

[22] Peak measures yielded similar results regarding the N2, P3, ERN, and Pe.

3.3.4. P3 amplitudes

The P3 waveforms for anger-related and neutral pictures in both groups are displayed in Fig. 2. Again, a robust main effect was found for Inhibition, $F(1, 83) = 194.05$, $p < .001$, $\eta_p^2 = .70$, indicating that the P3 amplitudes were on average larger for NoGo trials than for Go trials. No main effect of Picture was found, $F(1, 83) = 1.81$, $p = .182$, $\eta_p^2 = .02$, although the Picture × Inhibition interaction was significant, $F(1, 83) = 15.87$, $p < .001$, $\eta_p^2 = .16$. Follow-up analyses revealed that the P3 amplitudes on Go trials were larger for neutral Pictures (1.11 μV) than for anger-related pictures (0.14 μV), $F(1, 84) = 20.33$, $p < .001$, $\eta_p^2 = .20$, whereas no differences were found with respect to picture content on NoGo trials (5.07 μV and 5.45 μV for neutral and anger-related NoGo trials, respectively), $F(1, 84) = 1.61$, $p = .209$, $\eta_p^2 = .02$. Moreover, a main effect for Electrode was found, $F(4, 332) = 15.35$, $p < .001$, $\eta_p^2 = .16$, with the largest P3 at Cz and CPz (3.52 μV and 3.51 μV, respectively). Again, a significant Electrode × Inhibition interaction effect was found, $F(4, 332) = 50.51$, $p < .001$, $\eta_p^2 = .38$. Follow-up analyses showed that for each electrode the difference in P3 amplitudes for Go and NoGo trials was significant, all *t*s > 10.58 and all *p*s < .001, whereby this difference was somewhat smaller for the Electrode C4. Finally, although a Group × Electrode interaction was found, $F(4, 332) = 50.51$, $p < .001$, $\eta_p^2 = .38$, and the P3 on NoGo trials seemed lower in HTA individuals compared to LTA individuals yet not statistically significant (F [1, 83] = 2.76, $p = .100$), no other significant interaction or main effects including Group were found for the P3 amplitudes, all *F*s < 2.76 and all *p*s ≥ .100. Follow-up analyses regarding the Group × Electrode interaction revealed no group differences on each electrode separately, all *F*s < 2.376 and all *p*s > .14. In short, these results indicate that HTA individuals did not significantly differ from LTA individuals with respect to the P3 amplitude.

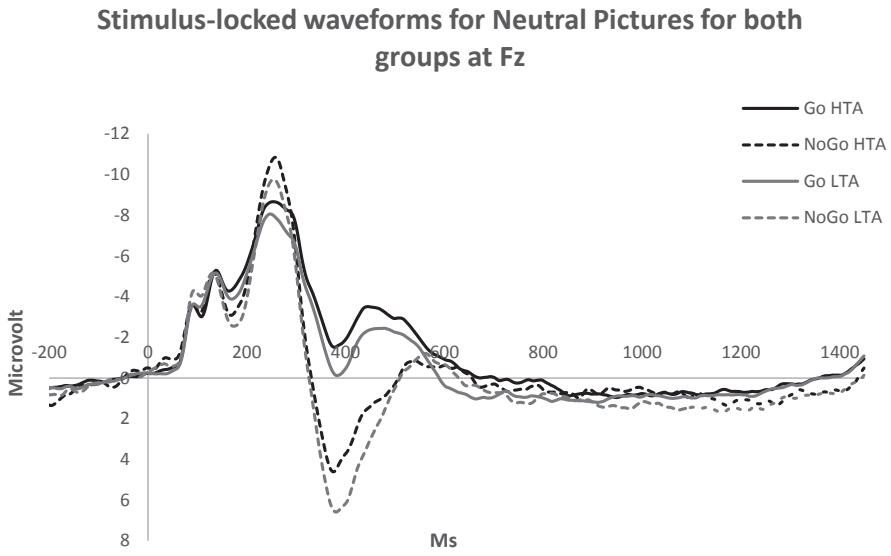
3.3.5. ERN

The response-locked ERP waveforms at FCz for correct Go and incorrect NoGo trials for both groups are depicted in Fig. 3. As expected, the ERN was larger for errors than for correct responses, $F(1, 86) = 258.40$, $p < .001$, $\eta_p^2 = .75$. There was also a main effect of Electrode, $F(2, 172) = 50.51$, $p < .001$, $\eta_p^2 = .38$, with the largest ERN at FCz (−2.52 μV). The Electrode × Accuracy interaction effect was significant as well, $F(2, 172) = 9.57$, $p < .001$, $\eta_p^2 = .10$. Follow-up analyses revealed that the difference in ERN amplitudes between accurate and incorrect trials was significant for each electrode, all *t*s > 15.37 and all *p*s < .001, whereby the difference was slightly smaller on CPz. Most importantly, no main or interaction effects including Group were found for the ERN, meaning that HTA individuals and LTA individuals did not differ regarding the ERN, all *F*s < 1.05 and all *p*s > .309.

3.3.6. Pe

Similar to the ERN, the Pe amplitudes were larger for errors than for correct responses, $F(1, 86) = 82.31$, $p < .001$, $\eta_p^2 = .49$. There was also a main effect for Electrode, $F(2, 172) = 15.71$, $p < .001$, $\eta_p^2 = .15$, with the largest Pe at Cz (9.18 μV), and a significant Electrode × Accuracy interaction effect, $F(2, 172) = 17.98$, $p < .001$, $\eta_p^2 = .17$, whereby the difference in Pe amplitudes between accurate and incorrect trials was smallest for the electrode FCz, $t = 6.82$, $p < .001$. No main effect was found for Group, $F(1, 86) = 3.61$, $p = .061$, $\eta_p^2 = .04$, nor was there a significant Group × Accuracy × Electrode interaction, $F(2, 172) = 3.01$, $p = .079$, $\eta_p^2 = .03$. Most importantly, there was a significant Group × Accuracy interaction, $F(1, 86) = 4.34$, $p = .040$, $\eta_p^2 = .05$.

Follow-up analyses indicated that the Pe amplitudes for errors, but not for correct responses, were significantly reduced in HTA individuals as compared to LTA individuals, $F(1, 86) = 4.97$, $p = .028$.



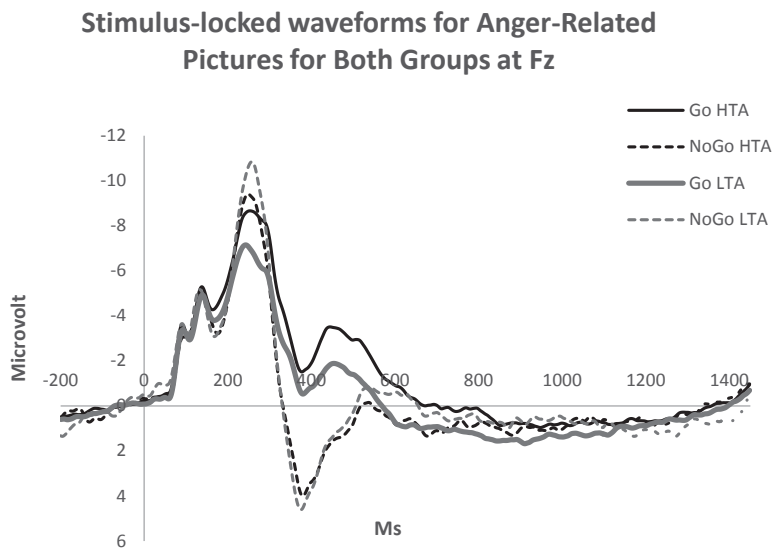


Figure 1. Grand-average stimulus-locked waveforms for Neutral and Anger-Related pictures at Fz for correct Go and NoGo trials in high (HTA) and low trait anger individuals (LTA).

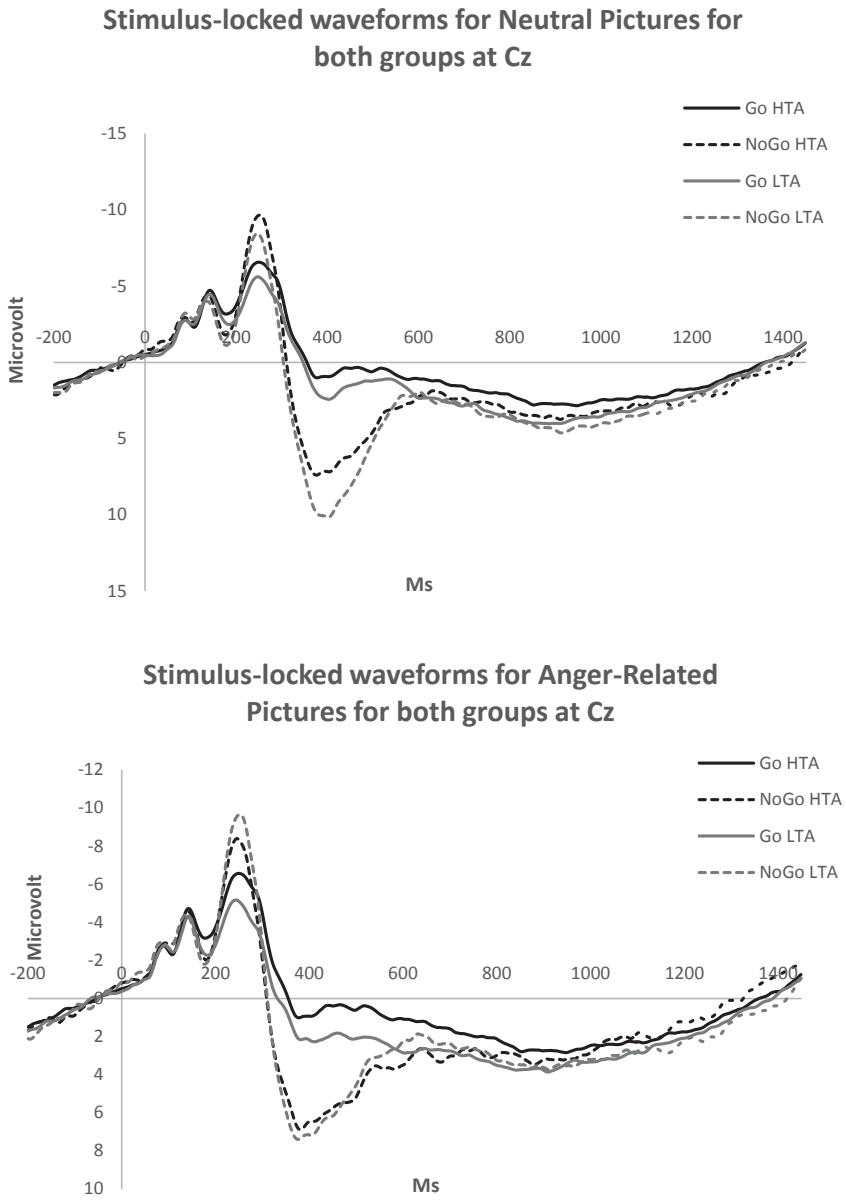


Figure 2. Grand-average stimulus-locked waveforms for Neutral and Anger-Related pictures at Cz for correct Go and NoGo trials in high (HTA) and low trait anger (LTA) individuals.

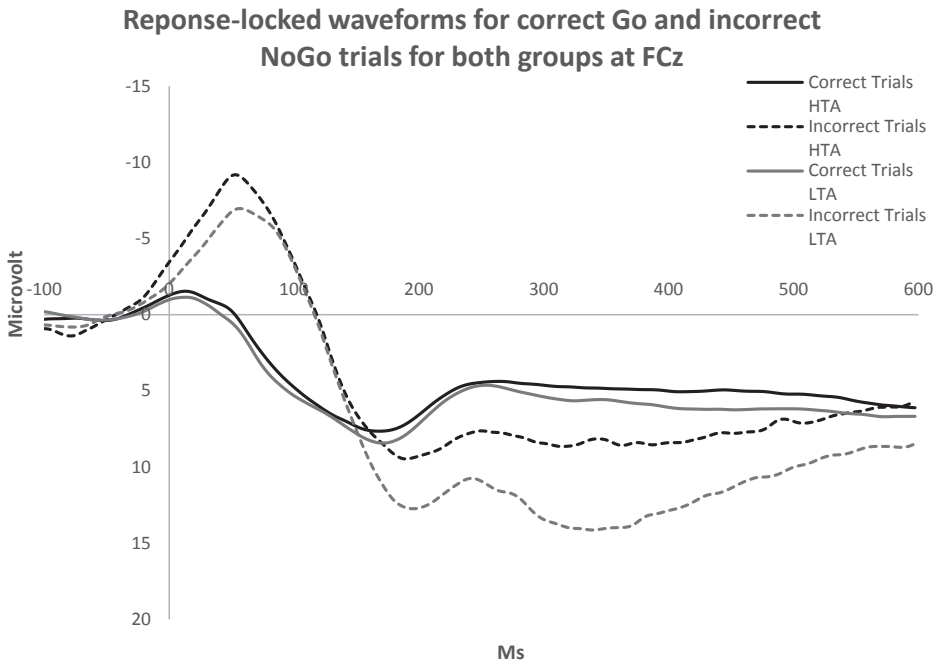


Figure 3. Grand-average reponse-locked waveforms at FCz for correct Go and incorrect NoGo trials in high (HTA) and low trait anger individuals (LTA).

3.4. Discussion

The main goal of the current study was to compare anger-primed inhibitory control and error-processing in HTA and LTA individuals. An affective Go/NoGo task was used to assess both processes in the HTA and LTA group. Based on prior research, we expected reduced inhibitory control in HTA individuals compared to LTA individuals as reflected in more commission errors and reduced N2 and P3 amplitudes on NoGo trials, especially for anger-related trials. Regarding error-processing, we explored whether reduced ERN and Pe amplitudes could be found for HTA individuals as compared to LTA individuals. Contrary to our predictions, we found no group differences regarding inhibitory control. That is, HTA and LTA individuals made comparable numbers of commission errors on NoGo trials and no significant differences were found on the N2 and P3 amplitudes. We did find consistently larger N2 and P3 amplitudes across groups for NoGo trials compared to Go trials, indicating that the affective Go/NoGo task showed the typical Go/NoGo effect. With respect to error-processing, we found reduced Pe amplitudes following commission errors on NoGo trials in HTA individuals as compared to LTA individuals, whereas the ERN amplitudes were comparable for both groups.

Given that the Pe is supposed to reflect motivational salience to errors (Overbeek et al., 2005), whereas the ERN is thought to reflect a more automatic process of error detection (Bernstein et al., 1995; Yeung et al., 2004), it seems that HTA and LTA individuals do not differ in automatic error detection. In contrast, HTA individuals show deficits in later stages of error-processing (i.e., the Pe component) compared to LTA individuals. In other words, the current study is the first study to indicate that HTA individuals show diminished motivational salience to errors compared to LTA individuals. Importantly, these deficits in later stages of error-processing, may provide an explanation for the continuation of impulsive behavior in HTA individuals despite its negative consequences. The combination of reduced Pe amplitudes and intact ERN amplitudes found in HTA individuals is in accordance with previous ERP studies showing error-processing deficiencies related to the more conscious and later processing of errors in populations characterized with externalizing psychopathology, including dysfunctional anger (Brazil et al., 2009; Chen et al., 2005; Maurer et al., 2015). Moreover, our results are in line with several fMRI studies showing reduced activation in brain regions related to error-processing (i.e., the anterior cingulate cortex and orbitofrontal cortex) in dysfunctional angry populations (Blair, 2012;

Coccaro, McCloskey, Fitzgerald, & Phan, 2007; Davidson, 2000; Fulwiler, King, & Zhang, 2012; Siever, 2008). This study adds to this literature by showing that error-processing deficits in later stages of processing are related to dysfunctional anger in healthy populations as well. However, it should be noted that, we found no group differences on the ERN, which is in contrast with previous studies that did find reduced ERN amplitudes in impulsive, externalizing populations (Dikman & Allen, 2000; J. Hall et al., 2007; Heritage & Benning, 2012; Munro et al., 2007b; Potts et al., 2006; Vilà-Balló et al., 2014; Von Borries et al., 2010). Although there is no straightforward explanation for these contradictory findings, it should be noted that the Pe seems to be more consistently identified as a marker in impulsive dysfunctional angry samples (Brazil et al., 2009; Chen et al., 2005; Maurer et al., 2015). Moreover, it seems that our finding on the Pe was not confounded by an overall reduced cognitive ability (e.g., deficits in earlier perceptual or attentional memory processes) as groups did not differ on the stimulus-locked N2 and P3 components.

Our finding that HTA and LTA individuals performed equally well on our task measuring inhibitory control was in contrast with previous studies that showed reduced hostility-primed inhibitory control in HTA individuals (Bresin & Robinson, 2013; Wilkowski & Robinson, 2008b, 2010), but in line with the study of Hull et al. (2003) who found no relation between trait anger and inhibitory control. Interestingly, studies conducted with individuals high on trait aggressiveness, a concept distinct from but closely related to trait anger (Bettencourt et al., 2006; Wilkowski & Robinson, 2010), have also shown mixed results (Denny & Siemer, 2012; Krämer, Kopyciok, Richter, Rodriguez-Fornells, & Münte, 2011; Pawliczek et al., 2013). One explanation could be that the anger-related pictures used in the current study were not salient enough to elicit sufficient recruitment of effortful resources in contrast to previous studies (Bresin & Robinson, 2013; Wilkowski & Robinson, 2008b, 2010). For instance, one difference between our study and these previous studies is that we used anger-related pictures instead of anger-related words to prime anger. Perhaps individuals are more likely to ruminate on anger in response to processing personally relevant anger-related words compared to anger-related pictures. Thus, perhaps stronger effects are to be found when relevant threat-related words are used instead of general hostile pictures (also see Siegle, Steinhauer, Thase, Stenger, & Carter, 2002; Verona & Bresin, 2015). However, in contrast to this explanation, we did find reduced accuracy rates

and N2 amplitudes for anger-related pictures compared to neutral pictures, indicating reduced response inhibition for anger-related pictures compared to neutral words.

The findings regarding the N2 and P3 components were in line with a previous study from Liu et al. (2014), who also failed to find reduced N2 and P3 components for HTA individuals compared to LTA individuals. The current study adds by showing that the N2 and P3 component were comparable in HTA and LTA individuals, even under conditions when anger is primed. Although response inhibition (i.e., accuracy, N2, and P3 amplitudes) seemed somewhat reduced when anger was primed, this effect was evident for both groups and not specific for HTA individuals. Interestingly, Munro et al. (2007b) employed a Go/NoGo task in violent offenders and found that violent offenders did not differ from healthy controls regarding the N2 and P3 components as well, although there seemed to be some evidence for reduced N2 NoGo effects in impulsive violent offenders low in psychopathy. Notably, Chen et al. (2005) did find lower N2 amplitudes in impulsive-violent offenders compared to offenders without an impulsive-violent criminal record. Moreover, studies conducted in juvenile violent offenders without psychopathy also revealed reduced N2 and P3 amplitudes compared to controls (Guan et al., 2015; Vilà-Balló et al., 2014). Hence, one potential explanation for these mixed findings is that, whereas neural indicators of response inhibition are related to impulsive aggressive behavior, they are not specifically related to trait anger or to instrumental aggressive behavior. Finally, one could argue that our task was not sensitive enough to detect individual differences in inhibitory control. We consider this explanation unlikely, however, since there was considerable interindividual variance in this study and prior studies have shown effects with the same task (Luijten, Littel, et al., 2011). Interestingly, using the same task, we did find trait anger to be inversely related with performance accuracy as well as error-processing in a forensic psychiatric sample, although no relation was found between trait anger and the N2 and P3 components (see chapter 4). Hence, an interesting avenue for future research would be to test the boundary conditions regarding the relation between inhibitory control, trait anger, and aggression. For instance, further research is warranted that takes in account related factors such as impulsive violent behavior and anxiety, and by specifying subgroups.

An important strength of the current study is that it allowed us to investigate underlying processes of trait anger, without addressing confounding comorbid psychopathology that is often present in violent samples, such as substance abuse.

However, extension of the present study to clinical samples, including samples with problematic anger, should provide further insight regarding anger-related response inhibition and error-processing on the neurophysiological level. Future studies should also investigate the moderating impact of motivation on the relation between trait anger on the one hand and response inhibition and error-processing on the other hand (J. Hall et al., 2007). Furthermore, studies might benefit from including trait anger as a covariate rather than dichotomizing into two groups, as this approach may be less susceptible to being influenced by confounds (e.g., anxiety). Another interesting avenue for future research would be to induce state anger and to investigate its impact on high and low trait anger individuals while performing tasks measuring inhibitory control. Finally, the reduced N2 and P3 amplitudes as well as the diminished performance on anger-related trials may be due to stimulus-order effects, given that participants always received the neutral blocks first. Other outcomes may have been obtained if the neutral and hostile pictures were presented using a random design (i.e., if picture type had been varied trial-by-trial).

In summary, our hypothesis that high trait anger individuals demonstrate impaired hostility-primed inhibitory control on both the behavioral level as well as the neurophysiological level could not be confirmed. Importantly, we did find initial evidence for impaired conscious error-processing in high trait anger individuals. This deficit may shed light on the neurobiological underpinnings of trait anger and may explain the continuation of inappropriate behavior (e.g., outward expressions of anger), despite its negative consequences. Future studies are needed to evaluate whether anger management results in reduced error-processing deficits. Another interesting avenue for research would be to investigate whether these error-processing deficits may predict treatment effects.

3.5. Conflicts of interest

The authors report no conflicts of interest.

3.6. Acknowledgments

The authors would like to thank Joost van Vierzen, Jeroen de Jong, and Emel Heybeli for their support with data collection.

Chapter Four

The Relation between Trait Anger and Impulse Control in Forensic Psychiatric Patients: An EEG Study

This chapter is submitted for publication as:
Lievaart, M., Veen, F.M. van der, Huijding, J., Franken, I.H.A., & Hovens, J.E. The Relation between Trait Anger and Impulse Control in Forensic Psychiatric Patients: An EEG Study. Manuscript submitted for publication.

Abstract

Inhibitory control is considered to be one of the key factors in explaining individual differences in trait anger and reactive aggression. Yet, only a few studies have assessed electroencephalographic (EEG) activity with respect to response inhibition in high trait anger individuals. The main goal of this study was therefore to investigate whether individual differences in trait anger in forensic psychiatric patients are associated with individual differences in hostility-primed inhibitory control using behavioral and electrophysiological measures of response inhibition. Thirty-eight forensic psychiatric patients who had a medium to high risk of recidivism of violent and/or non-violent behavior performed an affective Go/NoGo task while EEG was recorded. On the behavioral level, we found higher scores on trait anger to be accompanied by lower accuracy on NoGo trials, especially when anger was primed. With respect to the physiological data we found, as expected, a significant inverse relation between trait anger and the Error Related Negativity (ERN) amplitudes. Contrary to expectation, Trait anger was not related to the stimulus-locked Event Related Potentials (i.e., N2/P3). The results of this study support the notion that in a forensic population trait anger is inversely related to impulse control, particularly in hostile contexts. Moreover, our data suggest that higher scores on trait anger are associated with deficits in automatic error-processing which may contribute the continuation of impulsive angry behavior despite its negative consequences.

4.1. Introduction

Anger is seen as a negative approach-related emotion (Carver & Harmon-Jones, 2009) that typically entails the action tendency to counter or redress perceived wrongdoings (Fernandez, 2013). Anger can occasionally lead to positive outcomes, such as eliciting compliance and co-operation from others (e.g., van Doorn, Zeelenberg, & Breugelmans, 2014), gaining a stronger sense of control, and signaling the desire to change the problematic nature of a situation (Fischer & Roseman, 2007). Moreover, diary studies show that people experience anger regularly and mostly solve their anger in nonaggressive, prosocial ways (Averill, 1983; Kassinove et al., 1997). Anger can thus be considered as a basic and adaptive emotion which may not necessarily be problematic, if regulated properly. However, anger can also lead to less desirable outcomes. For instance, uncontrolled anger can make people say hurtful things they later regret and serves as a proximate cause of violent offending and aggressive behavior (Novaco, 2011). Anger becomes especially dysfunctional when regulated improperly or when it becomes part of someone's personality, such that it starts to interfere with daily functioning and becomes excessive in its frequency, intensity, duration, and expression (DiGiuseppe & Tafrate, 2010; Spielberger, 1999). For instance, high trait anger is a robust predictor of aggressive behavior (Bettencourt et al., 2006; Tafrate et al., 2002), and is associated with domestic violence (Barbour et al., 1998), poorer psychosocial functioning (McDermut et al., 2009), and interpersonal problems (Baron et al., 2006). Given the negative outcomes associated with anger combined with society's disapproval of angry disruptions (Stearns & Stearns, 1989), people are frequently motivated to regulate and control their angry impulses (Tice & Baumeister, 1993). Accordingly, brain regions corresponding with controlled top-down emotion regulation and inhibitory control, such as the lateral and medial prefrontal cortices, show increased activity after being provoked (Denson, Pedersen, Ronquillo, & Nandy, 2009; Krämer, Büttner, Roth, & Münte, 2008). Following this, it seems likely that anger-prone individuals lack the ability to override these angry impulses and control themselves (Davidson, 2000; Denson, 2015).

Inhibitory control is considered to be one of the key factors in explaining individual differences in trait anger and reactive aggression (Wilkowski & Robinson, 2008b, 2010). More specifically, Wilkowski and Robinson (2008b, 2010) propose in their integrative cognitive model of trait anger and reactive aggression that effortful control, the ability to

override dominant cognitive tendencies in favor of subdominant tendencies, mitigates tendencies toward anger and reactive aggression by (a) fostering reappraisal in favor of a non-hostile interpretation, (b) allowing individuals to disengage from hostile ruminative thoughts, and (c) suppressing angry expressions and aggressive behavior tendencies. Of further importance, they propose that effortful control is best conceptualized in terms of a flexible resource that can be exerted in specific contexts, such that individuals low in trait anger recruit sufficient effortful control resources in potential hostile situations in order to keep their cool, whereas high trait anger individuals do not. These assumptions concerning effortful control are in line with studies highlighting the impaired top-down control of the prefrontal cortex over limbic and subcortical regions in aggressive samples (Blair, 2012; Davidson, 2000; Siever, 2008) and with theories proposing that the cognitive control system is particularly activated under circumstances where individuals are motivated to override their automatic response tendencies (Lieberman, 2007). In line with the integrative cognitive model of trait anger and reactive aggression, convincing evidence has shown that high trait anger individuals demonstrate lower effortful control on implicit cognitive tasks, especially when anger is primed. For instance, using implicit cognitive tasks, Wilkowski and Robinson (2008a) have shown that low trait anger individuals demonstrated reduced interference compared to high trait anger individuals when primed with aggression-related words. Moreover, individuals high on trait anger showed slower response-inhibition processes for angry expressions and not for neutral expressions on a stop-signal task (Wilkowski, 2011). Finally, hostility-primed inhibitory control fostered forgiveness in both laboratory and real-life settings subsequently reducing anger and aggressive behavior (Wilkowski et al., 2010).

Despite these important insights, most studies using these implicit cognitive tasks were conducted within healthy subjects high on trait anger. Hence, there is a need for investigating the inverse relation between effortful control and trait anger in forensic patient samples, such as violent offenders. In addition, only a few studies have assessed electroencephalographic (EEG) activity while performing tasks that require effortful control. One study found reduced attentional control and diminished behavioral inhibition in high trait anger offenders on a continuous performance task as evidenced by fewer hits, more false alarm rates, and reduced relative right frontocortical activity compared to a control group (Jaworska et al., 2012). Another study found faster reaction times and a decreased P3

component using difference waveforms (NoGo minus Go) on a Go/NoGo task in high trait anger individuals compared to low trait individuals, suggesting impaired response inhibition (Liu et al., 2014). Notably, they found no differences between low and high trait anger individuals on the N2 component and the P3 component on NoGo trials. As both these studies used affectively neutral tasks, little is known about the time course of hostility-primed inhibitory control in high trait anger individuals. The current study was set up with these caveats in mind.

The main goal of the current study was to investigate whether individual differences in trait anger in violent offenders are associated with individual differences in hostility-primed inhibitory control using behavioral and electrophysiological measures of response inhibition. For this purpose a novel Go/NoGo task was developed including anger-related pictures and neutral pictures. Go/NoGo tasks are often used measures to measure the ability to inhibit motor responses. Event-related potentials measured in Go/NoGo tasks show increased N2 amplitudes in the frontal region and increased P3 amplitudes in the frontocentral region on NoGo trials compared to Go trials (Falkenstein et al., 1999). Although both components are generally assumed to reflect inhibitory processing, there is still ongoing debate about what these components reflect precisely and which component best reflects inhibitory processing. For instance, the N2 signal has also been proposed to reflect response conflict (Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003), and the signaling of deviant stimulus features (Fox, Michie, Wynne, & Maybery, 2000), whereas the P3 has been proposed to reflect the actual inhibitory process itself (Kok, Ramautar, De Ruiter, Band, & Ridderinkhof, 2004; Verleger, Paehge, Kolev, Yordanova, & Jaśkowski, 2006). As trait anger is negatively related to impulse control, we expected higher scores on trait anger to be associated with reduced N2 and P3 amplitudes. Importantly, in line with the view that high trait anger individuals recruit less cognitive control resources in hostile situations, we expected these associations to be more pronounced for Anger-Related pictures. On the behavioral level, we expected higher scores on trait anger to be associated with more mistakes on the infrequent NoGo stimuli, in particular for the Anger-Related pictures.

A second important aspect of cognitive control is error-processing, which allows individuals to select the appropriate behavior, to optimize goal-directed behavior, and to subsequently adapt their behavior (Botvinick et al., 2001; Ridderinkhof, Ullsperger, et al.,

2004). As deficits in error-processing may contribute to the maintenance of impulsive angry behavior despite its negative consequences, we additionally explored whether high scores on trait anger were accompanied by more prominent deficits in error-processing as reflected by reduced amplitudes on the Error Related Negativity (ERN) and the error positivity (Pe). The ERN is a negative-going response-locked component that arises shortly after making commission errors in reaction-time tasks and has traditionally been proposed to reflect online-monitoring of performance either through automatic error detection (Bernstein et al., 1995) or through conflict monitoring (Yeung et al., 2004). The Pe is a positive-going response locked component after commission errors that follows the ERN and appears to reflect the more conscious processing or awareness of the motivational significance of an error (Luijten et al., 2014; Overbeek et al., 2005). Based on previous research showing that externalizing psychopathology is associated with impaired error-processing (Hall et al., 2007; Olvet & Hajcak, 2008), we expected higher scores on trait anger to be accompanied by reduced ERN and Pe amplitudes.

4.2. Methods and Materials

4.2.1. *Participants*

Participants were 40 Dutch speaking psychiatric inpatients from a forensic department of a psychiatric hospital in Belgium. Two participants were discarded from the data analyses as they failed to comprehend the instructions during the experiment, leaving a total of 38 participants (25 males and 13 females). The mean age of this sample was 41 years (*SD* = 9.2) with an age range from 23 to 58 years. The sample consisted of patients with complex psychiatric disorders and various comorbidities who had a medium to high risk of recidivism of violent and/or non-violent behavior related to their psychopathology, such as theft, arson, robbery, drug and alcohol-related crimes, aggravating assault and battery, domestic violence, and murder. Patients with sexual offenses, primary psychopathy, paraphilias, or exclusive addiction problems are not treated in this forensic hospital and were thus not included in this study. This study was conducted according to the rules of the Helsinki Declaration on informed consent and confidentiality. Informed consent was obtained from all individual participants included in the study. Approval was obtained in writing by the coordinating Ethical Committee of the “Broeders van Liefde” hospitals. Table 1 presents the descriptive statistics for the demographic variables, Observed Aggressive Behavior, and Trait

Anger for the total inpatient forensic psychiatric sample. Notably, the mean Trait Anger score of our sample was in the 52nd percentile of the standardized sample (Hovens et al., 2014).

Table 1. Summary Statistics for Demographic variables, Observed Aggressive Behavior, and Trait Anger for the Forensic Psychiatric Inpatient Sample.

	M	*SD*
Demographic variables		
Age	41.00	9.20
Males (%)	65.8%	
Anger-Related Measures*		
TAS	17.45	4.71
OSAB-Agg	13.11	3.64

**Note*. TAS = Trait Anger Scale; OSAB-Agg = OSAB Aggressive Behavior.

4.2.2. Instruments

The Dutch version of the State Trait Anger Expression Inventory-2 (STAXI-2; Spielberger, 1999; Dutch translation: Hovens et al., 2014) measures the experience, expression, and control of anger. The STAXI-2 contains 57 items that are scored on a 4-point Likert scale (1 = "almost never", 2 = "sometimes", 3 = "often, 4 = "almost always"). The measure comprises six distinct scales, i.e. State Anger, Trait Anger, Anger Expression-In, Anger Expression-Out, Anger Control-In, and Anger Control-Out. In the current study we only focused on the Trait Anger scale (*T-Ang*; range 10 - 40), which assesses the general tendency to experience more frequent, more intense, and longer episodes of anger. The STAXI-2 has adequate psychometric properties (Hovens et al., 2014; Spielberger, 1999).

The Observation Scale for Aggressive Behavior (OSAB; Hornsveld, Nijman, Hollin, & Kraaimaat, 2007) measures observed ward behavior, and includes the subscales Irritation/anger, Anxiety/gloominess, Aggressive behavior, Antecedent (to aggressive behavior), Sanction (for aggressive behavior), and Social behavior. The OSAB comprises of 40 items. The staff scores the behavior of inpatients on the ward that has taken place in the preceding week on a four-point Likert scale (from 1 = "no" to 4 = "frequently"). The psychometric properties of the OSAB were evaluated in 220 violent forensic psychiatric inpatients and turned out to be good with sufficient internal consistency, adequate test-retest-reliability, and good inter-rater reliability (Hornsveld et al., 2007).

4.2.3. Task paradigm

E-Prime software (Version 2.0; Psychology Software Tools, Pittsburg, PA) was used to administer the tasks. Participants completed all tasks on windows based computers.

4.2.3.1. Aggression-related Go/NoGo Task

An aggression-related version of a Go/NoGo task adapted from Luijten, Littel, and Franken (2011) was used. In this task, participants viewed a series of pictures with an Anger-Related or Neutral content. Each picture was displayed for 200 ms and had a blue or yellow frame. The frame color indicated whether a stimulus was a Go or a NoGo trial. Response assignments were randomized across participants. Each stimulus was followed by a black screen for a randomly varying duration between 1020 ms and 1220 ms. Participants were explicitly instructed to respond as fast and as accurate as possible to the pictures in Go trials by pressing a button with their index finger, and to withhold their response for the NoGo trials. The task consisted of 112 different Anger-Related pictures and 112 Neutral pictures selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) and Google Images, that were matched for color, gender and number of people displayed on the pictures. Anger-Related pictures displayed scenes of angry and/or fighting people, whereas Neutral pictures showed similar scenes of people engaged in non-angry behavior. Each picture was presented four times, resulting in a total of 896 trials, of which 25% were NoGo and 75 % were Go trials. The amount of NoGo trials were equally divided over picture categories (i.e., 112 NoGo trials and 336 Go trials). We used a blocked design with four blocks consisting of 224 trials each. The first two blocks consisted of Neutral pictures and the last two blocks consisted of Anger-Related pictures. This fixed order was chosen to prevent priming and carry-over effects of the Anger-Related pictures onto the Neutral pictures. After each block, participants were given the opportunity to take a short break. The order of Go and NoGo trials was quasi randomized such that at most two NoGo trials were presented consecutively. Before starting the actual task participants performed 23 practice trials involving additional Neutral pictures. Total task duration was about 22 minutes.

The accuracy rates for Go and NoGo trials as well as the median reaction times (RT) for the Go trials only were used as performance measures for the Go/NoGo task. The

reaction time data for the Go trials were calculated after the deletion of incorrect responses and outliers for each individual, i.e., reaction times below 150ms or above 1500ms.

4.2.4. Procedure

Each patient was tested individually in a silent, secluded room. Participants were seated in a comfortable chair, and received a brief general introduction on the EEG measurements and the procedures. Next, electrodes were placed and participants were instructed to sit relaxed and to minimize eye-blinks and body movements during the experiment. Following these instructions, the experimenter explained the first task. Participants first completed an Emotion Recognition task, followed by an emotional Stroop task, and finally the Go/NoGo task during EEG recording. Task instructions were provided before the start of each task. After completion of the three tasks, electrodes were removed and participants completed the STAXI-2 along with additional measures. For sake of brevity only the results for the Go/NoGo Task and the STAXI-2 are reported in this study. After having completed the experiment, participants were thanked for their participation and were given their financial compensation. In total, testing lasted approximately 1 hour and 45 minutes. Finally, the same week in which the participants completed the experiment, the staff was asked to judge the behavior of the patient on the ward in the preceding week using the OSAB.

4.2.5. EEG Recording and data reduction

The electroencephalogram (EEG) was recorded using the BrainAmp MR Plus amplifier system (Brainproducts GmbH) with a 32 channel Acticap with Ag\AgCl electrodes (Fp1, Fp2, F7, F3, F2, F4, F8, FC5, FC1, FC2, FC6, T7, C3, C2, C4, T8, TP9, CP5, CP1, CP2, CP6, TP10, P7, P3, P2, P4, P8, O1, Oz, O2, EOG1, EOG2) according to the international 10/20 system. Two electrodes were placed to an infraorbital and a supraorbital region of the right eye for recording vertical electro-oculogram (VEOG) to correct for eye-movements and blinks. All signals were digitized with a sample rate of 500 Hz and 24-bit A/D conversion, and were filtered offline. BrainVision Recorder (Brain products GmbH, Munich, Germany) was used to process the offline data. Data were offline-referenced to the average reference on two electrodes attached to left and right mastoids. Off-line, EEG and EOG activity was filtered using phase shift-free Butterworth filters (24 dB/ Octave roll off) with a bandpass of .15 Hz to 30 Hz. The Go/NoGo task EEG data were segmented in epochs from 200ms before

stimulus presentation to 1450 ms after stimulus presentation and 100 ms before the response onset to 600ms after the response onset. Ocular correction was applied using the Gratton and Coles algorithm (Gratton et al., 1983). The mean 100 ms pre-stimulus period served as baseline. Artefacts were rejected by excluding epochs exceeding ± 100 μV from the average.

For the N2 and P3 components the average ERP waves were calculated for artifact free trials for Neutral Go, Neutral NoGo, Anger-Related Go, and Anger-Related NoGo stimuli separately. Moreover, for calculating these components segments with incorrect responses (miss for Go trials or false alarms for NoGo trials) were excluded from the analyses. The N2 was defined as the average activity within the 225-325 ms time interval after stimulus onset (based on visual inspection) and was studied at the midline frontal electrode site Fz as the N2 is predominantly examined and observed over anterior scalp sites (Falkenstein et al., 1999). The P3 was defined as the average value within the 350-550 ms time interval after stimulus onset (based on visual inspection). The P3 was studied at the midline central electrode site Cz as the P3 in this task is typically observed at midline electrodes (e.g., Rietdijk, Franken, & Thurik, 2014). The mean number of analyzable Go and NoGo epochs for the N2 and P3 components after removal of the artifacts was 268 and 73 for Anger-Related pictures and 275 and 75 for Neutral pictures respectively. Three participants in total were excluded from these ERP analyses; one because of less than 10 artifact free ERP epochs in at least one of the task conditions and two participants as a result of their low performance accuracy on the behavioral data (accuracy rate below 50% on Go trials).

For the ERN and Pe components the average ERP waves were calculated for artifact free trials for correct Go trials (hits for Go trials) and for incorrect NoGo trials (false alarms for NoGo trials). The ERN was defined as the average value in the 25-75 ms range after response onset (e.g., Luijten, van Meel, & Franken, 2011; Rietdijk et al., 2014). The Pe was defined as the average value in the 200-400 ms range after response onset (Rietdijk et al., 2014). Both the ERN and the Pe were most clearly visible at the midline electrode Cz, therefore these electrodes were chosen in the response-locked analyses (Easdon, Izenberg, Armilio, Yu, & Alain, 2005; Herrmann, Römmler, Ehlis, Heidrich, & Fallgatter, 2004). To obtain reliable information for the ERN and Pe at least 6 trials are needed (Olvet & Hajcak, 2009; Pontifex et al., 2010). In total, 6 participants were excluded from these ERP analyses; four participants because of fewer than 6 artifact free ERP epochs due to too few errors or

due to too many artefacts and two participants as a result of their low performance accuracy (<75% correct on Go trials). The mean number of analyzable epochs for the ERN and Pe components after removal of the artifacts was 598 for correct Go trials (300 and 298 for Neutral and Anger-Related Go trials respectively) and 47 for incorrect NoGo trials (25 and 27 for Neutral and Anger-Related NoGo trials respectively).

4.2.6. Statistical analyses

Data were analyzed using SPSS 22.0. Missing values on the STAXI-2 and the OSAB were replaced with the individual participant's series mean. A Repeated Measures Analysis of Variance (RM-ANOVA with Greenhouse-Geisser adjusted *p*-values) was used to analyze the accuracy rates on the Go/NoGo task with Inhibition (Go versus NoGo) as within subjects variable as well as for the reaction time data on Go trials with Picture (Anger-Related versus Neutral) as within-subjects factor. Further, Repeated-Measure Analyses of Covariance (RM-ANCOVA; with Greenhouse-Geisser adjusted *p*-values) were used to analyze the accuracy rates on the Go/NoGo task with Picture as within-subjects variable and Trait Anger as covariate. Finally, RM-ANCOVA's were used to analyze the ERP indices of response inhibition with Inhibition and Picture as within-subjects variables and Trait Anger as covariate for the N2 and P3 component, and with Accuracy (Correct Go versus Incorrect NoGo) as within-subjects variables for the ERN and Pe component. In order to prevent the covariate from altering the main effect of the repeated measure while running the ANCOVA, Trait Anger scores were centered using the method of Delaney and Maxwell (1981), i.e. using the individual Trait Anger score minus the mean Trait Anger score of all participants so that the mean of the covariate trait anger was by definition equal to zero. Pearson correlation coefficients were calculated for the accuracy on the Go/NoGo task and ERP indices on the one hand and Trait Anger on the other hand.

4.4. Results

4.4.1. *Behavioral Data: Trait anger and performance on inhibition*

Table 2 presents the accuracy and reaction time data on the Go/NoGo task. On average participants were less accurate on NoGo trials than on Go trials (77.7% versus 95.5% respectively), $F(1,35) = 71.82$, $p < .001$, $\eta_p^2 = .67$. With regard to the reaction time data, no

differences were found between Anger-Related Go trials (*M* = 371.78, *SD* = 83.28) and Neutral Go trials (*M* = 372.93, *SD* = 77.04), *F* < 1.

Table 2. Accuracy rates (in proportions) and reaction times (in ms) on the anger related Go/NoGo task

	M	*SD*
Acc Go Agr	.95	.07
Acc Go Neutr	.96	.06
Acc NoGo Agr	.76	.17
Acc NoGo Neutr	.79	.14
RT (ms) Go Agr	372	83
RT (ms) Go Neutr	373	77

One goal of this study was to examine whether Trait Anger was negatively associated with performance on inhibition (i.e., the NoGo trials), in particular for the Anger-Related pictures. Results show that on average the accuracy of responding on NoGo trials did not differ for Anger-Related pictures and Neutral pictures, *F*(1,34) = 3.38, *p* = .075, η_p^2 = .09. Importantly, Trait Anger was significantly associated with the accuracy of responding, *F*(1,34) = 7.89, *p* = .008, η_p^2 = .19. Moreover, a significant Trait Anger x Picture interaction, *F*(1,34) = 4.50, *p* = .041, η_p^2 = .12, indicated that higher scores on trait anger were accompanied by even lower accuracy for Anger-Related pictures than for Neutral pictures. To follow-up on these results, correlations between the Trait anger scale and the accuracy rates on the NoGo trials were calculated. As expected, higher trait anger scores were associated with lower accuracy on both Neutral (*r* = -.36, *p* =.034) and Anger-Related NoGo trials (*r* = -.47, *p* =.004). Moreover, accuracy was indeed lower for Anger-Related pictures than for neutral pictures.

To examine whether these effects of trait anger were specific for NoGo trials, we additionally explored whether trait anger influenced accuracy of responding on Go trials in a similar fashion. Results revealed that, similar to the accuracy of responding on NoGo trials, accuracy for the Go trials on average did not differ for Anger-Related pictures and Neutral pictures, *F*(1,34) = 3.78, *p* = .060, η_p^2 = .10. More importantly, trait anger was not related to the accuracy of responding on Go trails and neither showed an interaction with Picture content, both *F*s < 1.

4.4.2. ERP Data: Trait Anger and ERP indices of response inhibition

Another goal of this study was to investigate whether higher scores on trait anger were accompanied by decreased N2, P3, ERN, and Pe amplitudes and whether this effect was more pronounced for Anger-Related pictures.

Figure 1 depicts the grand-average stimulus locked waveforms for Neutral and Anger-Related pictures at Fz for both correct Go and NoGo trials. Contrary to expectation, the N2 amplitudes on Go and NoGo trials in general did not differ, $F(1, 33) = 2.61$, $p = .116$, $\eta_p^2 = .07$, and there was also no significant Picture x Inhibition interaction, $F < 1$. We did find a main effect of Picture on the N2 component, $F(1, 33) = 8.10$, $p = .008$, $\eta_p^2 = .20$, showing a less negative wave on the N2 component for the Anger-Related pictures compared to Neutral pictures. Importantly, Trait Anger was not related to the N2 amplitude, $F < 1$. As a general inhibition effect on the N2 amplitude was not found, no follow up analyses were conducted regarding the N2 component.

The P3 amplitudes for Anger-Related and Neutral pictures at Cz for correct Go and NoGo trials can be seen in Figure 2. As expected, the P3 amplitudes were generally larger for NoGo trials than for Go trials, $F(1,33) = 67.46$, $p < .001$, $\eta_p^2 = .67$. However, the P3 amplitudes did not significantly differ for Anger-Related trials and Neutral trials, $F(1,33) = 3.17$, $p = .084$, $\eta_p^2 = .09$. Moreover, the Inhibition x Picture interaction was not significant, $F(1,33) = 1.00$, $p = .323$, $\eta_p^2 = .03$. Importantly, Trait Anger was not related to the P3 component, $F(1,33) = 3.72$, $p = .062$, $\eta_p^2 = .10$, and no significant interaction effects of Trait Anger were found, all Fs <1. In sum, these results seem to suggest that Trait Anger is not associated with the inhibition associated P3 amplitude.

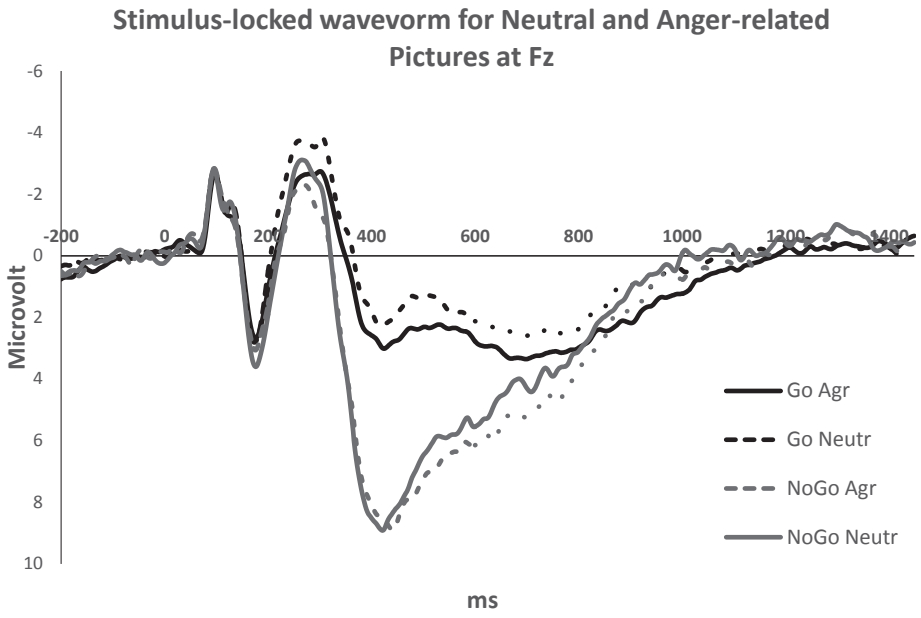
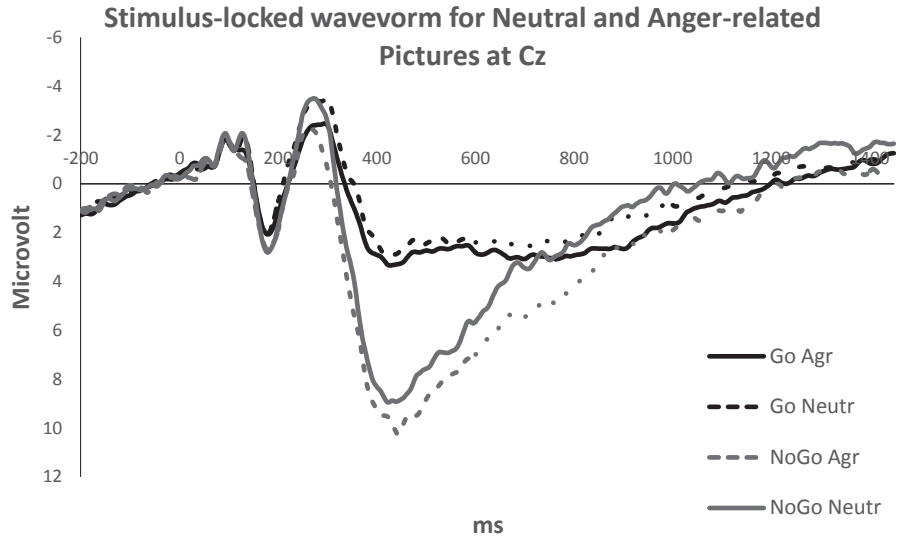


Figure 1. Grand-average stimulus-locked wavevorms for Neutral and Anger-Related pictures at Fz for correct Go and NoGo trials



.

Figure 2. Grand-average stimulus-locked wavevorms for Neutral and Anger-Related pictures at Cz for correct Go and NoGo trials.

The grand-average response-locked waveforms at Cz for correct Go and incorrect NoGo trials are depicted in Figure 3. According to expectation, the ERN was larger for errors

than for correct responses, $F(1,30) = 20.27$, $p < .001$, $\eta_p^2 = .40$. There was no main effect of Trait Anger, $F(1,30) = 2.04$, $p = .164$, $\eta_p^2 = .06$. In line with our hypothesis, we did find a significant Accuracy x Trait anger interaction, $F(1,30) = 4.75$, $p = .037$, $\eta_p^2 = .14$, indicating that higher scores on trait anger were associated with reduced ERN amplitudes[23,24].

Similar to the ERN, the Pe amplitudes were larger for errors than for correct responses, $F(1,30) = 22.82$, $p < .001$, $\eta_p^2 = .43$ (see Figure 3). However, no main or interaction effect of Trait Anger was found for the Pe, both *p*s > .80, indicating that Trait Anger was not significantly related to the Pe.

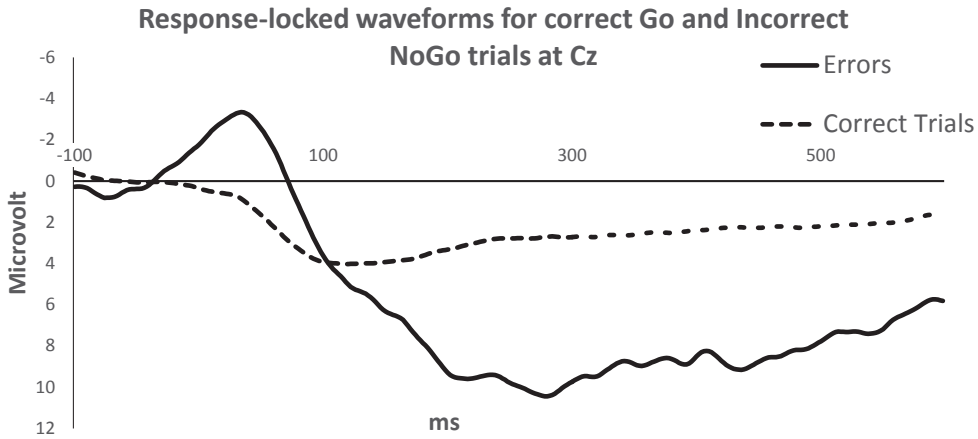


Figure 3. Grand-average response-locked wavevorms for Neutral and Anger-Related pictures at Cz for correct Go and incorrect NoGo trials.

[23] A reviewer suggested that since trait anger is related to accuracy, accuracy may have accounted for the ERN effects (as there may be more errors and therefore more segments to average together with the possibility of attenuated amplitudes). In order to rule out this possibility, we performed a partial correlation between trait anger and the ERN controlling for accuracy, which remained significant ($r = .424$, $p = .018$). Moreover, the ERN was not significantly related to accuracy.

[24] Importantly, peak measures yielded similar results. Moreover, the significant Accuracy x Trait Anger interaction was retained when both Fz and Cz were included in the Repeated Measures ANOVA.

4.5 Discussion

The main goal of the present study was to investigate whether individual differences in trait anger in violent offenders are associated with individual differences in hostility-primed inhibitory control using behavioral and electrophysiological measures of response inhibition. On the behavioral level, we expected higher scores on trait anger to be associated with more mistakes on the infrequent NoGo stimuli, in particular for the Anger-Related pictures. Consistent with these predictions, higher scores on trait anger were accompanied by lower accuracy on NoGo trials, and even lower accuracy for Anger-Related pictures. On the neurophysiological level, we expected higher scores on trait anger to be associated with reduced NoGo N2 and P3 amplitudes and with reduced ERN and Pe amplitudes. Contrary to our expectations, analyses of the stimulus locked ERP waves revealed that both the N2 and the P3 components were not related to trait anger. With respect to the response locked ERP waves, we did find a significant inverse relation between trait anger and the ERN amplitudes for incorrect NoGo trials, but not between trait anger and the Pe.

Previous behavioral studies have shown that inhibitory control is related to trait anger and reactive aggression, particularly in hostile contexts (Wilkowski et al., 2010; Wilkowski & Robinson, 2008a; Wilkowski, 2011). Most of these studies were conducted within healthy subjects high on trait anger and used a flanker task, leaving the question to what extent these results could be generalized to more pathological samples, such as violent offenders, and different measures of cognitive control. The current study shows that also in violent forensic psychiatric patients inhibitory control is inversely related to trait anger. Moreover, we found the same inverse relation among trait anger and hostility-primed inhibitory control with a new measure of response inhibition: an affective Go/NoTask. Hence, the current study adds further support to the integrative cognitive model of trait anger and reactive aggression (Wilkowski & Robinson, 2008b, 2010) and converges with theories emphasizing the situation-specificity of self-control (Kerns et al., 2004). Finally, results are also in line with other models highlighting the importance of self-control with respect to aggression and violence, such as the I3 theory (Finkel, 2007) and the General Aggression Model (DeWall et al., 2011).

Analyses of the stimulus locked ERP waves revealed that both the N2 and the P3 components were not related to trait anger. Given that individual differences in trait anger in violent offenders are related to deficits in impulse control on a behavioral level, these

relations are not easily explained by deficits in neural processes involved in response inhibition. The absence of an inverse relation between trait anger and the N2 and P3 in the current study seems in line with the results of the study of Liu and colleagues (2014), who also found no differences on the N2 and P3 components between low and high trait anger healthy participants on NoGo trials in a visual Go/NoGo paradigm, and with results from Munro and colleagues (2007b) who found that violent offenders with psychopathy made more errors of commission on NoGo trials, but did not differ from healthy controls regarding the N2 and P3 components. On the contrary, our results are not in line with those of Chen, Tien, Juan, Tzeng, and Hung (Chen et al., 2005), who found lower N2 amplitudes in impulsive-violent offenders compared to matched offenders that did not commit offenses of an impulsive-violent nature. These contrasting findings can possibly be ascribed to differences in the difficulty level and nature of the tasks that were used as well as by differences in the subtypes of violent offenders that were included. For instance, although trait anger is associated with the degree of clinical significant anger problems (DiGiuseppe & Tafrate, 2004), Davey, Day, and Howells (2005) showed that some violent offenders may be characterized by 'over-control' of anger whereas others may be better characterized by under-controlled anger. Furthermore, there are indications that especially impulsive violent offenders low in psychopathy demonstrate reduced NoGo N2 effects whereas violent offenders high in psychopathy do not (Munro et al., 2007; but also see Kiehl, Smith, Hare, & Liddle, 2000). Another possible explanation lies in the difference between the numbers of reactive and instrumental aggressors in the various research samples. Whereas reactive aggression is closely related to impulsivity, instrumental aggression is assumed to be more cold-blooded and thus less impulsive (Barratt, Stanford, Dowdy, Liebman, & Kent, 1999). As the study by Chen et al. (2005) in a sample of impulsive violent offenders, is the only one that found reduced N2 effects it seems likely that especially impulsive aggressive behavior is related to neurophysiological indices of response inhibition. Altogether, we propose that whereas reduced N2 and P3 amplitudes may be present in violent offenders, they are not related to the severity of problematic anger and are perhaps better explained by the degree of impulsive aggressive behavior exhibited. Future studies that take these subtypes into account should be conducted in order to test this idea. In doing so, these studies might benefit from including affective tasks instead of affective-neutral tasks as we found different results for hostility-primed inhibitory control compared to neutral inhibitory control.

With respect to the response locked ERP waves, we did find a significant inverse relation between trait anger and the ERN amplitudes, but not between trait anger and the Pe. Whereas the ERN is proposed to reflect the automatic stage of error detection or conflict monitoring (Bernstein et al., 1995; Yeung et al., 2004) or may also reflect attentional control to unexpected events (van Noordt et al., 2015), the Pe appears to reflect the more conscious reflection or awareness of the motivational salience of an error (Overbeek et al., 2005). Accordingly, the current study results suggest that individual differences in trait anger in violent offenders are related to individual differences in deficits in automatic error-processing, but not to deficits in more conscious stages of error-processing. It seems likely that these deficits in automatic error-processing contribute to the continuation of impulsive angry behavior in dispositionally angry people despite its negative consequences. However, it should be noted that the current study design does not allow drawing conclusions on causality. For example, it might also be that reduced error-processing predisposes individuals to impulsive behavior, including aggressive behavior, or that impulsive behavior leads to less error-processing in the long run. Moreover, the relation between trait anger and reduced error-processing may also be explained by an underlying externalizing factor as previous studies have shown impaired error monitoring to be associated with externalizing psychopathology (J. Hall et al., 2007; Olvet & Hajcak, 2008).

An important prerequisite for the current study was that the Go/NoGo task worked as intended given that we used a clinical sample as well as that the neurological indices reflected the proposed underlying processes. With respect to accuracy of responding, task performance was similar to the pattern that is usually obtained in Go/NoGo tasks, i.e. lower accuracy on NoGo trials than on Go trials (71.4% versus 95.6% respectively). Moreover, both the ERN and the Pe were significantly enhanced on incorrect NoGo trials compared to correct Go trials converging with the view these ERP's reflect error-processing. Interestingly, the P3 was significantly enhanced on NoGo trials compared to Go trials whereas the N2 was not. A possibility may be that the N2 does reflect inhibitory processing, but was not more pronounced on NoGo trials in the current study because we used a clinical violent sample who are proposed to have weakened NoGo N2 amplitudes and intact P3 amplitudes (Davidson, 2000). In line with this reasoning the N2 amplitudes were smaller for anger-related pictures than for neutral pictures, which could indicate that our violent offenders recruited less inhibitory control in hostile contexts. Moreover, we did find the general N2

Nogo effect in undergraduate students using the same task (see chapter 3). In short, we can conclude that the patients in the current study performed the task reasonably well and that the data obtained from this study are reliable.

While our study benefitted from the use of a forensic psychiatric sample and the inclusion of affective cognitive task allowing to examine neutral and anger-primed inhibitory control, the present study has some limitations that are important to address. One obvious limitation is the absence of a control group. However, our main focus was to investigate whether individual differences in impulse control are associated with individual differences in trait anger. Second, one could question the use of self-report data in forensic samples as forensic patients are sometimes inclined to minimize their experience of anger and aggression (McEwan, Davis, MacKenzie, & Mullen, 2009). However, even if this would be the case in the current study this would most likely lead to an underestimation of the actual effects and therefore have little impact on our main conclusions. Finally, our sample was relatively small.

In conclusion, the results of the current study showed that individual differences in trait anger in forensic psychiatric patients can be related to individual differences in impulse control on a behavioral level and reduced automatic error-detection on a neurophysiological level. However, the hypothesis that higher trait anger is associated with lower N2 and P3 amplitudes could not be confirmed. These results suggest that high trait anger forensic patients have difficulties with inhibitory control and error detection, which could explain the initiation and continuation of angry impulsive behavior despite its negative consequences.

4.6. Acknowledgements

We would like to thank Mike de Clercq, Els BourGonjon, Jan de Varé for their support with data collection.

4.7. Conflict-of-Interest Statement

The authors declare that they have no conflict of interest.

Part II

The Impact of Angry Rumination and Mental Fatigue on Anger and Self-Control

Chapter Five

The Impact of Angry Rumination on Anger-Primed Cognitive Control

This chapter is accepted for publication as:
Lievaart, M., Huijding, J., Veen, F.M. van der, Franken, I.H.A., & Hovens, J.E (In Press). The Impact of Angry Rumination on Anger-Primed Cognitive Control. *Journal of Behavioral Therapy & Experimental Psychiatry*.

Abstract

Background and Objectives: Recent research suggests that angry rumination augments aggressive behavior by depleting self-control resources. Yet, few studies have been conducted to empirically support this proposal. In the present study, we therefore sought to investigate the effects of angry rumination, relative to distraction, on self-reported anger and a behavioral indicator of self-control.

Methods: Seventy-two participants recalled and imagined an anger-inducing autobiographical memory and were instructed to engage in either angry rumination (n = 37) or distraction (n = 35). Following these emotion regulation instructions, participants performed an affective Go/NoGo task in order to assess behavioral self-control along with several questionnaires to assess anger related constructs.

Results: As expected, results revealed that angry rumination augmented anger, whereas anger decreased in the distraction condition. Contrary to predictions, we found no differences between both groups in performance on the affective Go/NoGo task.

Limitations: A potential limitation is we instructed our participants on how to regulate their emotions rather than letting angry rumination occur spontaneously.

Conclusions: The findings indicate that whereas angry rumination results in heightened anger, it does not seem to result in lower self-control as measured with a behavioral task that requires cognitive control. More research is needed to test the boundary conditions regarding the role of self-control in understanding rumination-induced aggression.

5.1. Introduction

People differ in how they deal with provoking situations. These differences can to a certain degree be explained by individual differences in cognitive processes. For instance, whereas some individuals easily distract themselves from a provoking event and move on, others keep dwelling and mentally rehearsing upon the provocation and fantasize on how to get back. This dwelling and revenge planning process is known as angry rumination (Sukhodolsky, Golub, & Cromwell, 2001). More narrowly defined, angry rumination refers to "perseverative thinking about a personally meaningful anger-inducing event" (Denson, 2013, p. 1). Angry rumination is typically initiated when there is a discrepancy between one's desired goal and one's actual state (L. Martin & Tesser, 1996), especially when there is a lack of perceived control over the discrepancy (Wänke & Schid, 1996). Angry rumination is often considered to be a key factor in explaining trait anger and aggression and can easily be incorporated in the main theories explaining dispositional anger and aggressive behavior (Denson, DeWall, et al., 2012; Denson, 2013; Wilkowski et al., 2010). Numerous studies have shown that people who ruminate on anger maintain or intensify their angry feelings (Bushman, 2002; Denson, Moulds, et al., 2012; Ray et al., 2008; Rusting & Nolen-Hoeksema, 1998). Moreover, ample research has shown that both state and trait angry rumination facilitate aggressive behavior (Anestis, Anestis, Selby, & Joiner, 2009; Bushman, 2002; Collins & Bell, 1997; Denson et al., 2011; Pedersen et al., 2011), including displaced aggression towards innocent victims after a seemingly minor anger-provoking event (Bushman, Bonacci, Pedersen, Vasquez, & Miller, 2005; Denson et al., 2011). Conversely, distracting oneself from ruminating (Konecni, 1974) or distancing oneself during ruminating (Mischkowski et al., 2012) decreases anger, aggressive thoughts, and aggressive behavior. Another cognitive factor that takes a central role in main theories on trait anger and reactive aggression is self-control (Denson, DeWall, et al., 2012; Denson, 2013; Wilkowski et al., 2010). Self-control refers to "the capacity for altering one's own responses, especially to bring them into line with standards such as ideals, values, morals, and social expectations, and to support the pursuit of long-term goals" (Baumeister, Vohs, & Tice, 2007, p. 1). A concept closely related to self-control is cognitive control, given that exerting self-control requires cognitive control. Cognitive control refers to the ability to flexibly, voluntarily, and adaptively coordinate behavior in the service of goal-directed behavior, and is underlain by several distinct, but interacting, components, including working memory, attentional

control, response inhibition, and error-processing (Luna et al., 2004). Self-control can be both regarded as a temperament based trait (i.e. the capacity to control ones impulses across time and situations) or as a capacity-limited commodity that can become depleted after repeated use (Baumeister et al., 2007). Both state and trait self-control deficits have been repeatedly related to aggression (DeWall et al., 2007; Stucke & Baumeister, 2006; Tice & Baumeister, 1993; Wilkowski et al., 2010; Wilkowski & Robinson, 2008b). Furthermore, patients with deficits in brain regions related to cognitive control, such as the inferior frontal cortex, often lack the ability to override their angry impulses and more often show aggressive behavior (Blair, 2012; Davidson, 2000; Siever, 2008). Adding further support to the causal relation between self-control and aggression, recent studies have shown that enhancing self-control reduces aggressive behavior (Denson, 2015; Wilkowski et al., 2015). In sum, both angry rumination and self-control deficits can be considered to be important cognitive risk-factors for anger and aggression.

Several lines of research provide indirect evidence that angry rumination and self-control may be related. Using self-report White and Turner (2014) showed that effortful control, a concept closely related to cognitive control, mediated the association between angry rumination and reactive aggression. Moreover, a recent study that used both self-report and behavioral tasks found that a disposition towards angry rumination was associated with deficient inhibition of related but at that time irrelevant information in long term memory (Whitmer & Banich, 2010). Another study conducted by Whitmer and Banich (2007) failed to find an association between a tendency towards rumination on anger and deficient inhibition in working memory, but did find angry rumination to be associated with difficulties switching to a new task set. Finally, evidence from neuroimaging research shows that angry rumination increases activity in regions related to cognitive control, including the (ventro)lateral prefrontal cortex, the dorsal medial prefrontal cortex, and the dorsal anterior cingulate cortex (Denson et al., 2009; Ray et al., 2005).

Aside from these empirical studies suggesting that angry rumination and self-control may be related, several researchers have theorized that high self-control mitigates angry rumination (Denson, 2013; Finkel, 2007; Wilkowski & Robinson, 2008b, 2010). Interestingly, Denson further proposes that angry rumination may lead to the loss of self-control and subsequent aggression by depleting self-control resources (also see Denson, DeWall, et al., 2012; Denson, 2009; DeWall et al., 2007). More specifically, he posits that stopping angry

rumination is challenging and depletes self-control resources as it requires individuals to down-regulate the intensity of their anger, to inhibit their angry thoughts, and to inhibit aggressive urges (Denson et al., 2011; Denson, 2013). Note that this account is based on ego depletion models of self-regulation (Baumeister et al., 2007), in which angry rumination is proposed to consume self-regulatory resources subsequently contributing to self-control failures, such as aggression, "in the same manner as refraining from eating a tempting donut" (Denson, 2009, P. 236). In order to answer this "causal question", experimental studies are needed. To our knowledge, the only direct investigation of the impact of angry rumination on self-control is a series of studies by Denson, Pedersen, Friese, Hahm, and Roberts (2011). In one study, these researchers showed that inducing angry rumination following provocation resulted in higher aggression and lower self-control (as measured via self-report) compared to distraction (2011; study 2), and that the reduction in self-control mediated the association between angry rumination and aggressive behavior. Moreover, another study (2011; study 4) found indirect support by demonstrating that glucose, which proposedly replenishes the ability to exercise self-control (Gailliot et al., 2007), improved performance on a Stroop task relative to placebo following angry rumination but not following distraction.

Our main goal was to conceptually replicate and further extend knowledge on the impact of angry rumination on self-control. In order to do so, we sought to investigate the effects of angry rumination on anger and using a behavioral indicator of a cognitive aspect of self-control. More specifically, we investigated whether angry rumination influenced experienced anger and performance on an anger-primed Go/NoGo task. An affective Go/NoGo task has been repeatedly used as a measure of response inhibition (e.g., Luijten, Littel, & Franken, 2011; Maurer et al., 2015; Munro et al., 2007), which is considered to be an important aspect of both cognitive control (e.g., Luna, Garver, Urban, Lazar, & Sweeney, 2004) and self-control (Muraven & Baumeister, 2000). Based on the work of Denson et al. (2011), we expected lower inhibitory control following angry rumination relative to distraction as evidenced by more commission errors on the Go/No Go task.

5.2. Materials and Methods

5.2.1. *Participants*

Seventy-three undergraduate psychology students took part in our study in return for course credits or a financial compensation of 10 euros. We randomly assigned the participants to one of two experimental conditions (Angry Rumination vs Distraction), such that approximately equal numbers of men and women were assigned to each condition. One participant was not able to come up with an autobiographical event in which he became very angry and was therefore excluded from our data analyses, leaving a total of 72 participants. Thirty-seven participants (28 women [75.7%]; *M* age = 19.97, *SD* = 1.95) were in the Angry Rumination condition, and 35 participants (26 women [74.3%]; *M* age = 20.46, *SD* = 2.20) were in the Distraction condition. The study was conducted according to the rules of the Helsinki Declaration on informed consent and confidentiality (World Medical Association, 2001) and all procedures were carried out with adequate understanding and written informed consent of the participants.

5.2.2. Materials and procedure

All participants were tested individually. Upon arrival at the laboratory, participants received general instructions regarding the experiment. Participants were then seated behind the computer screen to start with the baseline mood measure using Qualtrics Survey Software (Qualtrics Labs, Provo, Utah).

5.2.2.1. *Baseline Mood*

To measure baseline mood, participants were asked to rate their current emotional state on a Visual Analogue Scale (VAS; from 0 = “a little bit/ not at all” on the left end to 100 = “very much” on the right end) for 21 emotions. Eighteen emotions were derived from the Positive and Negative Affect Scale (PANAS; Watson, Clark, & Tellegen, 1988) and three emotions (angry, afraid, and happy) were added by the first author. The items “angry”, “hostile”, “irritated” were averaged to obtain a single measure of anger-hostility (α = .81). The remaining items were used as filler items to help disguise the experiment’s focus on anger.

5.2.3. Anger induction: Recalling an anger-inducing memory

Next, participants were given 5 minutes to write down in detail on a piece of paper three events in which they became very angry at another person. Moreover, they were instructed to indicate how angry they were at the time they experienced the anger-inducing event (from 1 "not at all angry" to 10 "extremely angry") and to what extent each situation had been solved (from 1 "completely unsolved" to 10 completely solved"). From these three reported events, the experimenter chose the least solved, most anger inducing event to discuss in more detail with the participant during an anger-inducing semi-structured interview. The interview took approximately 5 minutes. Participants received the following instructions: *"In a moment we shall discuss one of the events you have written down, in which you were really angry, during an interview. During this interview you should try to relive the memory as vividly as possible. It is important for this interview that you picture the surroundings and the situation you were in as clearly as you can. Picture the people and the objects again, hear the sounds, and let yourself relive the experience as it was. Discuss during this interview as best as you can the thoughts and feelings that you actually felt and experienced. Everything you say will stay between us, so try to answer the interview as honestly as you can".* The semi-structured nature allowed the interviewer to activate and encourage the subject to go into more detail about the anger-inducing event to evoke strong feelings of anger. This method has been shown to effectively induce anger (Lobbestael, Arntz, & Wiers, 2008). Directly after the anger-induction, participants rated their current emotional state for the second time using the VAS scales.

5.2.2.2. Emotion regulation manipulation: Angry rumination versus distraction

Following the anger induction, participants were assigned to one of the two experimental conditions: Angry Rumination versus Distraction. Specifically, both groups received a set of instructions presented on their computer screen for 45 seconds followed by six statements that were presented for 30 seconds each. Participants in the Angry Rumination condition were instructed to think back about the anger inducing memory and to focus on the emotional aspects. The specific instructions were taken from Fabiansson, Denson, Moulds, Grisham, and Schira (2012). Participants in the distraction condition were instructed to think about what the campus looks like (see Appendix A for materials).

5.2.2.3. Mood measure and emotion regulation manipulation check

After the experimental manipulation, participants were once more instructed to rate their current emotional state using the VAS scales. In addition, participants filled in some emotion regulation manipulation checks (e.g., Please indicate on a scale from 1 to 100 what percentage of the time during the past 5 minutes you thought about the angry memory you have discussed"; see Appendix B).

5.2.2.4. Anger-related Go/NoGo task

After filling in the questionnaires, participants performed a shortened version of an anger-related Go/NoGo Task (Lievaart et al., 2016)[25]. In this particular task, participants viewed a series of pictures with an Anger-Related or Neutral content. Each picture was displayed for 200 ms and had a blue or yellow frame. The frame color indicated whether a stimulus was a Go or a NoGo trial. Response assignments were randomized across participants. Each stimulus was followed by a black screen for a randomly varying duration between 1020 ms and 1220 ms. Participants were explicitly instructed to respond as fast and as accurate as possible to the pictures in Go trials by pressing a button with the right index finger, and to withhold their response for the NoGo trials. The task consisted of 56 different Anger-Related pictures and 56 Neutral pictures selected from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 2008) and Google Images, that were matched for color, gender and number of people displayed on the pictures. Anger-Related pictures displayed scenes of angry and/or fighting people, whereas Neutral pictures showed similar scenes of people engaged in non-angry behavior. Each picture was presented four times, resulting in a total of 448 trials, of which 25% were NoGo and 75 % were Go trials. The amount of NoGo trials were equally divided over picture categories (i.e., 56 NoGo trials and 168 Go trials). We used a blocked design with two blocks consisting of 224 trials each. The first block consisted of Neutral pictures and the last block consisted of Anger-Related pictures. This fixed order

[25] After completing the Go/NoGo task, participants also performed an Emotional Stroop task and an Ambiguous Hostile Stories Task. The results of these tasks are beyond the scope of this paper as both tasks do not measure inhibitory control and because of the time delay between the experimental manipulation and performing these tasks.

was chosen to prevent priming and carry-over effects of the Anger-Related pictures onto the Neutral pictures. After the first block, participants were given the opportunity to take a short break. The order of Go and NoGo trials was quasi randomized, such that at most two NoGo trials were presented consecutively. Before starting the actual task participants performed 23 practice trials involving additional Neutral pictures. Total task duration was about 15 minutes. The accuracy rates for NoGo trials was used as performance measure for the Go/NoGo task. One participant was excluded from the Go/NoGo task analyses as this participant failed to comprehend the instructions. This participant was included in all remaining data analyses.

5.2.2.5. *Personality questionnaires*

Lastly, participants filled in some personality questionnaires to ascertain that there were no important trait differences between the groups that could affect the results. *The Dutch version of the State Trait Anger Expression Inventory-2* (STAXI-2; Hovens, Lievaart, & Rodenburg, 2014; Spielberger, 1999) was used to measure the tendency to experience, express, and control anger. *The Dutch version of the Aggression Questionnaire* (AVL; Buss & Perry, 1992; Meesters, Muris, & Bosma, 1996) was used to measure trait aggressiveness. Finally, *the Dutch Version of the Barratt Impulsiveness Scale 11* (BIS-11; Lijffijt & Barratt, 2005; Patton, Stanford, & Barratt, 1995) was used to measure trait impulsivity. All the above mentioned questionnaires show good psychometric properties.

5.3. Results

5.3.1. ***Preliminary analyses***

5.3.1.1. Personality questionnaires and baseline assessments

One-way ANOVAs with Group (Angry Rumination, Distraction) as the independent variable revealed that the groups did not differ regarding their anger at baseline, F <1, nor were there group differences in how angry participants reported to feel during the recalled event, $F(1, 70) = 1.11$, $p = .296$, and the extent to which the recalled event had been solved, F <1. Lastly, there were no group differences in trait aggressiveness, $F(1, 70) = 2.14$, $p = .148$, trait impulsivity, $F < 1$, and their disposition to experience, express, and control anger, all ps >.124. Together, these data suggest that random assignment was successful.

5.3.1.2. Effectiveness of the anger induction

To examine the effectiveness of the anger induction on experienced anger, we conducted a 2 x 2 mixed ANOVA with Group (Angry Rumination, Distraction) as between-subjects variable and Time (baseline, after the anger induction) as within-subjects variable. This analysis, yielded a main effect of Time, $F(1, 70) = 133.48$, $p < .001$, $\eta_p^2 = .66$, indicating that participants felt more angry after the anger induction ($M = 35.81$, $SD = 21.12$) than they did at baseline ($M = 7.81$, $SD = 10.21$; see Figure 1). Importantly, there were no differences in experienced anger across the two conditions, nor was the Time x Group interaction significant (both Fs <1). These data imply that the anger induction was successful and had the same effect on both groups.

5.3.1.3. Effects of the Emotion Regulation Manipulation onto rumination and anger

To assess whether the participants from the Angry Rumination Condition ruminated more than participants in the Distraction condition, we performed one-way ANOVAs with Group as the independent variable and the emotion regulation manipulation checks as the dependent variables. As can be seen from Table 1, participants that were instructed to ruminate reported (a) to be more focused on their angry thoughts, their anger towards others, and their emotional response to the event, (b) to think about the angry memory more often and intensively, and (c) to have more difficulties to stop thinking about the angry memory than participants in the Distraction Condition.

To examine whether the groups differed in their anger experience after receiving the emotion regulation instructions, we conducted a 2 x 2 mixed ANOVA with Group (Angry Rumination, Distraction) as between-subjects variable and Time (after the anger induction, after the emotion regulation manipulations) as within-subjects variable. The analysis yielded a main effect of Time, $F(1, 70) = 23.22$, $p < .001$, $\eta_p^2 = .25$, and Group, $F(1, 70) = 12.09$, $p = .001$, $\eta_p^2 = .15$, that was qualified by a significant Time x Group interaction, $F(1, 70) = 73.20$, $p < .001$, $\eta_p^2 = .51$. As can be seen in Figure 1, this indicates that rumination and distraction had different effects on experienced anger. Follow-up paired t-tests within each condition, showed that rumination increased participants' anger, $t(36) = 2.72$, $p = .010$, $d = .91$, whereas distraction decreased participants' anger, $t(34) = 9.20$, $p < .001$, $d = 3.15$.

Table 1. Means (and Standard Deviations) of the Emotion Manipulation Check Questions per Condition.

	Rumination (*n* =37)	**Distraction (*n* =35)**	**Main effect of Condition**
	M (SD)	***M (SD)***	
Focused on angry thoughts	65.54 (17.74)	28.00 (25.73)	$F(1, 70) = 52.44$***
Focused on anger toward others	55.59 (25.81)	23.29 (24.77)	$F(1, 70) = 29.32$***
Focused on emotional response	66.30 (18.62)	35.29 (28.44)	$F(1, 70) = 30.29$***
Considered the positive aspects	16.92 (23.11)	16.00 (21.33)	$F(1, 70) < 1$
Considered how to deal with	36.81 (28.54)	22.37 (27.17)	$F(1, 70) = 4.82$*
Reconsidered the event from another perspective	31.97 (26.08)	19.51 (21.00)	$F(1, 70) = 4.95$*
Thought back about the angry memory	75.24 (14.70)	31.80 (26.62)	$F(1, 70) = 74.54$***
How strong/intense thought back about the anger-inducing event	63.78 (18.42)	38.23 (26.06)	$F(1, 70) = 23.29$***
Could not stop thinking about the angry memory #	4.86 (2.42)	2.94 (2.74)	$F(1, 70) = 9.98$**
Considered the situation from someone else's perspective	2.59 (2.39)	2.17 (2.42)	$F(1, 70) < 1$

 # The last two questions were scored on a scale ranging from 1 to 10 whereas the other questions were scored on a scale ranging from 1 to 100.
Note. * $p < .05$, ** $p < .01$, and *** $p < .001$

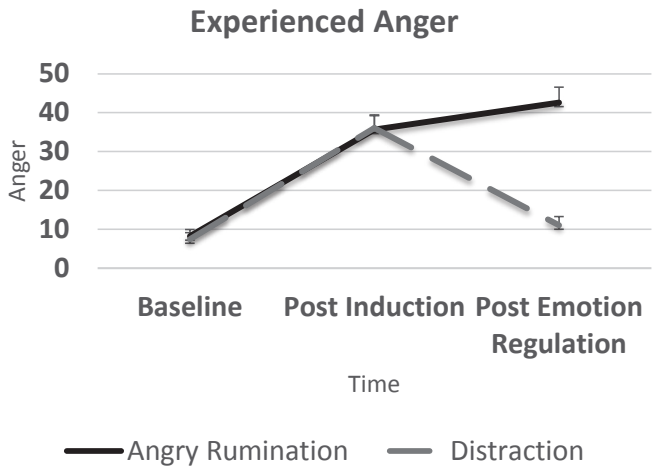


Figure 1. Experienced anger during the experiment per condition.

5.3.1.4. Associations between rumination and experienced anger

Interestingly, increased anger from time 2 (after the anger induction) to time 3 (after the emotion regulation manipulations) was associated with increased focus on the angry thoughts (r = .533, p < .001), anger towards others (r = .386, p = .001), and the emotional response to the event (r = .446, p < .001). Moreover, increased anger was associated with an increased tendency to think about the angry memory more frequently (r = .628, p < .001) and intensively (r = .495, p < .001), and with having difficulties to stop thinking about the angry memory (r = .395, p = .001). Finally, increased anger was not associated with questions related to re-appraisal, nor were angry rumination checks associated with anger reported at time 1 and time 2 (all ps > .115).

5.3.1.5. General performance on the anger-related Go/NoGo task

A 2 (Inhibition; NoGo versus Go) x 2 (Picture Content; Anger-Related versus Neutral) Repeated Measures ANOVA was conducted to assess whether the Go/NoGo task worked as supposed to by means of lower accuracy for NoGo Trials than for Go trials. As expected, there was a main effect of Inhibition, meaning that accuracy was lower for NoGo trials (79%) than for Go trials (98%), F(1, 70) = 212.02, p < .001, η_p^2 = .75. There was also a main effect of Picture Content, F(1, 70) = 34.30, p < .001, η_p^2 = .33, meaning that accuracy was lower for anger-related pictures than for neutral pictures. These effects were qualified by a significant Inhibition x Picture Content interaction, F(1, 70) = 20.14, p < .001, η_p^2 = .22, meaning that accuracy was lowest for anger-related No-Go trials. In short, the typical Go/NoGo effect was demonstrated, indicating the task worked as intended.

5.4. Main analyses

5.4.1. Effects of rumination and distraction on response inhibition

To determine whether rumination resulted in lower accuracy on NoGo trials on the affective Go/NoGo task compared to distraction, and whether this effect was more pronounced for Anger-Related Pictures compared to Neutral pictures, a 2 x 2 mixed ANOVA was conducted with Group as between-subjects variable and Picture Content (Anger-related, Neutral) as within-subjects variable for the NoGo trials. The analysis yielded a main effect of Picture Content, F(1, 69) = 27.88, p < .001, η_p^2 = .29, meaning that accuracy on NoGo trials was lower for anger-related pictures (77%) than for neutral pictures (82%). However, contrary to

expectation, the analysis yielded no main effect of Group, $F(1, 69) = 1.89$, $p = .173$, $\eta_p^2 = .03$, nor a Picture Content x Group interaction, $F < 1$, indicating that inhibitory performance was not differentially affected by rumination and distraction instructions (see Table 2). This was also true for the first 122 neutral trials, $t(69) = 1.22$, $p = .226$, indicating that shortly after the induction similar results were obtained with regard to inhibitory performance (suggesting that the duration of the task or the opportunity for 'replenishing breaks' during the task did not alter the results)[26]. Finally, a 2 x 2 mixed ANOVA revealed the groups did not differ in reaction time on Go trials, $F(1, 69) < 1$. In other words, both groups performed equally well on the affective Go/NoGo task.

Table 2. Accuracy rates (in proportions) and reaction times (in ms) per condition on the anger-related Go/NoGo task

	Distraction (*N* = 34)		**Angry Rumination (*N* = 37)**	
	M	***SD***	***M***	***SD***
Acc NoGo Agr	.80	.12	.75	.14
Acc NoGo Neutr	.84	.11	.81	.14
RT (ms) Go Agr	256	36	276	70
RT (ms) Go Neutr	262	31	276	56

5.5. Secondary analyses: Are rumination and anger associated with performance on inhibition?

To explore if the extent to which participants ruminated and felt angry after receiving the emotion regulation manipulations, regardless of which condition participants were in, was associated with inhibition performance, we examined the correlations among the accuracy rates on the NoGo trials on the one hand, and the rumination checks as well as anger at time 3 on the other hand. In general these correlations were weak and not significant (see Table 3), indicating that the degree to which participant ruminated or felt anger after the emotion regulation manipulations was not associated with performance on the Go/NoGo task

[26] A reviewer suggested that the task is quite long and potentially depleting or distracting (with breaks), which could have weakened the effects and muddled the interpretation of the study. Interestingly, a recent meta-analyses indicates that these sort of tasks do not result in ego depletion (Carter et al., 2015).

Table 3. Associations between accuracy rates on indices of response inhibition with anger at time 3 and rumination.

	Angry thoughts	Anger toward others	Emotional response	Thought back about the angry memory	Intensity	Could not stop thinking about the angry memory	Anger at time 3
Acc NoGo Agr	.07	-.08	.01	.09	-.06	-.03	.10
Acc NoGo Neutr	-.08	-.26*	-.08	-.03	-.13	-.11	-.15

Note. * $p < .05$

5.6. Discussion

Recent research suggests that angry rumination may lead to the loss of self-control and subsequent aggression by depleting self-control resources (Denson, DeWall, et al., 2012). However, few studies have investigated whether this is indeed the case (Denson et al., 2011). The present study sought to investigate the effects of angry rumination on anger and a behavioral indicator of a cognitive aspect of self-control (i.e., Go/NoGo task). Based on previous work (Denson et al., 2011), we expected lower inhibitory control following angry rumination relative to distraction. More specifically, we expected more commission errors on the Go/No Go task. However, contrary to our expectation, we found no group differences in the number of commission errors on the Go/NoGo task, indicating that inhibitory performance was not differentially affected by rumination and distraction instructions. This finding suggests that there are boundary conditions regarding the role of self-control as a mediator of the effect of angry rumination on aggression (Denson, 2013). Given that the present study differed in some aspects from the work of Denson et al. (2011) the current finding could help in identifying moderators of this relationship.

First, in the current study we used a behavioral indicator of a cognitive aspect of state self-control (i.e., Go/NoGo task), whereas Denson and colleagues used a self-report measure of state self-control (i.e., the State Self-Control Capacity Scale). As such, it could be the case that whereas the phenomenological experience of self-control is influenced by angry rumination (i.e., no longer feeling able to control oneself), angry rumination does not influence self-control as measured with behavioral tasks that require inhibitory control (note that the effects of angry rumination were marginally significant, $p = .06$ (one-tailed) on the Stroop task in the placebo condition of study 4 of Denson et al., 2011). Perhaps someone's perception of one's ability to control oneself (self-efficacy of self-control) is more important in explaining aggression than someone's actual self-control abilities. That is, if people no longer feel able or not motivated to contain themselves they may subsequently exert less self-control than they actually could. Alternatively, it may be that certain aspects of self-control are affected whereas other aspects of self-control are not, given that we specifically focused on inhibitory control in the current study as a measure of self-control. Perhaps the

cognitive restraint of aggression or cognitive modulation of the emotion anger may be affected by angry rumination whereas motoric inhibition of aggression is not. Hence, future studies should disentangle what kinds of self-control processes are affected by angry rumination and what processes are not. Moreover, there is a need of studies that distinguish between the effects of angry rumination on aggression via motivational processes (i.e., shifts in motivation orientation and attentional focus undermining self-control (Inzlicht & Schmeichel, 2012)) versus ego depletion accounts (failures in self-control as a result of exerting self-control resources from a limited resource (Baumeister et al., 2007)).

Second, in the current study anger was induced by means of reliving an interpersonal provocation instead of using an actual provocation. Anger inductions in which participants are directly insulted may elicit stronger inhibitory and emotion regulatory processes compared to inductions wherein anger is relived based on autobiographical memories. For instance, one could argue that because participants were instructed to think about an anger-inducing event rather than actually being provoked, participants were less inclined to adopt resource-depleting strategies (i.e., less downregulation of the anger experience, less suppression of angry thoughts, and fewer needs to control aggressive urges) making it less likely that self-control was reduced by means of resource depletion. If so, we suggest that self-control reductions can be better explained by *attempts* to stop angry rumination rather than angry rumination itself. However, note that in clinical practice angry rumination best accounts for aggressive acts that were conducted over long time periods, and are most likely initiated by reliving and thinking about an angering event (Sukhodolsky et al., 2001). As such, letting participants relive an anger-inducing event and ruminate about this in the lab has good external validity and should theoretically result in less self-control as well. For instance, given that anger is a negatively valenced affect (Fernandez, 2013), it is still very likely that participants attempted to down-regulate their anger and tried to suppress their angry thoughts following our angry rumination instructions. Indeed, subjects in the rumination condition reported to have difficulties stopping to think about the angry memory to a bigger extent than participants in the distraction condition. Moreover, most studies that have found effects of angry rumination on aggressive behavior used instructions to induce angry

rumination as well (Bushman et al., 2005; Bushman, 2002; Denson et al., 2009; Pedersen et al., 2011). Given that angry rumination in these studies was also not characterized by its spontaneous and uncontrollable aspects, additional studies are needed that investigate to which extent self-control is affected when these different kinds of anger inductions are used. For instance, future studies are needed in which the effects of angry rumination on past angering events are compared with the effects of angry rumination on more recent events regarding anger, aggression, and self-control.

Importantly, results showed that the manipulations led to the expected outcomes. Both groups reported higher levels of anger directly after the anger-inducing interview compared to baseline, indicating that recalling an angering event is an effective way to induce anger (Lobbestael et al., 2008). Moreover, participants in the Angry Rumination condition seemed to ruminate to a bigger extent than participants in the distraction condition. Additionally, consistent with previous studies showing angry rumination amplifies angry feelings, whereas distraction decreases angry feelings (Bushman, 2002; Rusting & Nolen-Hoeksema, 1998), participants in the Angry Rumination condition reported increased levels of anger, whereas participants in the Distraction condition reported decreased levels of anger after the emotion regulation manipulation. More specifically, the angry rumination condition was a mixture of provocation-focused and experiential rumination and adds support to Denson's suggestion that provocation-focused rumination most likely heightens angry feelings (Denson, 2013). In further support of this suggestion our study showed that higher scores on the angry rumination check questions were associated with higher levels of anger reported right after the emotion regulation manipulations, indicating that the degree to which participants adopted these types of angry rumination was associated with the amount of anger felt. In sum, our angry rumination manipulation was successful, corroborating the conclusions drawn from our study.

There are some methodological issues of the current study and suggestions for future research worth addressing. First, the participants in the current study were nonclinical undergraduate students who differ from clinical populations characterized by dysfunctional anger. For example, it is very likely that patients with dysfunctional anger ruminate more

intensively and are more frequently characterized by a loss of self-control following angry rumination. Hence, future studies are needed that investigate the causal relation between angry rumination and self-control failure in more dysfunctional angry samples. Second, the increases in angry feelings may be partly explained by demand characteristics as we used self-report measures of anger. Note however that experimental research has suggested that effects of mood inductions using explicit instruction (i.e., not masking the true purpose) are not solely artifacts of demand characteristics and can indeed lead to changes in affect (Polivy & Doyle, 1980). Third, one could argue that the effects of the angry rumination induction were negated because participants first indicated their mood before starting the cognitive control tasks. However, we consider this explanation unlikely as participants indicated to still feel anger while filling in these questions. Moreover, previous studies have demonstrated effects of angry rumination on aggressive behavior from 8 hours up to 24 hours after initial provocation (Bushman et al., 2005; Bushman & Gibson, 2010). Finally, we instructed our participants on how to regulate their emotions rather than letting rumination occur spontaneously. Future studies may benefit from the latter approach as spontaneous rumination may implicate different processes.

In conclusion, our study shows that whereas provocation-focused angry rumination resulted in heightened anger, it did not result in lower cognitive control as measured with an affective Go/NoGo. This finding has implications for understanding rumination-induced aggression. As previous research has shown that the phenomenological experience of self-control is influenced by angry rumination (Denson et al., 2011), it could be that someone's perceived ability to control oneself is more important than one's actual self-control abilities. Alternatively, it may be that rumination-induced aggression is more affected by certain types of anger inductions than others. Future research is needed to explore the boundary conditions of rumination-induced aggression.

5.7. Acknowledgements

We would like to thank Pryska Maas, Nathalie Fijan, and Natascha de Leeuw for their assistance with data collection and data management.

Appendix A. Distraction manipulation.

I want you to think about how the campus looks like in your mind and how you would describe the campus to someone who has never been on the campus before. Try to focus on the details in your mind's eye.

1. Think about how the campus looks like generally.
2. Think about which building you can find at the campus.
3. Think about the campus routes you normally walk.
4. Think about the facilities you can find at the campus.
5. Think about what makes the campus unique.
6. Think about what you would tell others about the campus.

Appendix B. Emotion regulation manipulation checks:

1. Specify to what extent during the past five minutes:
   - You reconsidered the event from another perspective.
   - You focused on your angry thoughts.
   - You focused on your anger toward others.
   - You considered the positive aspects of the event.
   - You focused on your emotional response to the event.
   - You considered how to deal with anger-inducing events.

All the above questions were scored on a VAS scale with a label of 0 = “Not at all” on the left end and a label of 100 = “Extremely” on the right end.

2. Please indicate on a scale from 1 to 100 what percentage of the time during the past 5 minutes you thought about the angry memory that you have discussed during the interview (Percentage of time: ranging from 0 = “Not at all” to 100 = “Very often”).

3. Please indicate on a scale from 1 to 100 how strong/intense you thought back about the anger inducing event (Intensity: ranging from 0 = “Not at all” to 100 = “Very intense”).

4. Please indicate on a scale from 1 to 10 to what extent during the past five minutes you could not stop thinking about the angry memory (ranging from 0 = “Very easy” to 10 = “Difficult to stop”).

- Please indicate on a scale from 1 to 10 to what extent you tried to consider the situation from someone else’s perspective (ranging from 0 = “Own perspective” to 10 = “Someone else’s perspective”).

Chapter Six

Does Mental Fatigue Have an Impact on Anger and Refusal to Cooperate Following Provocation?

This chapter is submitted for publication as:
Lievaart, M., Veen, F.M. van der, Huijding, J., Franken, I.H.A., & Hovens, J.E. Does Mental Fatigue Have an Impact on Anger and Prosocial Behavior Following Provocation? Manuscript submitted for publication.

Abstract

In the present study, we investigated whether fatigue caused by demanding mental activity leads to more anger and refusal to cooperate following provocation. Healthy participants either performed a mentally fatiguing 2-back task (n = 51) or watched a neutral documentary (n = 53) after which they were provoked by the experimenter. After participants believed the experiment to be finished, the experimenter asked participants a small request. Results showed that the manipulations led to the expected outcomes: higher fatigue after performing the 2-back task and higher anger following the provocation. However, fatigued participants did not report more anger compared to control subjects following the provocation. Moreover, most participants (>80%) cooperated with the experimenter regardless of the condition they were in. Based on the high compliance rates in this study, it seems to be the case that people can maintain control over their anger despite being mentally fatigued and provoked when motivation to do so is high.

6.1. Introduction

A driver who intentionally takes the parking spot you were about to take. Being insulted by a reviewer on your work. Potential provocations are common, and often evoke feelings of anger. Diary studies show that most people experience mild to moderate anger from several times daily to several times a week (Averill, 1983; Kassinove & Tafrate, 2002; Meltzer, 1933). Although anger can occasionally be positive (for instance, it can elicit compliance and cooperation from others (e.g., van Doorn, Zeelenberg, & Breugelmans, 2014; Van Kleef & Côté, 2007), it is typically viewed as undesirable. Especially in situations where the experienced and expressed anger is disproportional to the event that took place. For instance, anger is associated with the desire to harm the transgressor (Rubin, 1986; Tedeschi & Nesler, 1993), refusal to cooperate (Eckhardt & Deffenbacher, 1995), and can make people say hurtful things to the ones they like, love or rely on, subsequently damaging their social relationships (Baron et al., 2006). Anger is also related to workplace problems (Douglas & Martinko, 2001), health problems (T. Smith et al., 2004), and often precedes aggressive and violent behaviors (DiGiuseppe & Tafrate, 2010; Novaco, 2011). As a result of these negative consequences, people are frequently motivated to regulate and control the expression of anger in order to behave prosocially (Denson, DeWall, et al., 2012; Stearns & Stearns, 1989).

Fortunately, most of the times people are quite capable of regulating their anger. For instance, Averill (1983) showed that, although angry people often feel like being verbally aggressive (82% of the times) or physically aggressive (40% of the times), people resolve their anger mostly in nonaggressive, prosocial ways (also see Kassinove, Sukhodolsky, Tsytsarev, & Solovyova, 1997; Meltzer, 1933). Yet, people seem not always capable of regulating their anger, sometimes resulting in harmful consequences, including domestic violence, aggressive behaviors, and even murder. Therefore, detailed knowledge of the factors that determine whether people are able to control their anger and maintain prosocial behaviors is needed.

Evidently numerous factors determine whether someone is capable of controlling his anger following provocation. One important factor that is relevant for controlling anger is fatigue caused by poor sleep. Poor sleep is associated with greater irritability and short-

temperedness, and is found to be a potential risk factor for impulsive reactive aggression (Kamphuis, Meerlo, Koolhaas, & Lancel, 2012). Another factor is temporary reduced self-control as a result of ego depletion. Ego depletion refers to a state of diminished self-control due to previous acts of exerting self-control (Baumeister et al., 2007; Muraven & Baumeister, 2000). The act of exerting self-control is assumed to deplete self-control resources from a limited reservoir of self-control, leading to decreased performance of subsequent acts of self-control. Several studies have shown that participants in an ego depleted state show less anger control following provocation than non-depleted participants, such as decreased willingness to respond constructively to negative partner behavior (Finkel & Campbell, 2001), forcing a partner to maintain painful bodily poses for longer durations (Finkel et al., 2009), and more aggressive responding on laboratory aggression paradigms than controls following provocation (DeWall et al., 2007; Stucke & Baumeister, 2006). Interestingly, experimental ego depletion manipulations appear to coincide with subjective and physiological effort and feelings of fatigue (Hagger et al., 2010). Indeed, subjective reports of fatigue are often used as a manipulation check to see if the ego-depletion was successful. This raises the question to what extent effects of such ego depletion manipulations are caused specifically by engaging in acts of self-control (i.e., ego depletion) or may also be caused by a more general state of mental fatigue (Hagger et al., 2010; Kurzban, Duckworth, Kable, & Myers, 2013). In particular because, similar to "ego depletion", mental fatigue can result in diminished executive control as a result of previous demanding activity (van der Linden, Frese, & Meijman, 2003). The main difference, however, between the concepts "ego depletion" and "mental fatigue" is that ego depletion specifically refers to a state of diminished self-control as a result of engaging in acts of self-control (Baumeister et al., 2007), whereas mental fatigue can be more broadly applied as it results from demanding (cognitive) activity in general and is not restricted to acts of exerting self-control (Christodoulou, 2005; DeLuca, 2005).

One way to examine this issue would be to examine the effects of mental fatigue on anger control in conditions that *do not* tax self-control, and therefore should be unrelated to ego depletion. However, to our knowledge no such research has yet been conducted. The

main goal of this study was therefore to investigate whether fatigue caused by using a demanding, mentally fatiguing task that does not deplete self-control results in a heightened anger prone state and anger control following provocation. For this purpose, we used a 2-back task that has been previously used to induce mental fatigue (Hopstaken, van der Linden, Bakker, & Kompier, 2015). We chose to use the 2-back task because this measure requires updating of working memory representations and subsequently leads to mental fatigue (Hopstaken et al., 2015; Schmeichel, 2007), but requires little active inhibition or suppression of automatic tendencies and impulses (Miyake et al., 2000; Young et al., 2009), and therefore should not result in ego depletion. We expected that individuals who performed the fatiguing mental task would experience more anger following provocation than the non-fatigued individuals. Furthermore, we expected that the mentally fatigued individuals would be more likely to refuse to cooperate with the experimenter than the control group, given that anger is associated with refusal to cooperate (Eckhardt & Deffenbacher, 1995).

6.2. Method

6.2.1. Participants and design

One hundred and ten participants (undergraduate students) took part in our study in return for course credits. One participant was not able to complete the study due to illness and was therefore removed from our dataset. Moreover, 5 participants were excluded from our dataset due to expressed suspicion about the provocation procedure. This left a total of 104 participants (67 women and 37 men; *M* age = 21, *SD* = 3.45). We randomly assigned the participants to one of two experimental conditions (Mental Fatigue Condition versus Neutral Condition)[27], such that approximately equal numbers of men and women were assigned to

[27] Initially, this study had a 2 (Fatigue) x 2 (Reward) design. Given that the effect of the reward manipulation was insignificant and participants in the Mental Fatigue Condition or Neutral Condition followed the exact same procedure (performing a 2 back task versus watching a documentary) until the last part of the experiment where the experimenter requested participants for help, data was combined for both groups in the Mental Fatigue

each condition, χ^2 (1) = 0.03, *p* =.953. Fifty-one participants (33 women [64.7%]; *M* age = 20.96, *SD* = 3.83) were in the Mental Fatigue Condition and fifty-three participants (34 women [64.2%]; *M* age = 21.06, *SD* = 3.07) were in the Neutral Condition. There were no differences in age, trait anger, and agreeableness (all *F*s < 1), indicating random assignment was successful.

Approval for this study was obtained from the Ethical Committee of the department of psychology of the Erasmus University Rotterdam. Participation was based on a voluntary basis and participants were free to refrain from participation at any point-in-time.

6.2.2. Materials and procedure

All participants were tested individually. Upon arrival at the laboratory, participants received general instructions regarding the experiment and were asked to hand in their mobile phones and watches to make sure they could not keep track of the time during the experiment. Participants were then seated behind the computer screen to start with the baseline mood measure using Qualtrics Survey Software (Qualtrics Labs, Provo, Utah).

6.2.3. *Baseline mood*

Baseline mood was measured using *the shortened version of the Dutch Profile of Mood States* (POMS; McNair, Lorr, & Droppleman, 1971; Mellenbergh & Wald, 1990). The POMS comprises 32 items divided over five scales measuring general affective mood states, i.e. tension-anxiety, depression-dejection, anger-hostility, vigor-activity, fatigue-inertia. This five-factor model shows a good fit (Wicherts & Vorst, 2004). For each item participants were instructed to indicate their current mood state on a visual analogue scale (VAS) ranging from 0 to 100 with two opposite words on each end (for example, 0 = “active” on the left end and 100 = “passive” on the right end). The subscales were formed by reverse scoring each item

---

Condition as well as for both groups in the Neutral Condition (i.e., Mental Fatigue Condition vs Neutral Condition).

and computing the mean of the items for each subscale (see Table 1). For this study only the anger-hostility, vigor-activity, and fatigue-inertia subscales were used.

Table 1. Mood ratings per condition on baseline, post manipulation, and post anger induction.

	Baseline		Post Manipulation		Post Anger Induction	
	Neutral Condition	Mental Fatigue Condition	Neutral Condition	Mental Fatigue Condition	Neutral Condition	Mental Fatigue Condition
Anger	21.62 (13.02)	22.97 (14.61)	20.57 (13.25)	37.60 (21.44)	28.65 (18.09)	35.68 (21.57)
Fatigue	36.22 (17.70)	38.25 (20.04)	42.66 (16.64)	60.78 (19.03)	32.12 (18.11)	43.60 (20.26)
Vigor	67.68 (17.83)	66.05 (17.00)	60.24 (17.31)	38.89 (17.87)	66.71 (18.30)	57.40 (17.21)

6.2.4. Manipulation of mental fatigue

Participants were then randomly assigned to either a Mental Fatigue Condition or a Neutral Condition. Participants were told that we were developing a new intelligence test and that we wanted to study if we could influence results by priming the participants. We told them that we primed their intelligence by (a) performing a mentally challenging test, or by (b) watching an informative documentary, depending on which condition the participant was in.

Participants in the Mental Fatigue Condition then performed a visual letter 2-back task to induce a state of mental fatigue. They were asked to decide whether the letter presented on the screen was a target or non-target stimulus. In the 2-back task a stimulus is a target when the presented letter is the same as the letter presented 2 letters before. The stimuli were presented in the center of the screen and consisted of the letters B, C, D, E, G, J, P, T, V, and W in the font Palatino Linotype point size 40. In the Dutch language these letters are phonologically similar in order to prevent sound-related retrieval strategies. The letters were presented randomly with a target rate of 25%. The task lasted approximately one hour.

Participants in the Neutral Condition watched a documentary to induce a neutral, non-fatigued state. They could choose from four BBC documentaries, chosen as they had the same duration as the 2-back task and for their neutral contents. Participants were given choice out of the documentaries to make sure they were interested and to increase external validity (e.g., watching television to rest after an intensive day at work). A similar design has successfully been used in previous studies on mental fatigue (Marcora, Staiano, & Manning, 2009). After the fatigue induction participants rated their current emotional mood state using the POMS. This was followed by an anger induction.

6.2.5. Anger induction: the Trivial Pursuit task

Participants had to perform a frustrating task while being harassed by the experimenter. Participants received the following instructions: “Soon a few questions appear on your screen, with two possible answer categories. You can answer the questions by pressing the corresponding button on your keyboard. You will receive feedback whether your answer is correct or not. This task is a new intelligence measure that appears to be reliable and doesn’t

take as much time as other intelligence measures, such as the WAIS (Wechsler Adult Intelligence Scale). At the end of this task, you will receive your score and corresponding IQ. To make sure you will do your best, you can earn 25 euros if you reach the top twenty percent of the general population. Most undergraduate students think the test is doable and score above average to high. So give it your best shot". However, the questions in this task were trivial pursuit type of questions and the feedback was manipulated in such a way that all participants made 17 errors and received a corresponding IQ of 107, so that they were not able to reach the bonus score. Moreover, from the 17 answers that were displayed as wrong, four questions were easy to answer and obviously not wrong, which were included to induce a sense of injustice. The experimenter harassed the participant in multiple ways. At the start of the task, the experimenter stood closely behind the participant while smacking on his chewing gum. After the participant made a predetermined mistake (after one minute), the experimenter walked back towards his computer and made the following remark: "You should try better otherwise this will come to nothing". After two minutes the experimenter let a phone ring three times in a row, before picking it up and leaving the room, saying: "Hi. Can I call you back later? I am in the middle of an experiment. Ok, bye". When re-entering the room the experimenter monitored the participant shortly until the participant made another mistake, making a final remark: "Well, I think you can forget about those 25 euros". At the end of the test, participants were given the following feedback: "Okay, let's take a look at your IQ. You have made 17 errors; hence you score an IQ of 107. This score falls within the sixtieth percentile, which is an average score. In itself it is not so bad, but if you compare your score to the average IQ of undergraduate students, it is not so good either. You probably saw this coming, but you scored too low to get the extra reward". This method was adapted from Lobbestael, Arntz, and Wiers (2008) and has previously shown to effectively induce anger. After the anger induction participants were asked to rate their current emotional state on the POMS once more before they were told they were finished.

6.2.6. *Request*

As the participants were about to leave the room and thought the experiment was finished, the experimenter asked all participants for a small favor, to see whether participants were still willing to cooperate after being provoked by the experimenter. Participants received the following request: "Before you leave. Can I ask you something? For my own master thesis I need to test participants as well. My deadline is next week already and I still need to test 10 subjects. It concerns a task on the computer that takes 15 minutes. Would you like to help me?". Half of the participants received the same request with the addition of a promise of a reward: "I will give you a half hour of course credits in return". The response of the participant was coded categorically as "1 = Yes" or "0 = No". After participants gave a response to the request, they were debriefed about the actual goal of the study. During this debriefing we also checked for suspicion. Next, participants were scheduled for session 2 to fill in two questionnaires to control for potential confounds on experienced anger and helping behavior: the Dutch version of the State Trait Anger Expression Inventory-2 (STAXI-2, Hovens, Lievaart, & Rodenburg, 2014; Spielberger, 1999) measuring trait anger and the Dutch version of the NEO Five Factor Inventory (NEO-FFI; Costa & McCrae, 1992; Hoekstra, Ormel, & De Fruyt, 1996) measuring agreeableness. Session 2 was scheduled around a week later to avoid carry over effects of the anger induction while filling in the questionnaires.

6.2.7. Data analyses

An α of .05 was adopted for all analyses. Mixed ANOVAs were performed to test the hypotheses. Pearson's *r* is reported for the follow up t-tests and planned contrasts as a measure of effect size.

6.3. Results

6.3.1. Manipulation check: Fatigue and Vigor-activity

In order to test whether our manipulation led to a state of mental fatigue, mixed ANOVAs with ratings of fatigue and vigor-activity as the dependent variables were conducted. The baseline and post mental fatigue manipulation mood ratings are provided in Table 1.

A 2x2 (Group x Time) mixed ANOVA on the fatigue ratings revealed a main effect of Time, $F(1, 102) = 82.42$, $p < .001$, $\eta_p^2 = .45$, Group, $F(1, 102) = 10.40$, $p = .002$, $\eta_p^2 = .09$, and the expected Group x Time interaction, $F(1, 102) = 23.30$, $p < .001$, $\eta_p^2 = .19$. Because we expected no increase in ratings of fatigue from baseline to after watching the documentary for the Neutral Condition and an increase in ratings of fatigue from baseline to after performing the 2-back task for the Mental Fatigue Condition, we conducted follow-up tests within each group. These tests examined whether fatigue ratings following the manipulation (2-back versus documentary) were increased from baseline. There was an increase in ratings of fatigue from baseline to after the mental fatigue manipulation in both the Neutral Condition and Mental Fatigue Condition, respectively $t(52) = 3.72$, $p < .001$, $r = .46$ and $t(50) = 8.40$, $p < .001$, $r = .77$. Importantly, participants in the Mental Fatigue Condition reported higher ratings of fatigue compared to the participants in the Neutral Condition right after the Mental Fatigue manipulation, $t(102) = 5.24$, $p < .001$, $r = .46$, whereas groups did not differ on baseline, $t(102) = = .707$, $p = .481$, $r = .07$. Similar results were found for the vigor-activity subscale (see Table 1)[28]. In summary, these data suggest the mental fatigue manipulation was successful.

6.3.2. Manipulation check: anger experienced during the experiment

A 2x3 (Group x Time) mixed ANOVA was conducted to assess whether our anger induction resulted in more experienced anger and to see whether the effects of the anger induction were different for both groups. This analysis yielded a main effect of Time, $F(2, 204) = 27.73$, $p < .001$, $\eta_p^2 = .21$, Group, $F(1, 102) = 8.35$, $p = .005$, $\eta_p^2 = .08$, and the expected Group x Time interaction, $F(2, 204) = 15.37$, $p < .001$, $\eta_p^2 = .13$. Next, we conducted a planned contrast comparing anger on baseline with reported anger at time 2 (after watching documentary or performing the 2-back task), and reported anger at time 3 (after the anger induction). As expected, planned contrast revealed an increase in anger from baseline to time 2 (after

[28] For brevity the specific results are not reported here. Results can be obtained from the first author.

watching documentary or performing the 2-back task), $F(1, 102) = 28.87$, $p < .001$, $\eta_p^2 = .22$ as well as an expected increase in experienced anger from time 2 to time 3 (after the anger induction), $F(1, 102) = 6.62$, $p = .011$, $\eta_p^2 = .06$. Importantly, follow up paired t-tests showed that the participants in the Neutral Condition, according to expectation, reported no increase in their experienced anger after watching the documentary, $t(52) = 1.08$, $p = .285$, $r = .15$, whereas the anger induction in this group resulted in more experienced anger, $t(52) = 4.14$, $p < .001$, $r = .50$, indicating that the anger induction was successful. Moreover, the participants in the Mental Fatigue Condition experienced more anger directly after completing the 2-back task compared to baseline, $t(50) = 6.12$, $p < .001$, $r = .65$, whereby, although participants stayed angry, the anger induction had no additional effect on the experienced anger, $t(50) = 0.57$, $p = .574$, $r = .08$, indicating that performing the fatiguing task resulted in higher anger that remained till after the anger induction. Finally, numerically the participants from the Mental Fatigue Condition ($M = 36.64$, $SD = 21.79$) reported more anger after the anger induction than the participants from the Neutral Condition ($M = 28.88$, $SD = 18.39$); however, this difference was not statistically significant ($t(102) = 1.96$, $p = .052$, $r = .19$).

Importantly, the effect of both manipulations (mental fatigue manipulation and anger induction) resulted in the expected outcomes, participants in the Mental Fatigue Condition were more fatigued and reported less vigor than participants in the Neutral Condition, and after the anger induction all participants were angrier than at baseline. However, contrary to expectation the participants in the Mental Fatigue Condition did not experience more anger following provocation than the non-fatigued individuals.

6.4. Refusal to cooperate with the experimenter in response to provocation

To examine whether demanding mental activity leads to a higher likelihood of refusing to cooperate with the experimenter following provocation, we performed a Pearson Chi-Square test with Group and Cooperation as variables. We expected participants in the Mental Fatigue Condition to refuse to cooperate with the experimenter more often compared to the Neutral Condition. Unexpectedly, there was no significant association between the condition

participants were in and whether or not participants refused to cooperate, χ^2 (1) = 0.32, p = .858. Interestingly, in both conditions more than eighty percent of the participants were willing to cooperate with the experimenter despite the previous provocations (83.0% and 84.3% for the Neutral Condition and Mental Fatigue Condition respectively).

6.3.3. Mood state in relation to refusal to cooperate with the experimenter following provocation

To explore whether the participants who were not willing to cooperate with the experimenter (n = 17), differed in their mood state from the helping participants, we performed three separate independent sample t-tests with cooperation as the independent variable and ratings of fatigue, vigor, and anger reported right after the anger induction as the dependent variables. The cooperating participants did not differ from the refusing participants in their emotional states, all ts (102) < 1.

6.4. Discussion

The main goal of this study was to investigate whether fatigue caused by using a demanding mentally fatiguing task that does not deplete self-control results in a heightened anger prone state and more refusal to cooperate following provocation.

First of all, the results of our study show that performing a mentally demanding task - that does not deplete self-control - results in more subjective feelings of fatigue and experienced anger compared to watching a neutral documentary. This finding concurs with previous studies that found feelings of mental fatigue to be accompanied by heightened irritability (Lorist et al., 2000; Thackray, Bailey, & Touchstone, 1979). This seems to indicate that mental fatigue comes along with heightened anger. However, whether these angry feelings are associated with mental fatigue or are associated with the nature of these specific repetitive tasks deserves further attention.

Second, the results of the present study show that participants in the mental fatigue condition were not more susceptible to the anger induction and did not differ from the participants from the neutral condition with respect to cooperation. Overall, most

participants (more than 80 percent) cooperated regardless of the condition they were in, and regardless of the emotions they felt right after the provocation. Importantly, these results suggest that although mental fatigue is accompanied by a negative mood state, it does not necessarily result in higher angry reactivity following provocation as fatigued participants were not angrier in response to the anger induction than non-fatigued participants and also did not show less control over their anger (i.e., more refusal to cooperate).

The current study is somewhat in line with two previous studies who also found no effects of mental fatigue on emotional reactivity, although these studies did find effects on emotion regulation. More specifically, Schmeichel (2007) found that performing a working memory task reduced the ability to inhibit facial expressions of emotions in response to an emotional film clip, although they found no effects on self-reported emotional reactivity. Similarly, Grillon, Quispe-Escudero, Mathur, and Ernst (2015) found no differences in emotional reactivity in response to the pictures as measured with startle-reflex, although they did find support for reduced down regulation of aversive states after performing a working memory task compared to the control session. Our results, however, differ from studies showing that exerting self-control reduces capability to control their anger following insults (Denson et al., 2011; DeWall et al., 2007; Finkel et al., 2009; Stucke & Baumeister, 2006). As our study had the main focus on mental fatigue and not on ego depletion, this is the first study to indicate that whereas the exertion of self-control may lead to less anger control, mental fatigue seems not. These results are interesting as they suggest that self-control deficits following ego depletion with respect to anger control cannot easily be accounted for by mental fatigue effects. Yet, before such a firm conclusion can be drawn future studies should try to directly compare the effects of ego depletion tasks and demanding mental fatiguing tasks that do not require self-control on anger control.

Several alternative explanations for these results also deserve consideration. First, whereas all previous experiments have focused on aggressive responding following provocation, our study measured aggressive behavior indirectly by focusing on refusal to cooperate. It may be that the effects of mental fatigue differ for cooperative behavior in

response to provocation compared to aggressive responses in reaction to provocation (also see McGinley & Carlo, 2006). Finally, it may be that our anger induction did not elicit a sufficient amount of anger needed to evoke less constructive responding. Perhaps more intense feelings of anger are necessary in order to lose control over anger and to show more destructive behavioral responses (Potegal & Qiu, 2010). Note however, that we used a validated anger induction (Lobbestael et al., 2008) of which the effects on anger are comparable to previous studies that did find an effect of induced anger on aggressive (e.g., DeWall et al., 2007; Stucke & Baumeister, 2006).

A central tenet of motivational theories of fatigue and self-control is that exerting control and fatigue cause a shift in motivation towards more rewarding activities with less costs and away from less rewarding ones with higher costs (Hopstaken et al., 2015; Inzlicht & Schmeichel, 2012). Importantly, according to these theories, the ability to control oneself can be maintained when incentives are sufficient and motivation to do so is high. There were perhaps several motivational factors that could explain the high compliance rate (over 80 percent) in this study. Factors that could have motivated participants to cooperate despite being in an angry, fatigued state are: benefits for the self (e.g., the need for course credits), high motivation to comply with an authority figure (Milgram, 1974), the face-to-face return visit (Winstok, 2007). Regardless of the precise motive that may have driven participants' compliance, our main argument here is that even after performing a mentally demanding task and after being provoked most subjects maintained the ability to control their anger when motivated to do so. These results are in line with diary studies showing that people are mostly able to control their anger, despite being provoked (e.g., Averill, 1983). As the expression of anger can be considered a self-control failure, given that people in general are highly motivated to control their anger (Stearns & Stearns, 1989) and angry behaviors are oftentimes expressed despite being not beneficial for the self (as otherwise control over anger was not needed in the first place), the present study indicates that mental fatigue is not a disinhibiting factor.

An important limitation of this study is that we only used one simple request as outcome measure for cooperative behavior. Several measures of cooperative behavior or a

more quantitative approach might have been even more informative. A strong benefit of this study on the other hand is that the request used in this study (a small favor) has strong external validity. Finally, some may argue that working memory tasks require attentional control and should therefore be ego depleting. As explained above, self-control specifically refers to a subset of self-regulatory processes that "aim to override unwanted, prepotent impulses or urges" (Hofmann, Schmeichel, & Baddeley, 2012, p. 174). As the 2-back task requires no such kind of control, we consider the argument that working memory tasks works ego depleting as unlikely[29]. If these tasks are to be conceptualized as ego-depleting tasks, one could argue that any performance task can be conceptualized as such (as performance tasks always require some kind of executive function) and subsequently the distinction between the concepts "ego depletion" and "mental fatigue" becomes meaningless. Moreover, there is some evidence that performing working memory tasks do not result in ego depletion (Carter et al., 2015). In fact, these authors show in their meta-analyses that the ego depletion effects may not be a real phenomenon.

Mindful of these limitations, we conclude from our study that, although performing a mentally fatiguing task that does not deplete self-control is accompanied by heightened irritability, it does not necessarily result in higher angry reactivity following provocation and does not result in a higher likelihood of refusing to cooperate in response to a small request. Hence, it seems that mental fatigue does not lead to less anger control and mental fatigue is not a disinhibiting factor. Conversely, based on the high compliance rates in this study it seems to be the case that people can maintain control over their anger despite being fatigued and provoked when motivation to do so is high. These conclusions implicate that

[29] Although we do agree that working memory capacity can influence self-regulation capacities (Hofmann et al., 2012), for example by preventing external and internal distractions, we don't consider working memory capacity as a limited resource that can become depleted, but rather as a fixed capacity that can lie dormant under certain situations (e.g., when motivation to perform well on a memory task is low) and can be recruited in situations when necessary (e.g., when motivation is high to suppress angry thoughts). Hence, in our view anger control may be better explained by individual differences in executive capacity rather than resulting from exhaustion of these self-control resources.

people should be more aware of the fact that mental fatigue does not necessarily results in angry or less cooperative responding, and that perhaps motivation and implicit theories about one's ability to control their anger when tired may be more determining in whether one acts out in response to provocation.

6.5. Acknowledgements

We would like to thank Alexandros Solos, Jolien van der Haar, and Melissa van Rekom for assisting in the data collection.

Chapter Seven

Summary, Discussion, and Concluding Remarks

The principal aim of this thesis was to improve our understanding of (neuro)cognitive processes involved in anger and a disposition towards anger (i.e., trait anger). The first step towards reaching this goal was to validate the Dutch version of the STAXI-2, as shown in **chapter 2**, in order to have a valid and reliable instrument to measure state and trait anger for the following experiments. The next step was to replicate and extend knowledge about neurocognitive processes involved in trait anger. More specifically, in **chapter 3 and 4**, we provided information about neural and behavioral indicators of cognitive control (i.e., response inhibition and error-processing) in relation to trait anger under conditions where anger was primed. Finally, the last step was to further our knowledge about the impact of the cognitive processes on anger and their interrelations using experimental designs. In **chapter 5**, we investigated the impact of angry rumination on inhibitory control and anger. In **chapter 6**, we examined the impact of a mentally fatiguing task on anger and anger control following provocation by the experimenter. The current chapter provides a summary and discussion of the main findings described in chapters 2 to 6 of this thesis.

7.1. Assessing anger: The validity of the STAXI-2

Given that anger may bring about substantial costs to individuals and society at large, it is of vital importance to enquire about anger difficulties. Especially in clinical settings, where anger is highly prevalent and most often manifests itself in dysfunctional forms (e.g., McDermut, Fuller, DiGiuseppe, Chelminski, & Zimmerman, 2009), a proper anger assessment is critical. This has spurred many scholars to develop anger assessment tools; mostly in the form of self-report given the subjective nature of anger (for reviews, see Eckhardt, Norlander, & Deffenbacher, 2004; Fernandez, Day, & Boyle, 2015).

The STAXI-2 is one of the most often used self-report instruments for measuring anger worldwide, as it is considered to have strong psychometric properties and relies on a strong theoretical background. For instance, in their critical review, Eckhardt, Norlander, and Deffenbacher (2004) conclude that “The STAXI-2 is based upon a solid conceptual model and possesses strong psychometric properties across a wide variety of normative groups, thus making it an excellent choice for researchers as well as clinicians”. At the start of this project,

however, the Dutch version of the STAXI-2 was lacking. Moreover, little was known about differences in anger scores measured with the STAXI-2 between several groups of clinical populations and non-clinical populations. This information is important as it can bring to light differences in how clinical populations experience, express and control anger, thereby informing were upon anger treatments should focus. Therefore, the study described in **chapter 2**, further examined the validity of the STAXI-2 and compared anger scores between several clinical and non-clinical samples. Importantly, results confirmed that the STAXI-2 is a reliable, valid instrument for assessing the experience, expression, and control of anger. That is, the STAXI-2 was found to be reliable both in terms of internal consistency and test-retest reliability. Moreover, concurrent validity was supported by meaningful correlations between the STAXI-2 scales and anger-related constructs in both patient and non-clinical samples as well as by meaningful intrascale relationships. Additionally, a factor structure close to Spielberger's (1999) original conceptualization was found supporting both construct and cross-cultural validity. In terms of criterion validity, we demonstrated that patients were inclined to experience and express anger more frequently than people from the general population. Most importantly, we also found that forensic outpatients with addiction problems were more inclined to express their anger outwardly than general psychiatric patients, although they did not differ in their anger experience or self-reported control over anger. These results imply that how forensic patients deal with their anger seems more important than the frequency at which their anger occurs, and that treatments should especially focus on educating forensic patients to deal with their anger in more constructive, non-expressive ways. All in all, findings in this study indicate that the STAXI-2, including the Dutch version, is a valuable, empirically anchored assessment tool for clinicians and researchers who want to conduct an anger assessment. A point that is further stressed by chapter 3 and 4 of this dissertation by showing meaningful relationships between cognitive processes and the trait anger scale of the STAXI-2. Future studies are needed to explore the predictive validity of the STAXI-2 in terms of sensitivity and specificity. Moreover, more insight can be gained by including samples of patients primarily seeking treatment for anger problems (including covert anger problems).

Although the STAXI-2 is in general an empirically sound instrument for measuring anger, the STAXI-2 is not free from limitations. One of the main critics is that the STAXI-2 measures anger as a personality construct as opposed to a construct of clinical and diagnostic utility (Eckhardt, Norlander, & Deffenbacher, 2004). Related to that, the STAXI-2 is criticized to have a limited ability to distinguish between subtypes of dysfunctional angry clients (Digiuseppe & Tafrate, 2004). One counterargument to this proposition is that the STAXI-2 does have clinical utility, as differences between clinical and non-clinical samples were observed in our study using the STAXI-2. A second issue is that the STAXI-2 well informs about the experiential and behavioral components of anger, but provides insufficient information about cognitive (e.g., rumination, resentment) and motivational components (e.g., coercion, revenge). A third issue is that, like any other self-report measure of anger, the STAXI-2 provides good insight in subjective feeling of anger, but only provides an indirect assessment of other domains (e.g., the behavioral component of anger). As such, anger assessment using the STAXI-2 is prone to motivational and response distortions (e.g., social desirability response bias; McEwan, Davis, MacKenzie, & Mullen, 2009). Lastly, the STAXI-2 dichotomizes between state and trait anger, whereas in reality many different forms of angry affect may exist laying somewhere in-between these two extremes, such as angry mood states. Perhaps researchers and clinicians in the field of anger may improve their anger assessment by adding concurrent anger measures in addition to the STAXI-2, such as anger logs, multiple self-report anger questionnaires, and observational measures or staff ratings (for reviews, see Eckhardt et al., 2004; Fernandez et al., 2015) in order to capture the multidimensionality of the construct properly.

In short, results of chapter 2 revealed that the Dutch version of the STAXI-2 is a reliable, valid instrument for measuring the experience, expression, and control of anger and therefore can be meaningfully used to asses anger and anger regulation strategies in clinical and non-clinical samples.

7.2. Cognitive Control in Relation to Trait Anger

Cognitive control (cf. effortful control) refers to the ability to flexibly, voluntarily, and adaptively coordinate behavior in the service of goal-directed behavior (Luna, Garver, Urban, Lazar, & Sweeney, 2004). Cognitive control is presumed to be underlain by a set of separate but interacting functions, including (but not restricted to) response inhibition and action monitoring (Luna et al., 2004). For instance, response inhibition is needed in order to suppress automatic, inappropriate, goal-incompatible behavior, whereas action monitoring is required to monitor and evaluate ongoing behavior and to prevent future mistakes (Luijten et al., 2014). Studies into cognitive control have increasingly added neurobiological techniques (e.g., event-related potentials and functional magnetic resonance imaging) to gain more insight about the underlying neural networks. These studies have generally revealed that the subcomponents of cognitive control seem to have their specific but related neural networks (Luijten et al., 2014; Luna et al., 2004; Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004).

Cognitive control takes a central place in theoretical models of trait anger and reactive aggression, whereby cognitive control serves as a inhibiting factor of angry and aggressive feelings and behavior (Anderson & Bushman, 2002; Finkel, 2007; Slotter & Finkel, 2011; Wilkowski & Robinson, 2010). The reasoning is generally as follows: given the adverse outcomes associated with displays of anger and aggression, healthy individuals are usually motivated to regulate and control the experience and expression of anger (Stearns & Stearns, 1989; Tice & Baumeister, 1993), whereas high trait anger (HTA) individuals either lack sufficient cognitive control or allow these cognitive control processes to lay dormant in hostile situations resulting in more frequent and more aggressive anger episodes (Wilkowski & Robinson, 2008b, 2010). Indeed, several studies have demonstrated deficient inhibitory control in HTA individuals as compared to low trait anger (LTA) individuals on behavioral tasks measuring cognitive control, especially following exposure to anger-related stimuli (Wilkowski et al., 2010; Wilkowski & Robinson, 2007, 2008a; Wilkowski, 2011). Only a few EEG studies have been conducted to gain more insight in the neural mechanisms underlying the behavioral results found in HTA individuals (Fulwiler et al., 2012; Jaworska et al., 2012;

Liu et al., 2014). These studies have found somewhat conflicting results. Whereas the study of Jaworska (2012) found support for reduced attentional control and diminished response inhibition on a continuous performance task, the study of Liu (2014) could not find evidence for inhibitory control deficits on the neurophysiological level as no reduced N2 and P3 (i.e.,, two electrophysiological indices of response inhibition) amplitudes were found on NoGo trials. As Wilkowski and Robinson (2008b, 2010) postulate that HTA individuals allow cognitive control processes to lay dormant in hostile contexts in particular whereas LTA do not, inhibitory control deficits may have remained undetected given the study of Liu (2014) used an affectively neutral task. Our goal was therefore to investigate inhibitory control on a neurophysiological level in a context where anger was primed. Above that, little was known about error-processing on the neurophysiological level in relation to trait anger at the start of this project, although there were indications to expect that error-processing may be distorted in HTA individuals at the behavioral level (Robinson et al., 2012). Given the central role cognitive control takes in explaining individual differences in trait anger, better insight into the dysfunction of neural networks in HTA individuals could provide a better understanding of the problems these individuals have with controlling anger and aggression. As such, the main aim of the studies described in chapter 3 and 4, was to extend knowledge about neurocognitive control processes involved in trait anger when confronted with anger-related cues.

The main goal of the study described in **chapter 3** was to compare anger-primed inhibitory control and error-processing between HTA and LTA individuals using event related potentials (ERPs). For this purpose, an affective Go/NoGo task was developed including anger-related pictures while behavioral performance (accuracy) and ERPs (N2, P3, ERN, Pe) were recorded. An extreme groups design (i.e., highest vs lowest scoring subjects on the Trait Anger Scale of the STAXI-2) was used resulting in 45 LTA individuals and 49 HTA individuals. It was expected that HTA individuals would demonstrate reduced inhibitory control compared to LTA as reflected by reduced accuracy rates and attenuated N2 and P3 amplitudes on NoGo trials. As previous research indicates that inhibitory control deficits should become more apparent in hostile contexts for HTA individuals (2008b, 2010), we

further expected these inhibitory control deficits to be more pronounced for anger-related pictures. With respect to error-processing, we expected to find either reduced ERN or Pe amplitudes in HTA compared to LTA. Results revealed that in contrast to our expectations, HTA and LTA individuals performed equally well on the affective Go/NoGo Task. Moreover, no significant differences between groups emerged on the N2 and P3 amplitudes, whereas we did observe larger N2 and P3 across groups for NoGo trials compared to Go trials, suggesting that the affective Go/NoGo task worked as intended. Finally, we could not demonstrate more distinct deficits in inhibitory control in HTA individuals in the presence of anger-related cues. Together, these results suggest that groups did not differ regarding anger-primed inhibitory control. With regard to error-processing, results showed reduced Pe amplitudes following errors in HTA individuals compared to LTA individuals, whereas no differences were found on the ERN. These results indicate that HTA individuals are characterized by error-processing deficits during the more conscious stage of processing. Interestingly, these findings are in line with several neuroimaging studies showing attenuated activation in brain regions related to performance monitoring (i.e., the anterior cingulate cortex and orbitofrontal cortex) in dysfunctional angry populations (Blair, 2012; Coccaro et al., 2007; Davidson, 2000; Fulwiler et al., 2012). This study adds to the literature by showing a relation between error-processing deficits and trait anger in healthy samples as well.

In sum, contrary to expectation HTA individuals did not show impaired anger-primed inhibitory control on both the behavioral as well as the neurophysiological level, whereas HTA individual did show impaired conscious error-processing deficits. These error-processing deficits may explain the continuation of impulsive behavior in HTA individuals regardless of their negative consequences.

While for the study described in chapter 3 an extreme groups design was chosen, we took a continuous measure approach (i.e. correlational design) for the study reported in **chapter 4**. Moreover, instead of using HTA undergraduate students, we conducted this study in a forensic psychiatric patient sample. The main aim of this study was to investigate whether scores on trait anger in forensic psychiatric patients were associated with individual

differences in anger-primed inhibitory control and error-processing. Behavioral performance (accuracy) and ERPs (N2, P3, ERN, Pe) were recorded in 38 forensic psychiatric patients who had a medium to high risk of recidivism of offending using the same affective Go/NoGo task as was used in chapter 3. We hypothesized that higher scores on trait anger in these offenders would be inversely related to accuracy rates on NoGo trials and the N2 and P3 amplitudes on NoGo trials. Moreover, we expected that these effects would be more pronounced for anger-related pictures compared to neutral pictures. Additionally, we expected higher scores on trait anger to be accompanied by reduced ERN and Pe amplitudes. In line with expectations, we found higher scores on trait anger to be inversely related to accuracy, especially for anger-related pictures. That is, higher scores on trait anger were associated with less accurate performance on NoGo trials, especially for NoGo trials that included anger-related pictures. These findings suit well with the integrative cognitive model of trait anger and reactive aggression (Wilkowski & Robinson, 2008b, 2010). However, contrary to expectations, trait anger was not associated with the neurophysiological indices of response inhibition (N2 and P3). Currently, it seems that most reliable evidence for reduced N2 and P3 amplitudes have been found in impulsive (aggressive) offender samples (Chen et al., 2005; Guan et al., 2015; Munro et al., 2007b; Vilà-Balló et al., 2014). Our sample was quite heterogeneous (i.e. offenders with different types of underlying psychopathology) which may account for this inconsistent result. Alternatively, an explanation for this unexpected finding may be that whereas neural indicators of inhibitory control are associated with impulsive aggressive behaviors, they are not specifically related to trait anger or to planned aggressive behavior. Finally, whereas no significant relation was found between trait anger and Pe amplitudes, trait anger was significantly negatively associated with the ERN amplitudes. In other words, higher scores on trait anger in these patients were associated with reduced ERN amplitudes. These results are in line with neuroimaging studies showing diminished activation in the inferior frontal brain regions related to error monitoring (i.e., the anterior cingulate cortex and orbitofrontal cortex) in dysfunctional angry populations (Blair, 2012; Coccaro et al., 2007; Davidson, 2000; Fulwiler et al., 2012).

In sum, the findings described in chapter 4 suggest that in a forensic population trait anger is associated with reduced inhibitory control on a behavioral level and reduced automatic error-detection on a neurophysiological level, which may explain the initiation and continuation of impulsive behavior in HTA forensic patients despite its negative consequences.

When comparing the results of the two studies investigating inhibitory control and error-processing in relation to trait anger, several noteworthy findings emerge. For instance, it is remarkable that although in both studies support was found for error-processing deficits in relation to trait anger, mixed results were found as to what component drives these error-processing deficits. Whereas the study described in chapter 3 found evidence for reduced Pe amplitudes, indicating that deficits occur during more conscious stages of error-processing (Overbeek et al., 2005), the study described in chapter 4 found evidence for reduced ERN amplitudes, suggesting that deficits occur at more automatic stages of error-processing (Bernstein et al., 1995; Yeung et al., 2004). Other studies that focused on error-processing deficits in relation to externalizing psychopathology have also shown mixed results. For instance, whereas some studies found evidence for both reduced ERN and Pe amplitudes in undergraduate samples manifesting with externalizing symptomatology (e.g., Ruchsow, Spitzer, Grön, Grothe, & Kiefer, 2005) and patients samples characterized by externalizing symptomatology (Franken et al., 2007; Luijten, van Meel, et al., 2011), other studies found either evidence for reduced ERN amplitudes (Dikman & Allen, 2000; J. Hall et al., 2007; Heritage & Benning, 2012; Munro et al., 2007a; Potts et al., 2006; Vilà-Balló et al., 2014; Von Borries et al., 2010) or for reduced Pe amplitudes (Brazil et al., 2009; Chen et al., 2014; Maurer et al., 2015). These mixed results indicate that a better understanding is warranted as to what these components reflect precisely as well as to what extent these components are related to externalizing behaviour. Is the distinction between the ERN and the Pe truly a distinction between automatic or controlled processing or do these components share some overlap? Do these reduced Pe and ERN amplitudes represent markers for externalizing psychopathology in general or do they represent specific markers for only certain types of externalizing behaviour? To what extent are these error-processing deficits moderated by

other factors, such as motivational processes (Botvinick & Braver, 2015) or the presence of internalizing symptomatology (Olvet & Hajcak, 2008)? Currently, no definitive satisfactory answers can be provided to these questions, highlighting the need for further investigation. What does seem to become clear from our studies is that the reduced ERN or Pe amplitudes cannot easily be explained by a general deficit in information processing on a neurophysiological level, as in both our EEG studies no support was found for reduced N2 and P3 amplitudes.

Another noteworthy finding is that whereas we found no support for inhibitory control deficits in HTA undergraduate students (chapter 3), we did find evidence for an inverse relation between trait anger and inhibitory control in forensic psychiatric patients (chapter 4). The most obvious explanation for these mixed results would be that the forensic psychiatric patients were more characterized by impulse control problems as a result of their anger as compared to the relatively healthy successful undergraduate student sample, making it easier to find a relation between trait anger and impulse control problems in the forensic sample. In other words, even though these students may be characterized by frequent anger episodes, they may show less impulsive behaviors and may have less interference in their social and occupational functioning due to sufficient cognitive control. If so, whether trait anger serves as a clinically relevant construct and results in destructive consequences may be dependent on the presence of other moderating factors, such as cognitive control. This is in concordance with the General Aggression Model (Anderson & Bushman, 2002; DeWall et al., 2011) and the I^3 theory (Finkel, 2007; Slotter & Finkel, 2011) that consider trait anger as a variable that increases the chance of aggressive behaviors, but of which its influence can be mitigated by the presence of other inhibiting factors. Alternatively, it may be that these cognitive control deficits in our patient sample can be accounted for by the presence of co-morbid psychopathology, such as substance abuse and a criminal history (e.g., Chen, Tien, Juan, Tzeng, & Hung, 2005; Luijten, Littel, & Franken, 2011) or impulsivity in general. Lastly, it may be that cognitive control deficits are more strongly related to aggressive behavior than to anger per se.

Finally, it should be noted that while the forensic psychiatric patients scoring high on trait anger performed less well on the Go/NoGo task measuring inhibitory control, performance in general was still quite high with an average accuracy rate of 77.7% for the NoGo trials (compared to 75.6% correct for undergraduate students). More substantial differences between these patients and undergraduate students would have been expected given the central role cognitive control takes in the model of Wilkowski and Robinson (2008b, 2010). Several explanations can be provided for this somewhat unexpected finding. First, there are many indications that different measures of impulsivity and cognitive control tend to be weakly related and sometimes fail to be related to more objectively visible impulsive behavior (Dalley, Everitt, & Robbins, 2011; Miyake et al., 2000). As such, it may be that the broad construct cognitive control does serve as an important factor in explaining individual differences in trait anger, but that the affective Go/NoGo task measures only one subcomponent of it (i.e., inhibitory control), that may be less suited for explaining differences in trait anger than other subcomponents underlying cognitive control. In this dissertation it seems that error-processing deficits may be more consistently related to trait anger than inhibitory control deficits. Alternatively, it could be that inhibitory control is an important factor in explaining individual differences in trait anger, but only under certain conditions (e.g., during stress or following provocation) that were not sufficiently simulated in our lab. That is, in our studies we tried to prime anger by using anger-related pictures, whereas perhaps more pronounced effects were have been found if anger-related words were used (also see Siegle, Steinhauer, Thase, Stenger, & Carter, 2002; Verona & Bresin, 2015). One counterargument, however, is that inhibitory control deficits were more pronounced for anger-related pictures in our forensic psychiatric sample, indicating these pictures exerted some effects. Alternatively, different results may have been obtained if an anger-induction was conducted before participants performed the affective Go/NoGo task, as information processing deficits may be more likely to manifest themselves during state anger episodes in HTA individuals (Owen, 2011; Schultz et al., 2010).

To conclude, the results of chapter 3 and 4 indicate that trait anger is related to deficiencies in error-processing. These error-processing deficits may take a central role in

explaining the continuation of impulsive behaviour in HTA individuals despite its negative consequences. Additionally, results were mixed with regard to inhibitory control on a behavioural level, whereby a relation between inhibitory control deficits and trait anger was only found in a clinical forensic psychiatric sample. Finally, no evidence could be found for a relation between trait anger and inhibitory control on the neurophysiological level; not even in the clinical sample. These results seem to be better explained by the I^3 theory and the GAM than the integrative cognitive model of trait anger and reactive aggression. Although there are indications that under specific conditions using specific measures results are in line the integrative cognitive model of trait anger and reactive aggression, the current data is generally not in line with this model. Clearly, more research is needed to explore the boundary conditions regarding the relation between trait anger and inhibitory control.

7.3. The impact of angry rumination and mental fatigue on anger and self-control

The second main aim of this research project was to further our knowledge about the impact cognitive processes have on anger as well as to gain knowledge about their interrelations using experimental designs. More specifically, we examined the impact of angry rumination on inhibitory control and anger in chapter 5, and the impact of a mentally fatiguing task on anger and anger control following provocation in chapter 6. In both these experiments self-control took a central place. As such, before elaborating on the results of these experiments, we will shortly recapitulate the theoretical background of the construct self-control.

Thus far we have discussed cognitive control as a stable, fixed, and trait-like capacity on which individuals can differ, whereby some individuals are proposed to have high cognitive control capacities in all sorts of tempting or anger-provoking situations, whereas others seem to lack these capacities. A different way to approach cognitive control is to view it as a state-dependent process (Baumeister et al., 2007; Hofmann et al., 2012), whereby the ability to control oneself varies over time. For instance, individuals with a high degree of cognitive control in general may fail to hold their horses in some situations (e.g., a usually calm professor that suddenly snaps during an argument with a student). Currently, it remains a puzzle why people respond to some provocations with controlled anger but

explode with aggression at others. According to the Strength Model of Self-Control (Baumeister et al., 2007), these temporary reductions in self-control can be explained by previous attempts to exert self-control causing the depletion of self-control resources from a limited reservoir of self-control. For example, suppressing angry expressions towards a colleague at work during the day may deplete self-control resources, thereby reducing self-control resources left and increasing the likelihood of expressing anger towards a significant other during the evening.

In extension to the Strength model of Self-Control, the multiple systems model of angry rumination predicts that angry rumination may result in reduced self-control explaining rumination-induced aggression (Denson, 2013). That is, angry rumination should result in reduced self-control as a result of ego depletion following attempts to (a) reduce anger intensity, (b) inhibit angry thoughts, and (c) to inhibit aggressive inclinations, subsequently heightening the likelihood of aggressive behaviors. Note here that, while the integrative cognitive model of trait anger and reactive aggression suggests a unidirectional relation between cognitive control and angry rumination, whereby it is presumed that individuals with sufficient cognitive control can more easily distract themselves from ruminative thoughts, the multiple systems model of angry rumination suggests that the relation between angry rumination and self-control may be reciprocal. Direct investigation of this hypothesis, however, was scarce at the start of this project. To our knowledge, the only direct investigation of the impact on angry rumination on self-control was a series of studies conducted by Denson, Pedersen, Friese, Hahm, and Roberts (2011). In one of these experiments, these authors showed that angry rumination following provocation resulted in decreased self-control, relative to distraction, as measured via self-report (i.e., the State Self-Control Capacity Scale). In a second experiment they found indirect support for the role self-control plays in rumination-induced aggression, by showing that glucose consumption, which is proposed to replenish the ability to exert self-control (Gailliot et al., 2007), improved performance on a Stroop task relative to a placebo drink following angry rumination but not following distraction. A limitation of these studies, however, is that self-control was rather subjectively and indirectly measured, leaving the question whether angry rumination indeed

results in lower behavioral self-control. As such, the goal of the study described in **chapter 5** was to advance our knowledge about the impact of angry rumination on self-control by using a more direct indicator of self-control (i.e. behavior on the affective Go/NoGo task).

For the study, described in chapter 5, it was expected that angry rumination would augment anger and would lead to reduced self-control as reflected by higher self-reported anger and more commission errors on NoGo trials relative to distraction. First, participants were instructed to recall and imagine an autobiographical memory in which they became very angry in order to induce anger. Both before and after this anger-induction participants indicated their affective state. Next, participants either received an angry rumination (*n* = 37) or distraction (*n* = 35) manipulation. Participants in the angry rumination condition were instructed to recall the autobiographical memory and to focus upon the emotional aspects of this event. Participants in the distraction condition were instructed to focus on how campus looks like in their mind's eye (e.g., think about the campus routes you normally walk). Finally, all participants indicated their affective state once more and completed the affective Go/NoGo task. In line with previous research (Bushman, 2002; Denson, Moulds, et al., 2012; Denson, 2013; Fabiansson et al., 2012; Konecni, 1974; Mischkowski et al., 2012; Ray et al., 2008; Rusting & Nolen-Hoeksema, 1998), results revealed that provocation-focused angry rumination resulted in increased anger, whereas distraction resulted in decreased anger. However, contrary to expectations, both groups made a comparable number of errors on NoGo trials, indicating that self-control was not differentially affected by rumination and distraction manipulations.

The most forward explanation for the findings described in chapter 5, would be that angry rumination amplifies anger but has no impact on self-control; at least not as measured with inhibitory control. This finding may not be as surprising as initially thought given that accumulating research questions the existence of the ego depletion effect on which the multiple systems model of angry rumination is based. Some argue, for instance, that motivational processes better account for reductions in self-control rather than limited resources of self-control due to previous acts of self-control (Inzlicht & Schmeichel, 2012). In line with this motivational account as opposed to the ego depletion account, a recent review

by Botvinick and Braver (2015) highlights that ego depletion "involves changes in activity both in reward networks and in centers that have been linked with control costs" (p. 100). Others argue that the ego depletion effect is overestimated due to publication bias, and may not even be a real phenomenon warranting explanation (Carter et al., 2015). If so, then how could we explain the increased likelihood of aggressive behaviors following angry rumination? In line with the GAM, we suggest that the increased chance of aggressive behavior is better explained by the heightened accessibility of aggressive cognition, feelings, and arousal (Pedersen et al., 2011). In a somewhat similar vein, DiGiuseppe and Tafrate (2010) argue that with an increased number of aggressive urges due to angry feelings caused by angry rumination, chances of aggressive behavior become higher with each instance one ruminates about the anger provoking situation.

Alternatively, we could argue that angry rumination does have an impact on cognitive control, and that our study helps in identifying moderators of this relation, as our approach differed from the experiments conducted by Denson et al. (2011), potentially explaining our deviant results. For instance, as described in chapter 5, it may be that whereas perceived self-control may be affected by angry rumination, actual self-control abilities are not. This explanation would be in line with motivational accounts of self-control (Inzlicht & Schmeichel, 2012), as this explanation suggests that people may no longer feel able or motivated to control themselves causing them to exercise less self-control than they could rather than that they are actually no longer able to control themselves. As has been noted before, it may also be that not inhibitory control over motor responses, but other aspects underlying self-control may be affected by angry rumination, such as the cognitive restraint of aggression or the modulation of the emotion anger. Finally, it may be that the impact angry rumination has on self-control may differ as a result of how angry rumination was initiated. That is, it could be that angry rumination on past angering events has less strong effects on self-control than angry rumination caused by a recent actual provocation that is still fresh in the mind.

In short, the results described in chapter 5, suggest that whereas provocation-focused angry rumination amplifies anger, it does not result in reduced inhibitory control as

measured with an affective Go/NoGo task. As of yet, we could provide little support for a reciprocal relation between angry rumination and self-control. Future research is needed to explore the boundary conditions with regards to the role of self-control in understanding rumination-induced aggression.

As noted above, heavily debate exists as to what extent self-control failures can be explained for by ego depletion. Other potential candidates moderating the effects observed in ego depletion experiments may be motivational processes and a general state of mental fatigue. More specifically, an important question is whether effects of ego depletion manipulations on aggressive behavior are caused specifically by engaging in acts of self-control or may also be caused by a more general state of mental fatigue. In **chapter 6**, we therefore investigated the impact of a mentally fatiguing task (i.e. 2-back task), unrelated to ego depletion, on anger and anger control (cf. refusal to cooperate) following provocation by the experimenter. It was expected that mentally fatigued participants would experience more intense anger and would more likely refuse to cooperate with experimenter following provocation compared to non-fatigued individuals. First, baseline mood was measured after which participant were either instructed to perform a mentally fatiguing 2-back task or to watch a neutral documentary. Next, all participants performed a frustrating task while being harassed by the experimenter. Finally, when the participants believed the experiment to be finished, the experimenter asked participants for their cooperation with a small request. In between these manipulations mood measures were conducted. Results showed that participants were more fatigued following the 2-back task as compared to watching the documentary, and that all participants were angrier following the anger-induction than at baseline, indicating that the manipulations led to the expected outcomes. However, contrary to our expectations, the mentally fatigued participants did not experience more anger following the provocation than the non-fatigued participants. More importantly, a high number of participants cooperated with the experimenters' request (>80%) regardless of the condition they were in. These findings implicate that performing a mentally fatiguing task does not necessarily result in higher angry reactivity and lower anger control in reaction to provocation. It rather seems to be the case that even under stressing conditions (mental

fatigue combined with provocation) people are quite capable of regulating their anger when their motivation to do so is high.

To summarize, the results described in chapter 6 implicate that mental fatigue does not necessarily result in angry or less cooperative responding, and hence does not serve as a disinhibiting factor under conditions where individuals are highly motivated to contain themselves. These results are well in line with diary studies highlighting that even though people oftentimes experience the inclination to hurt someone when experiencing anger, they are highly capable of controlling their anger (e.g., Averill, 1983).

Finally, when taking the results from chapter 5 and 6 together, we can conclude that while angry rumination and mental fatigue do seem to be accompanied by heightened anger, these constructs cannot easily explain for state self-control reductions (as found in ego depletion experiments). As such, in the current dissertation we could not provide empirical evidence for interrelations between the cognitive processes implicated in trait anger and reactive aggression.

7.4. Limitations of the described studies and some theoretical considerations

Several limitations should be borne in mind. First, whether the neurocognitive aspects related to trait anger represent vulnerability factors for or epiphenomena of trait anger remains uncertain, as the studies presented in chapter 3 and 4 had an observational design and no causal inferences can be drawn from these studies. A third unknown variable could be responsible for the relationships found in the current dissertation as well. This could very well be the case with regard to the effects observed in chapter 4, as we examined a relatively heterogeneous offender sample. For instance, trait anger may be part of a broader spectrum of externalizing disorders, whereby the ERN represents a general vulnerability for this broader externalizing spectrum (Olvet & Hajcak, 2008). High levels of this psychological construct may, for example, indicate the presence of disruptive behaviour disorders, such as oppositional defiant and conduct disorder. Unfortunately, experimental studies can only inform about the causal impact of these neurocognitive processes on state anger, but cannot provide information about cause and effect regarding trait anger. Hence, longitudinal

research should be performed to clarify the role of these neurocognitive aspects in developing trait anger. A second limitation has to do with the anger-related Go/NoGo task used to measure inhibitory control in this dissertation. As participants always received the neutral blocks first, diminished performance on anger-related trials may be due to stimulus-order effects. Although we deliberately chose to (a) used a blocked design as previous studies indicate blocked designs elicit more interference and (b) to present the neutral blocks first in order to prevent carry-over effects, different results may have been obtained using a random design (whereby picture type had been varied trial-by-trial) or a counterbalanced design (i.e. alternating between with block participants receive first). Third, the amount of anger elicited may not have been sufficient to observe effects on response inhibition (chapter 3 & 4) or state self-control (chapter 5 & 6). It may be that intense feelings of anger are necessary in order for individuals to lose control (over their anger) (Potegal & Qiu, 2010). However, we would like to note that we did observe some effects of our anger-primed pictures in both EEG studies and that we used validated anger inductions (Lobbestael et al., 2008) for the studies described in chapter 5 and 6 of which the impact on anger was comparable with that found in other studies in the field of anger and aggression (e.g., DeWall et al., 2007; Stucke & Baumeister). Another limitation is that in all the studies included in this thesis trait anger was measured using a self-report questionnaire. Self-report is dependent on sufficient self-awareness of ones behavior as well ones motivation to genuinely report this behavior. As such, replication is needed using more objective measures of trait anger. Finally, some of the studies in this dissertation lacked sufficient power to detect effects of small effect size which could account for some null-findings.

Besides these limitations, several theoretical considerations are of importance. For instance, there seems to be some confusion regarding the constructs related to cognitive control. An important question relates to what extent the concepts “cognitive control”, “effortful control”, “self-control”, “response inhibition” differ from each other, and to what extent these construct refer to the same underlying construct? Currently, these concepts seem to be used interchangeably, although there might be nuanced differences. Similarly, researchers may confuse phenomena related to mental fatigue with phenomena related to

ego depletion, obscuring theoretical progress. Finally, the question remains to what extent effects observed in subclinical angry populations (i.e. healthy undergraduate students scoring high on a personality trait) can be generalized to patient samples manifesting with anger problems. Are the differences between these subclinical and actual patient samples of quantitative or qualitative nature? For instance, a limitation of this dissertation may be that most studies were conducted in relatively young, well-educated female subjects, thereby reducing the generalizability of results. At the end of the day most acts of violence are committed by men. Hence, perhaps the (neuro)cognitive processes investigated in this thesis may be of higher importance in male samples. However, we would like to note that several reviews point out that few gender differences exist with respect to anger (Kring, 2000).

7.5. Treatment implications and suggestions for future research

The STAXI-2 turned out to be a clinically relevant assessment tool for measuring the experience, expression and control of anger. Hence, similar to the English version, the Dutch version of the STAXI-2 can be used to investigate the presence of anger and the way patients deal with their anger. One intriguing research avenue would be to compare scores on the STAXI-2 between 'undercontrolled' violent offenders and 'overcontrolled' violent offenders which are characterized by a sudden anger outburst after keeping anger in for a period of time. Moreover, the Dutch version of the STAXI-2 can be used to evaluate treatment efficacy. Third, future studies should focus on different, more objective measures of trait anger. For instance, ecological momentary assessment can be used to measure the presence of trait anger as well as the extent to which (neuro)cognitive processes underlie this disposition (R. C. Martin & Vieaux, 2013). Finally, the current thesis focused mostly on trait anger with regard to cognitive control. Future studies could also focus on cognitive control in relation to the anger expression and control scales using the STAXI-2.

The correlational and experimental studies included in this dissertation are of a relatively fundamental nature and therefore may not have immediate clinical relevance. Although it may take some time for these fundamental findings to be translated into clinical

practice, our studies do, however, inform indirectly about practical implications next to providing important theoretical implications.

For instance, EEG might prove useful as a diagnostic tool in the treatment of dysfunctional anger in the near future. Especially, the ERN and the Pe, which show excellent psychometric properties and can be measured using relatively few trials (Hofmann et al., 2012; Olvet & Hajcak, 2009; Rietdijk et al., 2014), may inform about individuals at risk for relapsing into negative behavior despite their negative consequences. Moreover, these measures may be used to predict efficacy of and dropout from anger management therapies. Previous studies, for instance, have shown that cognitive control deficits may indicate fewer capacities to recognize problematic behavior, reduced motivation for treatment as well as dropout from therapy in substance (Ersche & Sahakian, 2007; Severtson, von Thomsen, Hedden, & Latimer, 2010); similar results may be expected for HTA individuals. However, before implementing EEG as a diagnostic tool, future studies are also needed to examine the predictive validity (sensitivity and specificity) of the ERN and the Pe as predictors of trait anger and relapse of aggressive behavior.

Although EEG studies provide important information on temporal processing, these studies provide little information about spatial processing. Hence, other imaging methods, such as Functional Magnetic Resonance Imaging, Structural Magnetic Resonance Imaging, and Diffusion Tensor Imaging, need to be implemented as well in order to gain more complementary insight about the neurocognitive aspects related to trait anger. Similarly, functional connectivity patterns in HTA individuals may bring important insights regarding the connectivity of brain regions underlying neurocognitive functions (Fulwiler et al., 2012). Finally, direct training of brain regions related to cognitive control, such as the anterior cingulate cortex, inferior frontal gyrus and dorsolateral prefrontal cortex, via neurofeedback techniques, deep brain stimulation or via repetitive transmagnetic stimulation can inform about the causal relation between neurocognitive processes and trait anger. Recent studies have also shown that cognitive bias modification paradigms targeting some of the (neuro)cognitive processes investigated in these thesis, may be effective in reducing anger or aggression (Wilkowski et al., 2015).

As shown in this thesis, investigating (neuro)cognitive process related to trait anger under conditions of emotional arousal and provocation is of essential importance. Hence, future studies are needed in which state anger is elicited using validated anger inductions in order to examine the presence and influence of neurocognitive processes. Especially in jail settings, virtual reality technology may prove useful in doing so. Using this relatively new technique patients may be exposed to anger inducing situations in safe and controlled settings.

Another interesting avenue for research is to explore to which extent self-control failures with respect to anger and aggression may be explained by motivational accounts. The results from the current dissertation suggest that perceived reduced self-control may be more important in explaining aggressive behavior than actual, objectively measured self-control reductions. As such, HTA individuals should be made aware of the fact that they are still highly capable of controlling their mood states despite being in a fatigued, agitated state. By doing so, HTA individuals may feel more accountable for their actions, which may lead to higher self-control over anger and aggressive behavior in provoking situations. Moreover, it would be of interest to explore the development of the (neuro)cognitive biases themselves that were investigated in this thesis. For instance, there are indications that these biases stem from harsh treatments of parents (e.g., Pollak, Cicchetti, Hornung, & Reed, 2000). Finally, more studies are needed exploring the interrelations and combined influence of the neurocognitive processes involved in trait anger.

7.6. Main conclusions

Based on the results of the studies described in this thesis, several conclusions can be drawn. First, it can be concluded that the Dutch version of the STAXI-2 is a reliable instrument to assist the clinical practitioner or researcher in measuring the experience, expression, and control of anger. Second, trait anger seems to be related to deficits in cognitive control under both neutral and anger-inducing conditions, of which error-processing deficits seem to be most consistently related to trait anger. Reduced anger-primed inhibitory control in relation to trait anger, however, could not be consistently confirmed. Finally, the studies described in this thesis showed that although angry rumination and mental fatigue seem to be accompanied by increased anger, these processes seem to have little effect on cognitive control. Overall, the current thesis increased knowledge about the impact of neurocognitive

processes in relation to trait anger. At the same time, this thesis highlights that more research is needed in order to explore the impact of neurocognitive processes on trait anger as well as their interrelations.

References

Ahadi, S., & Rothbart, M. K. (1994). Temperament, development and the BigFive. In C. F. Halverson, D. Kohnstamm, & R. Martin (Eds.), *Development of the structure of temperament and personality from infancy to adulthood* (pp. 189–208). Hillsdale, NJ: Erlbaum.

Akhtar, N., & Bradley, E. J. (1991). Social information processing deficits of aggressive children: Present findings and implications for social skills training. *Clinical Psychology Review*, *11*(5), 621–644. http://doi.org/10.1016/0272-7358(91)90007-H

Alexopoulos, G. S., Murphy, C. F., Gunning-Dixon, F. M., Kalayam, B., Katz, R., Kanellopoulos, D., … Foxe, J. J. (2007). Event-related potentials in an emotional go/no-go task and remission of geriatric depression. *Neuroreport*, *18*(3), 217–221. http://doi.org/10.1097/WNR.0b013e328013ceda

Anderson, C. A., & Bushman, B. J. (2002). Human aggression. *Annual Review of Psychology*, *53*, 27–51. http://doi.org/10.1016/0191-8869(94)90294-1

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. http://doi.org/10.1016/j.paid.2008.09.026

Anton, S. D., & Miller, P. M. (2005). Do negative emotions predict alcohol consumption, saturated fat intake, and physical activity in older adults? *Behavior Modification*, *29*(4), 677–688. http://doi.org/10.1177/0145445503261164

Arrindell, W. A., & Ettema, J. H. M. (1986). *SCL-90: Handleiding bij een multidimensionele psychopathologie-indicator. [SCL90: Manual for a multidimensional indicator of psychopathology]*. Lisse: Swets & Zeitlinger.

Averill, J. (1983). Studies on anger and aggression: Implications for theories of emotion. *American Psychologist*, *38*(11), 1145–1160. http://doi.org/10.1037/0003-066X.38.11.1145

Barbour, K. A., Eckhardt, C. I., Davison, G. C., & Kassinove, H. (1998). The experience and expression of anger in maritally violent and maritally discordant-nonviolent men. *Behavior Therapy*, *29*(2), 173–191. http://doi.org/10.1016/S0005-7894(98)80001-4

Baron, K. G., Smith, T. W., Butner, J., Nealey-Moore, J., Hawkins, M. W., & Uchino, B. N. (2006). Hostility, anger, and marital adjustment: Concurrent and prospective associations with psychosocial vulnerability. *Journal of Behavioral Medicine*, *30*(1), 1–10. http://doi.org/10.1007/s10865-006-9086-z

Barratt, E. S., Stanford, M. S., Dowdy, L., Liebman, M. J., & Kent, T. A. (1999). Impulsive and premeditated aggression: A factor analysis of self-reported acts. *Psychiatry Research*, *86*(2), 163–173. http://doi.org/10.1016/S0165-1781(99)00024-4

Baumeister, R. F., Vohs, K. D., & Tice, D. M. (2007). The Strength Model of Self-Control. *Current Directions in Psychological Science*, *16*(6), 351–355. http://doi.org/10.1111/j.1467-8721.2007.00534.x

Beck, A. T. (2000). *Prisoners of Hate: The cognitive basis of anger, hostility, and violence*. New York: HarperCollins Publishers.

Beck, R., & Fernandez, E. (1998). Cognitive-behavioral therapy in the treatment of anger: A meta-analysis. *Cognitive Therapy and Research*, *22*(1), 63–74. http://doi.org/10.1023/a:1018763902991

Berkowitz, L. (1990). On the formation and regulation of anger and aggression. A cognitive-

neoassociationistic analysis. *The American Psychologist*, *45*(4), 494–503. http://doi.org/10.1037/0003-066X.45.4.494

Berkowitz, L. (2010). Appraisals and anger: How complete are the usual appraisal accounts of anger? In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 267–286). New York, NY: Springer New York.

Berkowitz, L. (2012). A different view of anger: The cognitive-neoassociation conception of the relation of anger to aggression. *Aggressive Behavior*, *38*(4), 322–333. http://doi.org/10.1002/ab.21432

Berkowitz, L., & Harmon-Jones, E. (2004). Toward an understanding of the determinants of anger. *Emotion*, *4*(2), 107–130. http://doi.org/10.1037/1528-3542.4.2.107

Bernstein, P. S., Scheffers, M. K., & Coles, M. G. H. (1995). "Where did I go wrong?" A psychophysiological analysis of error detection. *Journal of Experimental Psychology: Human Perception and Performance*, *21*(6), 1312–1322. http://doi.org/10.1037/0096-1523.21.6.1312

Bettencourt, B. A., Talley, A., Benjamin, A. J., & Valentine, J. (2006). Personality and aggressive behavior under provoking and neutral conditions: A meta-analytic review. *Psychological Bulletin*, *132*(5), 751–777. http://doi.org/10.1037/0033-2909.132.5.751

Blair, R. (2012). Considering anger from a cognitive neuroscience perspective. *Wiley Interdisciplinary Reviews: Cognitive Science*, *3*(1), 65–74. http://doi.org/10.1002/wcs.154

Botvinick, M. M., & Braver, T. S. (2015). Motivation and cognitive control: From behavior to neural mechanism. *Annual Review of Psychology*, *66*(1), 83–113. http://doi.org/10.1146/annurev-psych-010814-015044

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological Review*, *108*(3), 624–652. http://doi.org/10.1037/0033-295X.108.3.624

Brazil, I. A., de Bruijn, E. R. A., Bulten, B. H., von Borries, A. K. L., van Lankveld, J. J. D. M., Buitelaar, J. K., & Verkes, R. J. (2009). Early and late components of error monitoring in violent offenders with psychopathy. *Biological Psychiatry*, *65*(2), 137–143. http://doi.org/10.1016/j.biopsych.2008.08.011

Bresin, K., & Robinson, M. D. (2013). Losing control, literally: Relations between anger control, trait anger, and motor control. *Cognition & Emotion*, *27*(6), 995–1012. http://doi.org/10.1080/02699931.2012.755119

Bushman, B. J. (2002). Does venting anger feed or extinguish the flame? Catharsis, rumination, distraction, anger, and aggressive responding. *Personality and Social Psychology Bulletin*, *28*(6), 724–731. http://doi.org/10.1177/0146167202289002

Bushman, B. J., Bonacci, A. M., Pedersen, W. C., Vasquez, E. A., & Miller, N. (2005). Chewing on it can chew you up: Effects of rumination on triggered displaced aggression. *Journal of Personality and Social Psychology*, *88*(6), 969–983. http://doi.org/10.1037/0022-3514.88.6.969

Buss, A. H. (1966). Instrumentality of aggression, feedback, and frustration as determinants of physical aggression. *Journal of Personality and Social Psychology*, *3*(2), 153–162. http://doi.org/10.1037/h0022826

Buss, A. H., & Perry, M. (1992). The Aggression Questionnaire. *Journal of Personality and Social Psychology*, *63*(3), 452–459. http://doi.org/10.1037/0022-3514.63.3.452

Carter, E. C., Kofler, L. M., Forster, D. E., & McCullough, M. E. (2015). A series of meta-analytic tests of the depletion effect: Self-control does not seem to rely on a limited resource. *Journal of Experimental Psychology: General*, *144*(4), 796–815. http://doi.org/10.1037/xge0000083

Carver, C. S., & Harmon-Jones, E. (2009). Anger is an approach-related affect: Evidence and implications. *Psychological Bulletin*, *135*(2), 183–204. http://doi.org/10.1037/a0013965

Chen, C.-Y., Muggleton, N. G., & Chang, J.-R. (2014). Inefficiency of post-error adjustment in impulsive violent offenders. *Neuroreport*, *25*(13), 1024–1029. http://doi.org/10.1097/WNR.0000000000000212

Chen, C.-Y., Tien, Y.-M., Juan, C.-H., Tzeng, O. J. L., & Hung, D. L. (2005). Neural correlates of impulsive-violent behavior: An event-related potential study. *Neuroreport*, *16*(11), 1213–1216. http://doi.org/10.1097/00001756-200508010-00016

Christodoulou, C. (2005). The assessement and measurement of fatigue. In J. DeLuca (Ed.), *Fatigue as a window to the brain* (pp. 19 –36). Cambridge, MA: The MIT Press.

Clore, G. L., & Centerbar, D. B. (2004). Analyzing anger: How to make people mad. *Emotion*, *4*(2), 139–144. http://doi.org/10.1037/1528-3542.4.2.139

Coccaro, E. F., McCloskey, M. S., Fitzgerald, D. A., & Phan, K. L. (2007). Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biological Psychiatry*, *62*(2), 168–178. http://doi.org/10.1016/j.biopsych.2006.08.024

Collins, K., & Bell, R. (1997). Personality and aggression: the Dissipation-Rumination Scale. *Personality and Individual Differences*, *22*(5), 751–755. http://doi.org/10.1016/S0191-8869(96)00248-6

Costa, P. T., & McCrae, R. R. (1992). *Revised NEO Personality Inventory (NEO-PI-R) and the Five Factor Inventory (NEO-FFI): Professional Manual*. Odessa, Florida: Psychological Assessment Resources Inc.

Cullari, S. (1994). Levels of anger in psychiatric inpatients and normal subjects. *Psychological Reports*, *75*(3), 1163–1168.

Dalley, J. W., Everitt, B. J., & Robbins, T. W. (2011). Impulsivity, compulsivity, and top-down cognitive control. *Neuron*, *69*(4), 680–694. http://doi.org/10.1016/j.neuron.2011.01.020

Daniel, S. S., Goldston, D. B., Erkanli, A., Franklin, J. C., & Mayfield, A. M. (2009). Trait anger, anger expression, and suicide attempts among adolescents and young adults: a prospective study. *Journal of Clinical Child and Adolescent Psychology*, *38*(5), 661–71. http://doi.org/10.1080/15374410903103494

Davey, L., Day, A., & Howells, K. (2005). Anger, over-control and serious violent offending. *Aggression and Violent Behavior*, *10*(5), 624–635. http://doi.org/10.1016/j.avb.2004.12.002

Davidson, R. J. (2000). Dysfunction in the Neural Circuitry of Emotion Regulation--A Possible Prelude to Violence. *Science*, *289*(5479), 591–594. http://doi.org/10.1126/science.289.5479.591

De Castro, B. O., Veerman, J. W., Koops, W., Bosch, J. D., & Monshouwer, H. J. (2002). Hostile attribution of intent and aggressive behavior: A meta-analysis. *Child Development*,

73(3), 916–934.
Deffenbacher, J. L. (2011). Cognitive-behavioral conceptualization and treatment of anger. *Cognitive and Behavioral Practice*, *18*(2), 212–221. http://doi.org/10.1016/j.cbpra.2009.12.004
Deffenbacher, J. L. (2013). Cognitive-behavioral therapy for angry drivers. In E. Fernandez (Ed.), *Treatments for Anger in Specific Populations: Theory, Application, and Outcome* (pp. 15–32). New York, NY: Oxford University Press.
Deffenbacher, J. L., Dahlen, E. R., Lynch, R. S., Morris, C. D., & Gowensmith, W. N. (2000). An application of Beck's cognitive therapy to general anger reduction. *Cognitive Therapy & Research*, *24*(6), 689. http://doi.org/10.1023/A:1005539428336
Deffenbacher, J. L., Lynch, R. S., Oetting, E. R., & Yingling, D. A. (2001). Driving anger: Correlates and a test of state-trait theory. *Personality and Individual Differences*, *31*(8), 1321–1331. http://doi.org/10.1016/S0191-8869(00)00226-9
Deffenbacher, J. L., Oetting, E. R., Lynch, R. S., & Morris, C. D. (1996). The expression of anger and its consequences. *Behaviour Research and Therapy*, *34*(7), 575–590. http://doi.org/10.1016/0005-7967(96)00018-6
Deffenbacher, J. L., Oetting, E. R., Thwaites, G. A., Lynch, R. S., Baker, D. A., Stark, R. S., … Eiswerth-Cox, L. (1996). State-Trait Anger Theory and the utility of the Trait Anger Scale. *Journal of Counseling Psychology*. http://doi.org/10.1037/0022-0167.43.2.131
Del Vecchio, T., & O'Leary, K. D. (2004). Effectiveness of anger treatments for specific anger problems: A meta-analytic review. *Clinical Psychology Review*, *24*(1), 15–34. http://doi.org/10.1016/j.cpr.2003.09.006
Delaney, H. D., & Maxwell, S. E. (1981). On using analysis of covariance in repeated measures designs. *Multivariate Behavioral Research*, *16*(1), 105–123. http://doi.org/10.1207/s15327906mbr1601_6
DeLuca, J. (2005). Fatigue, cognition and mental effort. In J. DeLuca (Ed.), *Fatigue as a window to the brain* (pp. 37–57). Cambridge, MA: The MIT Press.
De-Moja, C. A., & Spielberger, C. D. (1997). Anger and drug addiction. *Psychological Reports*, *81*, 152–154.
Denny, K. G., & Siemer, M. (2012). Trait aggression is related to anger-modulated deficits in response inhibition. *Journal of Research in Personality*, *46*(4), 450–454. http://doi.org/10.1016/j.jrp.2012.04.001
Denson, T. F. (2009). Angry rumination and the self-regulation of aggression. In J. P. Forgas, R. F. Baumeister, & D. M. Tice (Eds.), *The Psychology of Self-Regulation* (pp. 233–248). New York, NY: US: Psychology Press.
Denson, T. F. (2013). The multiple systems model of angry rumination. *Personality and Social Psychology Review*, *17*(2), 103–123. http://doi.org/10.1177/1088868312467086
Denson, T. F. (2015). Four promising psychological interventions for reducing reactive aggression. *Current Opinion in Behavioral Sciences*, *3*, 136–141. http://doi.org/10.1016/j.cobeha.2015.04.003
Denson, T. F., DeWall, C. N., & Finkel, E. J. (2012). Self-Control and aggression. *Current Directions in Psychological Science*, *21*(1), 20–25. http://doi.org/10.1177/0963721411429451
Denson, T. F., Moulds, M. L., & Grisham, J. R. (2012). The effects of analytical rumination,

reappraisal, and distraction on anger experience. *Behavior Therapy*, *43*(2), 355–364. http://doi.org/10.1016/j.beth.2011.08.001

Denson, T. F., Pedersen, W. C., Friese, M., Hahm, A., & Roberts, L. (2011). Understanding impulsive aggression: Angry rumination and reduced self-control capacity are mechanisms underlying the provocation-aggression relationship. *Personality & Social Psychology Bulletin*, *37*(6), 850–862. http://doi.org/10.1177/0146167211401420

Denson, T. F., Pedersen, W. C., Ronquillo, J., & Nandy, A. S. (2009). The angry brain: neural correlates of anger, angry rumination, and aggressive personality. *Journal of Cognitive Neuroscience*, *21*(4), 734–744. http://doi.org/10.1162/jocn.2009.21051

DeWall, C. N., Anderson, C. A., & Bushman, B. J. (2011). The general aggression model: Theoretical extensions to violence. *Psychology of Violence*, *1*(3), 245–258. http://doi.org/10.1037/a0023842

DeWall, C. N., Baumeister, R. F., Stillman, T. F., & Gailliot, M. T. (2007). Violence restrained: Effects of self-regulation and its depletion on aggression. *Journal of Experimental Social Psychology*, *43*(1), 62–76. http://doi.org/10.1016/j.jesp.2005.12.005

DiGiuseppe, R., & Tafrate, R. C. (2003). Anger treatment for adults: A meta-analytic review. *Clinical Psychology: Science and Practice*, *10*(1), 70–84. http://doi.org/10.1093/clipsy/10.1.70

DiGiuseppe, R., & Tafrate, R. C. (2004). *Anger disorders scale*. North Tonawanda, NY: Multi-Health systems.

DiGiuseppe, R., & Tafrate, R. C. (2010). *Understanding anger disorders*. New York, NY: Oxford University Press.

Dikman, Z. V, & Allen, J. J. B. (2000). Error monitoring during reward and avoidance learning in high- and low-socialized individuals. *Psychophysiology*, *37*(1), 43–54. http://doi.org/doi:10.1017/S0048577200980983

Distel, M. A., Roeling, M. P., Tielbeek, J. J., van Toor, D., Derom, C. A., Trull, T. J., & Boomsma, D. I. (2012). The covariation of trait anger and borderline personality: A bivariate twin-siblings study. *Journal of Abnormal Psychology*, *121*(2), 458–466. http://doi.org/10.1037/a0026393

Dodge, K. A. (2006). Translational science in action: Hostile attributional style and the development of aggressive behavior problems. *Development and Psychopathology*, *18*(3), 791–814. http://doi.org/10.1017/S0954579406060391

Dodge, K. A., & Crick, N. R. (1990). Social information-processing bases of aggressive behavior in children. *Personality and Social Psychology Bulletin*, *16*(1), 8–22. http://doi.org/10.1177/0146167290161002

Dollard, J., Miller, N. E., Doob, L. W., Mowrer, O. H., & Sears, R. R. (1939). *Frustration and aggression*. New Haven, CT: Yale University Press.

Douglas, S. C., & Martinko, M. J. (2001). Exploring the role of individual differences in the prediction of workplace aggression. *Journal of Applied Psychology*, *86*(4), 547–559. http://doi.org/10.1037/0021-9010.86.4.547

Easdon, C., Izenberg, A., Armilio, M. L., Yu, H., & Alain, C. (2005). Alcohol consumption impairs stimulus- and error-related processing during a Go/No-Go Task. *Brain Research. Cognitive Brain Research*, *25*(3), 873–83. http://doi.org/10.1016/j.cogbrainres.2005.09.009

Eckhardt, C. I., & Cohen, D. J. (1997). Attention to anger-relevant and irrelevant stimuli following naturalistic insult. *Personality and Individual Differences*, *23*(4), 619–629. http://doi.org/10.1016/S0191-8869(97)00074-3

Eckhardt, C. I., & Deffenbacher, J. L. (1995). Diagnosis of anger disorders. In H. Kassinove (Ed.), *Anger disorders: Definition, diagnosis, and treatment* (pp. 27–48). Washington, DC: Taylor & Francis.

Eckhardt, C. I., Norlander, B., & Deffenbacher, J. L. (2004). The assessment of anger and hostility: A critical review. *Aggression and Violent Behavior*, *9*(1), 17–43. http://doi.org/10.1016/S1359-1789(02)00116-7

Ekman, P. (1999). Basic emotions. In *The handbook of cognition and emotion* (pp. 45–60). Sussex, Uk: John Wiley and Sons, Ltd.

Ellis, A. (1977). *Anger: How to live without it*. Secaucus, NJ: Citadel Press.

Epps, J., & Kendall, P. C. (1995). Hostile attributional bias in adults. *Cognitive Therapy and Research*, *19*(2), 159–178. http://doi.org/10.1007/BF02229692

Ersche, K. D., & Sahakian, B. J. (2007). The Neuropsychology of Amphetamine and Opiate Dependence: Implications for Treatment. *Neuropsychology Review*, *17*(3), 317–336. http://doi.org/10.1007/s11065-007-9033-y

Erwin, B. A., Heimberg, R. G., Schneier, F. R., & Liebowitz, M. R. (2003). Anger experience and expression in social anxiety disorder: Pretreatment profile and predictors of attrition and response to cognitive-behavioral treatment. *Behavior Therapy*, *34*(3), 331–350. http://doi.org/10.1016/S0005-7894(03)80004-7

Etzler, S. L., Rohrmann, S., & Brandt, H. (2014). Validation of the STAXI-2: A study with prison inmates. *Psychological Test and Assessment Modeling*, *56*, 178–194.

Everaert, J., Koster, E. H. W., & Derakshan, N. (2012). The combined cognitive bias hypothesis in depression. *Clinical Psychology Review*, *32*(5), 413–424. http://doi.org/10.1016/j.cpr.2012.04.003

Everaert, J., Tierens, M., Uzieblo, K., & Koster, E. H. W. (2013). The indirect effect of attention bias on memory via interpretation bias: Evidence for the combined cognitive bias hypothesis in subclinical depression. *Cognition & Emotion*, *27*(8), 1450–1459. http://doi.org/10.1080/02699931.2013.787972

Evers, A., Vliet-Mulder, J. C. Van, & Groot, C. J. (2000). *Documentatie van tests en testresearch in Nederland, deel 1 en 2. [Documentation of tests and test research in the Netherlands]*. Amsterdam/Assen: NIP/ Van Gorcum.

Fabiani, M., Gratton, G., & Coles, M. G. H. (2000). Event related brain potentials: Methods, theory and applications. In J. T. Cacioppo, L. G. Tassinary, & G. Bernston (Eds.), *Tutorials in psychophysiology: Physical, social and inferential elements* (pp. 53–84). New York: Cambridge University Press.

Fabiansson, E. C., Denson, T. F., Moulds, M. L., Grisham, J. R., & Schira, M. M. (2012). Don't look back in anger: neural correlates of reappraisal, analytical rumination, and angry rumination during recall of an anger-inducing autobiographical memory. *NeuroImage*, *59*(3), 2974–2981. http://doi.org/10.1016/j.neuroimage.2011.09.078

Falkenstein, M. (2006). Inhibition, conflict and the Nogo-N2. *Clinical Neurophysiology*, *117*(8), 1638–1640. http://doi.org/10.1016/j.clinph.2006.05.002

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in Go/Nogo tasks

and their relation to inhibition. *Acta Psychologica*, *101*(2-3), 267–291. http://doi.org/10.1016/S0001-6918(99)00008-6

Fassino, S., Abbate-Daga, G., Pierò, A., Leombruni, P., & Rovera, G. G. (2003). Dropout from brief psychotherapy within a combination treatment in bulimia nervosa: Role of personality and anger. *Psychotherapy and Psychosomatics*, *72*(4), 203–210. http://doi.org/10.1159/000070784

Fassino, S., Amianto, F., Abbate-Daga, G., & Leombruni, P. (2007). Personality and psychopathology correlates of dropout in an outpatient psychiatric service. *Panminerva Medica*, *9*(1), 7–15.

Fernandez, E. (2013). Anger dysfunction and its treatment. In E. Fernandez (Ed.), *Treatments for Anger in Specific Populations: Theory, Application, and Outcome* (pp. 1 –14). New York, NY: Oxford University Press.

Fernandez, E., Day, A., & Boyle, G. J. (2015). Measures of anger and hostility in adults. In *Measures of Personality and Social Psychological Constructs* (pp. 74–100). http://doi.org/10.1016/B978-0-12-386915-9.00004-8

Fessler, D. M. T. (2010). Madmen: An evolutionary perspective on anger and men's violent responses to transgression. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 361–384). New York, NY: Springer New York.

Finkel, E. J. (2007). Impelling and inhibiting forces in the perpetration of intimate partner violence. *Review of General Psychology*, *11*(2), 193–207. http://doi.org/10.1037/1089-2680.11.2.193

Finkel, E. J., & Campbell, W. K. (2001). Self-control and accommodation in close relationships: An interdependence analysis. *Journal of Personality and Social Psychology*, *81*(2), 263–277. http://doi.org/10.1037/0022-3514.81.2.263

Finkel, E. J., DeWall, C. N., Slotter, E. B., Oaten, M., & Foshee, V. A. (2009). Self-regulatory failure and intimate partner violence perpetration. *Journal of Personality and Social Psychology*, *97*(3), 483–499. http://doi.org/10.1037/a0015433

Fischer, A. H., & Roseman, I. J. (2007). Beat them or ban them: The characteristics and social functions of anger and contempt. *Journal of Personality and Social Psychology*, *93*(1), 103–115. http://doi.org/10.1037/0022-3514.93.1.103

Fox, A., Michie, P., Wynne, C., & Maybery, M. (2000). ERP correlates of response inhibition to elemental and configural stimuli in a negative patterning task. *Clinical Neurophysiology*, *111*, 1045–1053.

Franken, I. H. A., van Strien, J. W., Franzek, E. J., & van de Wetering, B. J. M. (2007). Error-processing deficits in patients with cocaine dependence. *Biological Psychology*, *75*(1), 45–51. http://doi.org/10.1016/j.biopsycho.2006.11.003

Frijda, N. H. (1988). The laws of emotion. *American Psychologist*, *43*(5), 349–358. http://doi.org/10.1037/0003-066X.43.5.349

Fulwiler, C. E., King, J. A., & Zhang, N. (2012). Amygdala–orbitofrontal resting-state functional connectivity is associated with trait anger. *NeuroReport*. http://doi.org/10.1097/WNR.0b013e3283551cfc

Gailliot, M. T., Baumeister, R. F., DeWall, C. N., Maner, J. K., Plant, E. A., Tice, D. M., … Schmeichel, B. J. (2007). Self-control relies on glucose as a limited energy source:

Willpower is more than a metaphor. *Journal of Personality and Social Psychology*, *92*(2), 325–336. http://doi.org/10.1037/0022-3514.92.2.325

Gratton, G., Coles, M., & Donchin, E. (1983). A new method for off-line removal of ocular artifact. *Electroencephalography and Clinical Neurophysiology*, *55*(4), 468–884.

Green, J. A., Whitney, P. G., & Gustafson, G. E. (2010). Vocal expressions of anger. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 139–156). New York, NY: Springer New York.

Grillon, C., Quispe-Escudero, D., Mathur, A., & Ernst, M. (2015). Mental fatigue impairs emotion regulation. *Emotion*, *15*(3), 383–9. http://doi.org/10.1037/emo0000058

Guan, M., Liao, Y., Ren, H., Wang, X., Yang, Q., Liu, X., & Wang, W. (2015). Impaired response inhibition in juvenile delinquents with antisocial personality characteristics: A preliminary ERP study in a Go/Nogo task. *Neuroscience Letters*, *603*, 1–5. http://doi.org/10.1016/j.neulet.2015.06.062

Hagger, M. S., Wood, C., Stiff, C., & Chatzisarantis, N. L. D. (2010). Ego depletion and the strength model of self-control: a meta-analysis. *Psychological Bulletin*, *136*(4), 495–525. http://doi.org/10.1037/a0019486

Hall, J., Bernat, E. M., & Patrick, C. (2007). Externalizing psychopathology and the error-related negativity. *Psychological Science*, *18*(4), 326–333. http://doi.org/10.1111/j.1467-9280.2007.01899.x

Hall, S. P. (2009). *Anger, rage and relationship: An empathic approach to anger management*. East Sussex: Routledge.

Harmon-Jones, E., Peterson, C. K., & Harmon-Jones, C. (2010). Anger, motivation, and asymmetrical frontal cortical activations. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 61–78). New York, NY: Springer New York.

Hazebroek, J. F., Howells, K., & Day, A. (2001). Cognitive appraisals associated with high trait anger. *Personality and Individual Differences*, *30*(1), 31–45. http://doi.org/10.1016/S0191-8869(00)00007-6

Heritage, A. J., & Benning, S. D. (2012). Impulsivity and response modulation deficits in psychopathy: Evidence from the ERN and N1. *Journal of Abnormal Psychology*, *122*(1), 215–222. http://doi.org/10.1037/a0030039

Herrmann, M. J., Römmler, J., Ehlis, A.-C., Heidrich, A., & Fallgatter, A. J. (2004). Source localization (LORETA) of the error-related-negativity (ERN/Ne) and positivity (Pe). *Brain Research. Cognitive Brain Research*, *20*(2), 294–299. http://doi.org/10.1016/j.cogbrainres.2004.02.013

Hirsch, C. R., Clark, D. M., & Mathews, A. (2006). Imagery and Interpretations in Social Phobia: Support for the Combined Cognitive Biases Hypothesis. *Behavior Therapy*, *37*(3), 223–236. http://doi.org/10.1016/j.beth.2006.02.001

Hoekstra, H. A., & De Fruyt, F. (2014). *Handleiding NEO-3 persoonlijkheidsvragenlijst [Manual NEO 3 personality questionnaire]*. Amsterdam: Hogrefe Uitgevers.

Hoekstra, H. A., Ormel, J., & De Fruyt, F. (1996). *Handleiding NEO persoonlijkheids-vragenlijsten NEO-PI-R en NEO-FFI [Manual NEO Personality Inventories: NEO-PI-R and NEO-FFI]*. Lisse: Swets and Zeitlinger.

Hofmann, W., Schmeichel, B. J., & Baddeley, A. D. (2012). Executive functions and self-regulation. *Trends in Cognitive Sciences*, *16*(3), 174–180. http://doi.org/10.1016/j.tics.2012.01.006

Hoglund, C. L., & Nicholas, K. B. (1995). Shame, guilt, and anger in college students exposed to abusive family environments. *Journal of Family Violence*, *10*(2), 141–157. http://doi.org/10.1007/BF02110597

Honk, J. Van, Tuiten, A., van den Hout, M., Putman, P., de Haan, E., & Stam, H. (2001). Selective attention to unmasked and masked threatening words: Relationships to trait anger and anxiety. *Personality and Individual Differences*, *30*(4), 711–720. http://doi.org/10.1016/S0191-8869(00)00160-4

Hopstaken, J. F., van der Linden, D., Bakker, A. B., & Kompier, M. A. J. (2015). A multifaceted investigation of the link between mental fatigue and task disengagement. *Psychophysiology*, *52*(3), 305–315. http://doi.org/10.1111/psyp.12339

Hornsveld, R. H. J. (2007). *Assessment and treatment of violent forensic psychiatric patients with a conduct or an antisocial personality disorder in the Netherlands*. Radboud Universiteit Nijmegen.

Hornsveld, R. H. J., Nijman, H. L. I., Hollin, C. R., & Kraaimaat, F. W. (2007). Development of the Observation Scale for Aggressive Behavior (OSAB) for Dutch forensic psychiatric inpatients with an antisocial personality disorder. *International Journal of Law and Psychiatry*, *30*(6), 480–491. http://doi.org/10.1016/j.ijlp.2007.09.009

Hovens, J. E., Lievaart, M., & Rodenburg, J. J. (2014). *STAXI-2: Vragenlijst over boosheid*. Amsterdam: Hogrefe.

Hubbard, J. A., Romano, L. J., McAuliffe, M. D., & Morrow, M. T. (2010). Anger and the reactive-proactive aggression distinction in childhood and adolescence. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 231–239). New York, NY: Springer New York.

Hubble, M. A., Duncan, B. L., & Miller, S. D. (2004). *The heart and soul of change: What works in therapy*. Washington, DC: American Psychological Association.

Hutcherson, C. A., & Gross, J. J. (2011). The moral emotions: A social–functionalist account of anger, disgust, and contempt. *Journal of Personality and Social Psychology*, *100*(4), 719–737. http://doi.org/10.1037/a0022408

Inzlicht, M., & Schmeichel, B. J. (2012). What is ego depletion? Toward a mechanistic revision of the Resource Model of Self-Control. *Perspectives on Psychological Science*, *7*(5), 450–463. http://doi.org/10.1177/1745691612454134

Jaworska, N., Berrigan, L., Fisher, D., Ahmed, A. G., Gray, J., Bradford, J. M., … Knott, V. (2012). A pilot study of electrocortical activity in dysfunctional anger: Decreased frontocortical activation, impaired attention control, and diminished behavioral inhibition. *Aggressive Behavior*, *38*(6), 469–80. http://doi.org/10.1002/ab.21449

Kamphuis, J., Meerlo, P., Koolhaas, J. M., & Lancel, M. (2012). Poor sleep as a potential causal factor in aggression and violence. *Sleep Medicine*, *13*(4), 327–334. http://doi.org/10.1016/j.sleep.2011.12.006

Kassinove, H., Sukhodolsky, D. G., Tsytsarev, S. V, & Solovyova, S. (1997). Self-reported anger episodes in Russia and America. *Journal of Social Behavior & Personality*, *12*, 301–324.

Kassinove, H., & Tafrate, R. C. (2002). *Anger Management: The complete treatment guidebook for practitioners*. Atascadero CA: Impact Publishers.

Kassinove, H., & Tafrate, R. C. (2006). Anger-related disorders: Basic issues, models, and diagnostic considerations. In E. L. Feindler (Ed.), *Anger-Related Disorders: A Practitioner's Guide to Comparative Treatments* (pp. 1–27). New York, NY: Springer Publishing Company.

Kassinove, H., & Tafrate, R. C. (2011). Application of a flexible, clinically driven approach for anger reduction in the case of mr. P. *Cognitive and Behavioral Practice*, *18*(2), 222–234. http://doi.org/10.1016/j.cbpra.2009.08.008

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*(5660), 1023–1026. http://doi.org/10.1126/science.1089910

Kiehl, K. A., Smith, A. M., Hare, R. D., & Liddle, P. F. (2000). An event-related potential investigation of response inhibition in schizophrenia and psychopathy. *Biological Psychiatry*, *48*(3), 210–221. http://doi.org/10.1016/S0006-3223(00)00834-9

Kline, R. B. (2005). *Principles and practice of structural equation modeling (2nd ed.)*. New York: The Guilford Press.

Koh, K. B., Kim, C. H., & Park, J. K. (2002). Predominance of anger in depressive disorders compared with anxiety disorders and somatoform disorders. *Journal of Clinical Psychiatry*, *63*(6), 486–492. http://doi.org/10.4088/JCP.v63n0604

Kok, A., Ramautar, J. R., De Ruiter, M. B., Band, G. P. H., & Ridderinkhof, K. R. (2004). ERP components associated with successful and unsuccessful stopping in a stop-signal task. *Psychophysiology*, *41*(1), 9–20. http://doi.org/10.1046/j.1469-8986.2003.00127.x

Konecni, V. J. (1974). Self-arousal, dissipation of anger, and aggression. *Personality and Social Psychology Bulletin*, *26*, 1587–1597.

Korman, L. M., Collins, J., Dutton, D., Dhayananthan, B., Littman-Sharp, N., & Skinner, W. (2008). Problem gambling and intimate partner violence. *Journal of Gambling Studies*, *24*(1), 13–23. http://doi.org/10.1007/s10899-007-9077-1

Krämer, U. M., Büttner, S., Roth, G., & Münte, T. F. (2008). Trait aggressiveness modulates neurophysiological correlates of laboratory-induced reactive aggression in humans. *Journal of Cognitive Neuroscience*, *20*(8), 1464–1477. http://doi.org/10.1162/jocn.2008.20103

Krämer, U. M., Kopyciok, R. P. J., Richter, S., Rodriguez-Fornells, A., & Münte, T. F. (2011). The role of executive functions in the control of aggressive behavior. *Frontiers in Psychology*, *2*, 152. http://doi.org/10.3389/fpsyg.2011.00152

Kring, A. M. (2000). Gender and anger. In A. H. Fischer (Ed.), *Gender and emotion* (pp. 211–231). Cambridge: Cambridge University Press. http://doi.org/10.1017/CBO9780511628191.011

Kuppens, P. (2005). Interpersonal determinants of trait anger: Low agreeableness, perceived low social esteem, and the amplifying role of the importance attached to social relationships. *Personality and Individual Differences*, *38*(1), 13–23. http://doi.org/10.1016/j.paid.2004.03.006

Kuppens, P., & Tuerlinckx, F. (2007). Personality traits predicting anger in self-, ambiguous-, and other caused unpleasant situations. *Personality and Individual Differences*, *42*(6),

1105–1115. http://doi.org/10.1016/j.paid.2006.09.011
Kuppens, P., van Mechelen, I., Smits, D. J. M., De Boeck, P., & Ceulemans, E. (2007). Individual differences in patterns of appraisal and anger experience. *Cognition and Emotion*, *21*(4), 689–713. http://doi.org/10.1080/02699930600859219
Kurzban, R., Duckworth, A., Kable, J. W., & Myers, J. (2013). An opportunity cost model of subjective effort and task performance. *The Behavioral and Brain Sciences*, *36*(6), 661–679. http://doi.org/10.1017/S0140525X12003196
Lachmund, E., DiGiuseppe, R., & Fuller, J. R. (2005). Clinicians' diagnosis of a case with anger problems. *Journal of Psychiatric Research*, *39*(4), 439–447. http://doi.org/10.1016/j.jpsychires.2004.10.009
Ladouceur, C. D., Dahl, R. E., Birmaher, B., Axelson, D. A., & Ryan, N. D. (2006). Increased error-related negativity (ERN) in childhood anxiety disorders: ERP and source localization. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *47*(10), 1073–1082. http://doi.org/10.1111/j.1469-7610.2006.01654.x
Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (2008). *International affective picture system (IAPS): Affective ratings of pictures and instruction manual. Technical Report A-8.* Gainesville; FL: University of Florida.
Lazarus, R. S. (1991). *Emotion and adaptation*. Oxford: Oxford University Press.
Leibsohn, M. T., Oetting, E. R., & Deffenbacher, J. L. (1994). Effects of trait anger on alcohol consumption and consequences. *Journal of Child & Adolescent Substance Abuse*, *3*(3), 17–32. http://doi.org/10.1300/J029v03n03_02
Lieberman, M. D. (2007). Social cognitive neuroscience: A review of core processes. *Annual Review of Psychology*, *58*, 259–289. http://doi.org/10.1146/annurev.psych.58.110405.085654
Lievaart, M., van der Veen, F. M., Huijding, J., Naeije, L., Hovens, J. E., & Franken, I. H. A. (2016). Trait anger in relation to neural and behavioral correlates of response inhibition and error-processing. *International Journal of Psychophysiology*, *99*, 40–47. http://doi.org/10.1016/j.ijpsycho.2015.12.001
Lijffijt, M., & Barratt, E. S. (2005). *Unpublished translation of the Barratt Impulsiveness Scale*.
Liu, Y., Zhan, X., Li, W., Han, H., Wang, H., Hou, J., … Wang, Y. (2014). The trait anger affects conflict inhibition: A Go/Nogo ERP study. *Frontiers in Human Neuroscience*, *8*, 1076. http://doi.org/10.3389/fnhum.2014.01076
Lobbestael, J., Arntz, A., & Wiers, R. W. (2008). How to push someone's buttons: A comparison of four anger-induction methods. *Cognition & Emotion*, *22*(2), 353–373. http://doi.org/10.1080/02699930701438285
Lorenz, K. (1966). *On Aggression*. New York: Harcourt.
Lorist, M. M., Klein, M., Nieuwenhuis, S., Jong, R., Mulder, G., & Meijman, T. F. (2000). Mental fatigue and task control: Planning and preparation. *Psychophysiology*, *37*(5), 614–625. http://doi.org/10.1111/1469-8986.3750614
Luijten, M., Littel, M., & Franken, I. H. A. (2011). Deficits in inhibitory control in smokers during a Go/NoGo task: An investigation using event-related brain potentials. *PloS One*, *6*(4), e18898. http://doi.org/10.1371/journal.pone.0018898
Luijten, M., Machielsen, M. W. J., Veltman, D. J., Hester, R., de Haan, L., & Franken, I. H. A. (2014). Systematic review of ERP and fMRI studies investigating inhibitory control and

error processing in people with substance dependence and behavioural addictions. *Journal of Psychiatry & Neuroscience*, *39*(3), 149–169. http://doi.org/10.1503/jpn.130052

Luijten, M., van Meel, C. S., & Franken, I. H. A. (2011). Diminished error processing in smokers during smoking cue exposure. *Pharmacology, Biochemistry, and Behavior*, *97*(3), 514–520. http://doi.org/10.1016/j.pbb.2010.10.012

Luna, B., Garver, K. E., Urban, T. A., Lazar, N. A., & Sweeney, J. A. (2004). Maturation of cognitive processes from late childhood to adulthood. *Child Development*, *75*(5), 1357–1372. http://doi.org/10.1111/j.1467-8624.2004.00745.x

Mahon, N. E., Yarcheski, A., & Yarcheski, T. J. (2000). Positive and negative outcomes of anger in early adolescents. *Research in Nursing & Health*, *23*(1), 17–24.

Mammen, O., Shear, K., Greeno, C., Wheeler, S., & Hughes, C. (1997). Anger attacks and treatment nonadherence in a perinatal psychiatry clinic. *Psychopharmacology Bulletin*, *33*(1), 105 –108.

Martin, L., & Tesser, A. (1996). Some ruminative thoughts. In R. S. Wyer (Ed.), *Advances in social cognition* (9th ed., pp. 1–48). Hillsdale, NJ: Erlbaum.

Martin, R. C., & Vieaux, L. E. (2013). Angry thoughts and daily emotion logs: Validity of the Angry Cognitions Scale. *Journal of Rational - Emotive and Cognitive - Behavior Therapy*, *31*, 219–230. http://doi.org/10.1007/s10942-013-0171-2

Martin, R., Wan, C. K., David, J. P., Wegner, E. L., Olson, B. D., & Watson, D. (1999). Style of anger expression: Relation to expressivity, personality, and health. *Personality and Social Psychology Bulletin*, *25*(10), 1196–1207. http://doi.org/10.1177/0146167299258002

Martin, R., Watson, D., & Wan, C. (2000). A three-factor model of trait anger: Dimensions of affect, behavior, and cognition. *Journal of Personality*, *68*(5), 869–897.

Matsumoto, D., Hee Yoo, S., & Chung, J. (2010). The expression of anger across cultures. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 125–137). New York, NY: Springer New York.

Maurer, J. M., Steele, V. R., Edwards, B. G., Bernat, E. M., Calhoun, V. D., & Kiehl, K. A. (2015). Dysfunctional error-related processing in female psychopathy. *Social Cognitive and Affective Neuroscience*, *11*(7), 1059–1068. http://doi.org/10.1093/scan/nsv070

McCrae, R. R., & Costa P.T. (2007). Brief versions of the NEO-PI-3. *Journal of Individual Differences*, *28*(3), 116.

McDermut, W., Fuller, J. R., DiGiuseppe, R., Chelminski, I., & Zimmerman, M. (2009). Trait Anger and Axis I Disorders: Implications for REBT. *Journal of Rational-Emotive & Cognitive-Behavior Therapy*, *27*(2), 121–135. http://doi.org/10.1007/s10942-009-0092-2

McEwan, T. E., Davis, M. R., MacKenzie, R., & Mullen, P. E. (2009). The effects of social desirability response bias on STAXI-2 profiles in a clinical forensic sample. *The British Journal of Clinical Psychology / the British Psychological Society*, *48*(4), 431–436. http://doi.org/10.1348/014466509X454886

McGinley, M., & Carlo, G. (2006). Two sides of the same coin? The relations between prosocial and physically aggressive behaviors. *Journal of Youth and Adolescence*, *36*(3),

337–349. http://doi.org/10.1007/s10964-006-9095-9
McNair, D. M., Lorr, M., & Droppleman, L. F. (1971). *Manual for the Profile of Mood States*. San Diego, CA: Education and Industrial Testing Service.
Meesters, C., Muris, P., Bosma, H., Schouten, E., & Beuving, S. (1996). Psychometric evaluation of the Dutch version of the Aggression Questionnaire. *Behaviour Research and Therapy*, *34*(10), 839–843. http://doi.org/10.1016/0005-7967(96)00065-4
Mellenbergh, G. J., & Wald, F. D. M. (1990). De verkorte versie van de Nederlandse vertaling van de Profile of Mood States (POMS) [The shortened version of the Dutch translation of the Profile of Mood States (POMS)]. *Nederlands Tijdschrift Voor de Psychologie*, *45*, 86–90.
Meltzer, H. (1933). Students' adjustments in anger. *The Journal of Social Psychology*, *4*(3), 285–309. http://doi.org/10.1080/00224545.1933.9919324
Milgram, S. (1974). *Obedience to authority*. Harpercollins.
Mischkowski, D., Kross, E., & Bushman, B. J. (2012). Flies on the wall are less aggressive: Self-distancing "in the heat of the moment" reduces aggressive thoughts, angry feelings and aggressive behavior. *Journal of Experimental Social Psychology*, *48*(5), 1187–1191. http://doi.org/10.1016/j.jesp.2012.03.012
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. http://doi.org/10.1006/cogp.1999.0734
Munro, G. E. S., Dywan, J., Harris, G. T., McKee, S., Unsal, A., & Segalowitz, S. J. (2007a). ERN varies with degree of psychopathy in an emotion discrimination task. *Biological Psychology*, *76*(1-2), 31–42. http://doi.org/10.1016/j.biopsycho.2007.05.004
Munro, G. E. S., Dywan, J., Harris, G. T., McKee, S., Unsal, A., & Segalowitz, S. J. (2007b). Response inhibition in psychopathy: the frontal N2 and P3. *Neuroscience Letters*, *418*(2), 149–153. http://doi.org/10.1016/j.neulet.2007.03.017
Muraven, M., & Baumeister, R. F. (2000). Self-regulation and depletion of limited resources: Does self-control resemble a muscle? *Psychological Bulletin*, *126*(2), 247–259. http://doi.org/10.1037//0033-2909.126.2.247
Muris, P., Meesters, C., Morren, M., & Moorman, L. (2004). Anger and hostility in adolescents: Relationships with self-reported attachment style and perceived parental rearing styles. *Journal of Psychosomatic Research*, *57*(3), 257–264. http://doi.org/10.1016/S0022-3999(03)00616-0
Nederlof, A. F., Muris, P., & Hovens, J. E. (2011). Threat/control-override symptoms and emotional reactions to positive symptoms as correlates of aggressive behavior in psychotic patients. *The Journal of Nervous and Mental Disease*, *199*(5), 342–347. http://doi.org/10.1097/NMD.0b013e3182175167
Nieuwenhuis, S., Yeung, N., van den Wildenberg, W. P. M., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of response conflict and trial type frequency. *Cognitive, Affective, & Behavioral Neuroscience*, *3*(1), 17–26. http://doi.org/10.3758/CABN.3.1.17
Nomellini, S., & Katz, R. C. (1983). Effects of anger control training on abusive parents. *Cognitive Therapy and Research*, *7*(1), 57–67. http://doi.org/10.1007/BF01173424

Novaco, R. W. (2010). Anger and psychopathology. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 465–497). New York, NY: Springer New York. http://doi.org/10.1007/978-0-387-89676-2

Novaco, R. W. (2011). Anger dysregulation: Driver of violent offending. *Journal of Forensic Psychiatry & Psychology*, *22*(5), 650–668. http://doi.org/10.1080/14789949.2011.617536

Novaco, R. W., & Whittington, R. (2013). Anger regulation for psychotic patients. In E. Fernandez (Ed.), *Treatments for Anger in Specific Populations: Theory, Application, and Outcome* (pp. 114 –137). New York: Oxford University Press.

Olvet, D. M., & Hajcak, G. (2008). The error-related negativity (ERN) and psychopathology: Toward an endophenotype. *Clinical Psychology Review*, *28*(8), 1343–1354. http://doi.org/10.1016/j.cpr.2008.07.003

Olvet, D. M., & Hajcak, G. (2009). The stability of error-related brain activity with increasing trials. *Psychophysiology*, *46*(5), 957–961. http://doi.org/10.1111/j.1469-8986.2009.00848.x

Overbeek, T. J. M., Nieuwenhuis, S., & Ridderinkhof, K. R. (2005). Dissociable components of error processing. *Journal of Psychophysiology*, *19*(4), 319–329. http://doi.org/10.1027/0269-8803.19.4.319

Owen, J. M. (2011). Transdiagnostic cognitive processes in high trait anger. *Clinical Psychology Review*, *31*(2), 193–202. http://doi.org/10.1016/j.cpr.2010.10.003

Panksepp, J. (1998). *Affective Neuroscience: Foundations of human and animal emotions*. Oxford: Oxford University Press.

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. http://doi.org/10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1

Pawliczek, C. M., Derntl, B., Kellermann, T., Gur, R. C., Schneider, F., & Habel, U. (2013). Anger under control: Neural correlates of frustration as a function of trait aggression. *PLoS ONE*, *8*(10), 1–10. http://doi.org/10.1371/journal.pone.0078503

Pease, C. R., & Lewis, G. J. (2015). Personality links to anger: Evidence for trait interaction and differentiation across expression style. *Personality and Individual Differences*, *74*, 159–164. http://doi.org/10.1016/j.paid.2014.10.018

Pedersen, W. C., Denson, T. F., Goss, R. J., Vasquez, E. A., Kelley, N. J., & Miller, N. (2011). The impact of rumination on aggressive thoughts, feelings, arousal, and behaviour. *The British Journal of Social Psychology*, *50*(2), 281–301. http://doi.org/10.1348/014466610X515696

Polivy, J., & Doyle, C. (1980). Laboratory induction of mood states through the reading of self-referent mood statements: Affective changes or demand characteristics? *Journal of Abnormal Psychology*, *89*(2), 286–290. http://doi.org/10.1037/0021-843X.89.2.286

Pollak, S. D., Cicchetti, D., Hornung, K., & Reed, A. (2000). Recognizing emotion in faces: Developmental effects of child abuse and neglect. *Developmental Psychology*, *36*(5), 679–688. http://doi.org/10.1037//0012-1649.36.5.679

Pontifex, M. B., Scudder, M. R., Brown, M. L., O'Leary, K. C., Wu, C.-T., Themanson, J. R., & Hillman, C. H. (2010). On the number of trials necessary for stabilization of error-related

brain activity across the life span. *Psychophysiology*, *47*, 767–773. http://doi.org/10.1111/j.1469-8986.2010.00974.x

Posternak, M. A., & Zimmerman, M. (2002). Anger and aggression in psychiatric outpatients. *Journal of Clinical Psychiatry*, *63*, 665–672.

Potegal, M., & Novaco, R. W. (2010). A brief history of anger. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 9 – 24). New York, NY: Springer New York.

Potegal, M., & Qiu, P. (2010). Anger in children's tantrums: A new quantitative behaviorally based model. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *Internation Handbook of Anger* (pp. 193 –217). New York: Springer.

Potegal, M., & Stemmler, G. (2010a). Constructing a neurology of anger. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 39–59). New York, NY: Springer New York.

Potegal, M., & Stemmler, G. (2010b). Cross-disciplinary views of anger: Consensus and controversy. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 3–7). New York, NY: Springer New York. http://doi.org/10.1007/978-0-387-89676-2

Potts, G. F., George, M. R. M., Martin, L. E., & Barratt, E. S. (2006). Reduced punishment sensitivity in neural systems of behavior monitoring in impulsive individuals. *Neuroscience Letters*, *397*(1-2), 130–134. http://doi.org/10.1016/j.neulet.2005.12.003

Putman, P., Hermans, E., & van Honk, J. (2004). Emotional stroop performance for masked angry faces: it's BAS, not BIS. *Emotion*, *4*(3), 305–311. http://doi.org/10.1037/1528-3542.4.3.305

Quinn, C. A., Rollock, D., & Vrana, S. R. (2014). A test of Spielberger's state-trait theory of anger with adolescents: Five hypotheses. *Emotion*, *14*(1), 74–84. http://doi.org/10.1037/a0034031

Ray, R. D., Ochsner, K. N., Cooper, J. C., Robertson, E. R., Gabrieli, J. D. E., & Gross, J. J. (2005). Individual differences in trait rumination and the neural systems supporting cognitive reappraisal. *Cognitive, Affective, & Behavioral Neuroscience*, *5*(2), 156–168. http://doi.org/10.3758/CABN.5.2.156

Ray, R. D., Wilhelm, F. H., & Gross, J. J. (2008). All in the mind's eye? Anger rumination and reappraisal. *Journal of Personality and Social Psychology*, *94*(1), 133–145. http://doi.org/10.1037/0022-3514.94.1.133

Reuter, M. (2010). Population and molecular genetics of anger and aggression: Current state of the art. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 27–37). New York, NY: Springer New York.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004). The role of the medial frontal cortex in cognitive control. *Science*, *306*(5695), 443–447. http://doi.org/10.1126/science.1100301

Ridderinkhof, K. R., van den Wildenberg, W. P. M., Segalowitz, S. J., & Carter, C. S. (2004).

Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition*, *56*(2), 129–140. http://doi.org/10.1016/j.bandc.2004.09.016

Rietdijk, W. J. R., Franken, I. H. A., & Thurik, A. R. (2014). Internal consistency of event-related potentials associated with cognitive control: N2/P3 and ERN/Pe. *PloS One*, *9*(7), e102672. http://doi.org/10.1371/journal.pone.0102672

Rizvi, S. L., Vogt, D. S., & Resick, P. A. (2009). Cognitive and affective predictors of treatment outcome in cognitive processing therapy and prolonged exposure for posttraumatic stress disorder. *Behaviour Research and Therapy*, *47*(9), 737–743. http://doi.org/10.1016/j.brat.2009.06.003

Roberts, B. W., Kuncel, N. R., Shiner, R., Caspi, A., & Goldberg, L. R. (2007). The power of personality: The comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. *Perspectives on Psychological Science*, *2*(4), 313–345. http://doi.org/10.1111/j.1745-6916.2007.00047.x

Robinson, M. D., Wilkowski, B. M., Meier, B. P., Moeller, S. K., & Fetterman, A. K. (2012). Counting to ten milliseconds: Low-anger, but not high-anger, individuals pause following negative evaluations. *Cognition & Emotion*, *26*(2), 261–281. http://doi.org/10.1080/02699931.2011.579088

Rohrmann, S., Hodapp, V., Schnell, K., Tibubos, A., Schwenkmezger, P., & Spielberger, C. D. (2013). *STAXI-2, Das State-Trait-Argerausdrucks-Inventar–2*. Bern: Huber.

Rollnik, J. D., Schröder, C., Rodríguez-Fornells, A., Kurzbuch, A. R., Däuper, J., Möller, J., & Münte, T. F. (2004). Functional lesions and human action monitoring: Combining repetitive transcranial magnetic stimulation and event-related brain potentials. *Clinical Neurophysiology*, *115*(1), 145–153. http://doi.org/10.1016/j.clinph.2003.05.001

Roseman, I. J. (2004). Appraisals, rather than unpleasantness or muscle movements, are the primary determinants of specific emotions. *Emotion*, *4*(2), 145–150. http://doi.org/10.1037/1528-3542.4.2.145

Rothbart, M. K., Ahadi, S., & Evans, D. (2000). Temperament and personality: origins and outcomes. *Journal of Personality and Social Psychology*, *78*(1), 122–135. http://doi.org/10.1037//0022-3514.78.1.122

Rothbart, M. K., & Bates, J. (2006). Temperament. In W. Damon & N. Eisenberg (Eds.), *Handbook of Child Psychology: Vol. 3. Social, emotional, and personality development.* (6th ed., pp. 105–176). New York, NY: Wiley.

Rubin, J. (1986). The emotion of anger: Some conceptual and theoretical issues. *Professional Psychology: Research and Practice*, *17*(2), 115–124. http://doi.org/10.1037/0735-7028.17.2.115

Ruchsow, M., Spitzer, M., Grön, G., Grothe, J., & Kiefer, M. (2005). Error processing and impulsiveness in normals: evidence from event-related potentials. *Cognitive Brain Research*, *24*(2), 317–325. http://doi.org/10.1016/j.cogbrainres.2005.02.003

Rüsch, N., Schiel, S., Corrigan, P. W., Leihener, F., Jacob, G. A., Olschewski, M., … Bohus, M. (2008). Predictors of dropout from inpatient dialectical behavior therapy among women with borderline personality disorder. *Journal of Behavior Therapy and Experimental Psychiatry*, *39*(4), 497–503. http://doi.org/10.1016/j.jbtep.2007.11.006

Russell, J. A., & Fehr, B. (1994). Fuzzy concepts in a fuzzy hierarchy: Varieties of anger.

Journal of Personality and Social Psychology, *67*(2), 186–205. http://doi.org/10.1037/0022-3514.67.2.186

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*(3), 790–803. http://doi.org/10.1037/0022-3514.74.3.790

Sanz, J., García-Vera, M. P., & Magán, I. (2010). Anger and hostility from the perspective of the Big Five personality model. *Scandinavian Journal of Psychology*, *51*(3), 262–270. http://doi.org/10.1111/j.1467-9450.2009.00771.x

Schieman, S. (2010). The sociological study of anger: Basic social patterns and contexts. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 329–347). New York, NY: Springer New York.

Schmeichel, B. J. (2007). Attention control, memory updating, and emotion regulation temporarily reduce the capacity for executive control. *Journal of Experimental Psychology: General*, *136*(2), 241–255. http://doi.org/10.1037/0096-3445.136.2.241

Schneider, K. L., Appelhans, B. M., Whited, M. C., Oleski, J., & Pagoto, S. L. (2010). Trait anxiety, but not trait anger, predisposes obese individuals to emotional eating. *Appetite*, *55*(3), 701–706. http://doi.org/10.1016/j.appet.2010.10.006

Schultz, D., Grodack, A., & Izard, C. E. (2010). State and trait anger, fear, and social information processing. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 311–325). New York, NY: Springer New York.

Severtson, S. G., von Thomsen, S., Hedden, S. L., & Latimer, W. (2010). The association between executive functioning and motivation to enter treatment among regular users of heroin and/or cocaine in Baltimore, MD. *Addictive Behaviors*, *35*(7), 717–720. http://doi.org/10.1016/j.addbeh.2010.02.012

Shopshire, M. S., & Reilly, P. (2013). Anger management treatment and substance use disorders. In E. Fernandez (Ed.), *Treatments for Anger in Specific Populations: Theory, Application, and Outcome* (pp. 74–91). New York: Oxford University Press.

Siegle, G. J., Steinhauer, S. R., Thase, M. E., Stenger, V. A., & Carter, C. S. (2002). Can't shake that feeling: Event-related fMRI assessment of sustained amygdala activity in response to emotional information in depressed individuals. *Biological Psychiatry*, *51*(9), 693–707. http://doi.org/10.1016/S0006-3223(02)01314-8

Siever, L. J. (2008). Neurobiology of aggression and violence. *American Journal of Psychiatry*, *165*(4), 429–442. http://doi.org/10.1176/appi.ajp.2008.07111774

Slotter, E. B., & Finkel, E. J. (2011). *Human aggression and violence: Causes, manifestations, and consequences.* (P. R. Shaver & M. Mikulincer, Eds.). Washington: American Psychological Association. http://doi.org/10.1037/12346-000

Sluyter, F., Keijser, J., Boomsma, D. I., van Doornen, L., van den Oord, E., & Snieder, H. (2000). Genetics of testosterone and the aggression–hostility–anger (AHA) syndrome: A study of middle–aged male twins. *Twin Research*, *3*(4), 266–276. http://doi.org/10.1375/136905200320565247

Smith, C. A., & Kirby, L. D. (2004). Appraisal as a pervasive determinant of anger. *Emotion*, *4*(2), 133–138. http://doi.org/10.1037/1528-3542.4.2.133

Smith, P., & Waterman, M. (2004). Role of Experience in Processing Bias for Aggressive Words in Forensic and Non-Forensic Populations, *30*, 105–122. http://doi.org/10.1002/ab.20001.r

Smith, T., Glazer, K., Ruiz, J., & Gallo, L. (2004). Hostility, anger, aggressiveness, and coronary heart disease: An interpersonal perspective on personality, emotion, and health. *Journal of Personality*, *72*(6), 1217 –1270.

Spielberger, C. D. (1988). *Manual for the State-Trait Anger Expression Inventory*. Odessa, FL: Psychological Assessment Resources.

Spielberger, C. D. (1999). *The State-Trait Anger Expression Inventory-2 (STAXI-2): Professional manual*. Odessa, FL: Psychological Assessment Resources, Inc.

Spielberger, C. D., Foreyt, J. P., Goodrick, G. K., & Reheiser, E. C. (1995). Personality characteristics of users of smokeless tobacco compared with cigarette smokers and non-users of tobacco products. *Personality and Individual Differences*, *19*(4), 439–448.

Spielberger, C. D., & Reheiser, E. C. (2010). The nature and measurement of anger. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 403–412). New York, NY: Oxford University Press.

Stanford, M. S., Mathias, C. W., Dougherty, D. M., Lake, S. L., Anderson, N. E., & Patton, J. H. (2009). Fifty years of the Barratt Impulsiveness Scale: An update and review. *Personality and Individual Differences*, *47*(5), 385–395. http://doi.org/10.1016/j.paid.2009.04.008

Stearns, C. Z., & Stearns, P. N. (1989). *Anger: The struggle for emotional control in America's history*. Chicago, IL: University of Chicago Press.

Stemmler, G. (2010). Somatovisceral activation during anger. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 103 –124). New York, NY: Springer New York.

Stosny, S. (1995). *Treating attachment abuse: A compassionate approach*. New York: Springer Publishing Company.

Strongman, K. T. (2003). *The psychology of emotion.* Chichester: John Wiley and Sons, Ltd.

Stucke, T. S., & Baumeister, R. F. (2006). Ego depletion and aggressive behavior: Is the inhibition of aggression a limited resource? *European Journal of Social Psychology*, *36*(1), 1–13. http://doi.org/10.1002/ejsp.285

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. http://doi.org/10.1016/S0191-8869(00)00171-9

Tafrate, R. C., Kassinove, H., & Dundin, L. (2002). Anger episodes in high- and low-trait-anger community adults. *Journal of Clinical Psychology*, *58*(12), 1573–1590. http://doi.org/10.1002/jclp.10076

Tedeschi, J. T., & Nesler, M. S. (1993). Grievances: Development and reaction. In T. B. Felson & J. T. Tedeschi (Eds.), *Aggression and Violence: Social interactionist perspectives* (pp. 115 –124). Washington, DC: American Psychological Association.

Thackray, R. I., Bailey, J. P., & Touchstone, R. M. (1979). The effect of increased monitoring load on vigilance performance using a stimulated radar display. *Ergonomics*, *22*, 529 – 539.

Tibubos, A. N., & Schnell, K. (2013). Anger makes you feel Stronger: The positive in fluence of trait anger in a real-life experiment. *Polish Psychological Bulletin*, *44*(2), 147–156.
Tice, D. M., & Baumeister, R. F. (1993). Controlling anger: Self-induced emotion change. In D. M. Wegner & J. W. Pennebaker (Eds.), *Handbook of mental control* (pp. 393–409). Englewood Cliffs, NJ: Prentice Hall.
van der Linden, D., Frese, M., & Meijman, T. F. (2003). Mental fatigue and the control of cognitive processes: effects on perseveration and planning. *Acta Psychologica*, *113*(1), 45–65. http://doi.org/10.1016/S0001-6918(02)00150-6
van Doorn, J., Zeelenberg, M., & Breugelmans, S. M. (2014). Anger and prosocial behavior. *Emotion Review*, *6*(3), 261–268. http://doi.org/10.1177/1754073914523794
van Kleef, G. A., & Côté, S. (2007). Expressing anger in conflict: When it helps and when it hurts. *The Journal of Applied Psychology*, *92*(6), 1557–1569. http://doi.org/10.1037/0021-9010.92.6.1557
Verleger, R., Paehge, T., Kolev, V., Yordanova, J., & Jaśkowski, P. (2006). On the relation of movement-related potentials to the go/no-go effect on P3. *Biological Psychology*, *73*(3), 298–313. http://doi.org/10.1016/j.biopsycho.2006.05.005
Verona, E., & Bresin, K. (2015). Aggression proneness: Transdiagnostic processes involving negative valence and cognitive systems. *International Journal of Psychophysiology*, 1–9. http://doi.org/10.1016/j.ijpsycho.2015.03.008
Vilà-Balló, A., Hdez-Lafuente, P., Rostan, C., Cunillera, T., & Rodriguez-Fornells, A. (2014). Neurophysiological correlates of error monitoring and inhibitory processing in juvenile violent offenders. *Biological Psychology*, *102*, 141–152. http://doi.org/10.1016/j.biopsycho.2014.07.021
Vocat, R., Pourtois, G., & Vuilleumier, P. (2008). Unavoidable errors: A spatio-temporal analysis of time-course and neural sources of evoked potentials associated with error processing in a speeded task. *Neuropsychologia*, *46*(10), 2545–2555. http://doi.org/10.1016/j.neuropsychologia.2008.04.006
Von Borries, A. K. L., Brazil, I. A., Bulten, B. H., Buitelaar, J. K., Verkes, R. J., & de Bruijn, E. R. A. (2010). Neural correlates of error-related learning deficits in individuals with psychopathy. *Psychological Medicine*, *40*(09), 1559–1568. http://doi.org/10.1017/S0033291709992017
Walfish, S., Massey, R., & Krone, A. (1990). Anxiety and anger among abusers of different substances. *Drug and Alcohol Dependence*, *25*(3), 253–256. http://doi.org/10.1016/0376-8716(90)90150-D
Wänke, M., & Schid, J. (1996). Rumination: When all else fails. In R. . Wyer (Ed.), *Ruminative thoughts: Advances in social cognition* (9th ed., pp. 177–187). Mahwah, NJ: Erlbaum.
Watson, D., Clark, L. a., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology*, *54*(6), 1063–1070. http://doi.org/10.1037/0022-3514.54.6.1063
White, B. A., & Turner, K. A. (2014). Anger rumination and effortful control: Mediation effects on reactive but not proactive aggression. *Personality and Individual Differences*, *56*, 186–189. http://doi.org/10.1016/j.paid.2013.08.012
Whiteman, M. C., Bedford, A., Grant, E., Fowkes, F. G. R., & Deary, I. J. (2001). The five-factor model (NEO-FFI) and The Personality Deviance Scales-Revised (PDS-R): Going around in

interpersonal circles. *Personality and Individual Differences*, *31*(2), 259–267. http://doi.org/10.1016/S0191-8869(00)00134-3

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

Whitmer, A. J., & Banich, M. T. (2010). Trait rumination and inhibitory deficits in long-term memory. *Cognition & Emotion*, *24*(1), 168–179. http://doi.org/10.1080/02699930802645762

Wicherts, J. M., & Vorst, H. C. M. (2004). Modelpassing van de Verkorte Profile of Mood States en meetinvariantie over mannen en vrouwen [Model fit of the Dutch shortened Profile of Mood States and measurement invariance over gender]. *Nederlands Tijdschrift Voor de Psychologie*, *59*, 12–21.

Wilkowski, B. M. (2011). Responding to social signals for response inhibition: A psychological process underlying trait anger. *Social Psychological and Personality Science*, *3*(1), 72–79. http://doi.org/10.1177/1948550611409246

Wilkowski, B. M., Crowe, S. E., & Ferguson, E. L. (2015). Learning to keep your cool: Reducing aggression through the experimental modification of cognitive control. *Cognition & Emotion*, *29*(2), 251–265. http://doi.org/10.1080/02699931.2014.911146

Wilkowski, B. M., & Robinson, M. D. (2007). Keeping one's cool: Trait anger, hostile thoughts, and the recruitment of limited capacity control. *Personality & Social Psychology Bulletin*, *33*(9), 1201–1213. http://doi.org/10.1177/0146167207301031

Wilkowski, B. M., & Robinson, M. D. (2008a). Guarding against hostile thoughts: Trait anger and the recruitment of cognitive control. *Emotion*, *8*(4), 578–583. http://doi.org/10.1037/1528-3542.8.4.578

Wilkowski, B. M., & Robinson, M. D. (2008b). The cognitive basis of trait anger and reactive aggression: an integrative analysis. *Personality and Social Psychology Review*, *12*(1), 3–21. http://doi.org/10.1177/1088868307309874

Wilkowski, B. M., & Robinson, M. D. (2010). The anatomy of anger: An integrative cognitive model of trait anger and reactive aggression. *Journal of Personality*, *78*(1), 9–38. http://doi.org/10.1111/j.1467-6494.2009.00607.x

Wilkowski, B. M., Robinson, M. D., & Troop-Gordon, W. (2010). How does cognitive control reduce anger and aggression? The role of conflict monitoring and forgiveness processes. *Journal of Personality and Social Psychology*, *98*(5), 830–840. http://doi.org/10.1037/a0018962

Willner, P., Jahoda, A., & Larkin, P. (2013). Management of anger in people with intellectual disabilities. In E. Fernandez (Ed.), *Treatments for Anger in Specific Populations: Theory, Application, and Outcome* (pp. 92 –113). New York: Oxford University Press.

Wingrove, J., & Bond, A. J. (2005). Correlation between trait hostility and faster reading times for sentences describing angry reactions to ambiguous situations. *Cognition & Emotion*, *19*(3), 463–472. http://doi.org/10.1080/02699930441000229

Winstok, Z. (2007). Perceptions, emotions, and behavioral decisions in conflicts that escalate to violence. *Motivation and Emotion*, *31*(2), 125–136. http://doi.org/10.1007/s11031-007-9061-1

World Medical Association. (2001). Declaration of Helsinki World Medical Association

Declaration of Helsinki. *Bulletin of the World Health Organization*, *79*(4), 373–374.
Wranik, T., & Scherer, K. R. (2010). Why do I get angry? A componential appraisal approach. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International Handbook of Anger: Constituent and Concomitant Biological, Psychological, and Social Processes* (pp. 243–266). New York, NY: Oxford University Press.
Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review*, *111*(4), 931–959. http://doi.org/10.1037/0033-295X.111.4.931
Young, S. E., Friedman, N. P., Miyake, A., Willcutt, E. G., Corley, R. P., Haberstick, B. C., & Hewitt, J. K. (2009). Behavioral disinhibition: Liability for externalizing spectrum disorders and its genetic and environmental relation to response inhibition across adolescence. *Journal of Abnormal Psychology*, *118*(1), 117–130. http://doi.org/10.1037/a0014657
Zhang, P., Roberts, R. E., Liu, Z., Meng, X., Tang, J., Sun, L., & Yu, Y. (2012). Hostility, physical aggression and trait anger as predictors for suicidal behavior in Chinese adolescents: A school-based study. *PLoS ONE*, *7*(2), e31044. http://doi.org/10.1371/journal.pone.0031044

Nederlandse samenvatting

Achtergrond, Resultaten & Conclusie

Achtergrond: Boosheid als emotionele staat en als dispositie

Boosheid wordt in het kort gedefinieerd als "*een emotionele gemoedstoestand die bestaat uit gevoelens die in intensiteit variëren van milde ergernis en irritatie tot woede en razernij, en die gepaard gaat met arousal*[30] *van het autonome zenuwstelsel*" (Spielberger, 1988, p.1; vrij vertaald) Een bredere en preciezere omschrijving van boosheid als emotionele staat wordt geboden door DiGiuseppe en Tafrate (2010, p. 21; vrij vertaald):

"*Boosheid is een subjectief ervaren emotionele toestand die gepaard gaat met hoge sympathische arousal. Deze wordt in eerste instantie opgeroepen door een waargenomen dreiging (voor iemands fysieke welbevinden, eigendommen, huidige of toekomstige bronnen, zelfbeeld, sociale status of het beeld van de groep, de sociale regels die het dagelijks leven reguleren, of comfort), maar ze kan ook aanhouden nadat de dreiging geweken is. Boosheid gaat ook gepaard met attributionele en evaluatieve cognities die de wandaden van anderen benadrukken en die de persoon tot antagonistische reacties aanzetten, zoals het blokkeren, verhinderen of aanvallen van, of wraak nemen op de bron van de waargenomen dreiging. Boosheid wordt gecommuniceerd via gezichtsuitdrukking en/of lichaamshouding, of via intonatie, aversieve uitspraken of agressief gedrag. De strategie die iemand gebruikt om boosheid te communiceren kan variëren naar gelang de sociale rol, leertheorie en omgevingsfactoren*"[31].

Boosheid, als emotionele staat, wordt gezamenlijk met blijdschap, verdriet en angst in iedere classificatie van basisemoties opgenomen (Ekman, 1999; Panksepp, 1998; Strongman, 2003), hetgeen betekent dat het een emotie betreft die vermoedelijk

[30] Arousal geeft een algemene staat van lichamelijke opwinding weer; specifieker beschrijft arousal de activatietoestand van het centrale en autonome zenuwstelsel.

[31] N.B.: individuele verschillen in de frequentie, intensiteit en duur waarmee woede wordt ervaren, ontbreekt in deze omschrijving. Ook komen biologische (predisposerende) factoren, zoals temperament (Rothbart, Ahadi, & Evans, 2000) of frontale lesies (Potegal & Stemmler, 2010a), in deze definitie gebrekkig aan bod.

aangeboren is en universeel bij alle mensen voorkomt en herkend wordt[32]. Dagboekonderzoeken tonen aan dat mensen boosheid in milde vormen vrijwel dagelijks ervaren (Averill, 1983; Kassinove, Sukhodolsky, Tsytsarev, & Solovyova, 1997; Meltzer, 1933; Schieman, 2010; Tafrate, Kassinove, & Dundin, 2002). Veel van deze boze episoden ontstaan tijdens interpersoonlijke contacten met de mensen die we aardig vinden en/of waar we van houden (Averill, 1983; Schieman, 2010). Verscheidene factoren kunnen boosheid ontlokken. Situaties die het meest vaak woede ontlokken, betreffen situaties waarin we dreiging ervaren, doelbewust pijn gedaan worden, niet gerespecteerd worden, beledigd worden, onrecht ervaren, ongewenste acties van anderen waarnemen en wanneer onze normen en waarden overschreden worden (Potegal & Novaco, 2010). Voornamelijk situaties die te voorkomen waren en waarin er doelbewust gehandeld werd, zullen boosheid ontlokken. Net als bij andere emoties, kan de intensiteit waarmee boosheid ervaren wordt op een continuüm geplaatst worden variërend van milde vormen van boosheid (bijvoorbeeld irritatie en ergernis) tot meer hevige vormen van boosheid (bijvoorbeeld kwaadheid, woede, razernij)[33]. Bovendien is boosheid, net als andere emoties, veelal van korte duur (Potegal & Qiu, 2010). Gewoonlijk houdt boosheid ongeveer zo'n vijf tot 15 minuten aan (Potegal & Qiu, 2010). Echter, boos rumineren (herkauwen)[34] en wraakgedachten kunnen de duur van boosheid behoorlijk verlengen (Novaco, 2011). Ook kan boosheid over langere tijdsperioden ervaren worden met een lage intensiteit (variërend van enkele uren tot enkele dagen); veelal spreken we dan van een boze stemming (Fernandez, 2013).

---

[32] Hoewel boosheid als een basisemotie beschouwd wordt die universeel geuit en herkend wordt, kan de manier waarop boosheid geuit wordt per cultuur verschillen (Matsumoto et al., 2010). Sommigen wetenschappers veronderstellen zelfs dat woede een geleerd script betreft dat niet eenduidig en met absolute zekerheid gedefinieerd kan worden (Russell & Fehr, 1994).

[33] In dit proefschrift wordt boosheid gezien als één construct dat kan variëren in intensiteit in plaats van onderscheid te maken tussen kwalitatief verschillende vormen van boosheid.

[34] Boos rumineren betreft het langdurig, herhaaldelijk terugdenken aan een autobiografische betekenisvolle woede-opwekkende gebeurtenis.

Net als bij andere emoties, wordt verondersteld dat boosheid een adaptieve functie heeft en ingebakken is ons neurobiologisch systeem (Panksepp, 1998). Sommige onderzoekers hangen het idee aan dat boosheid dient om sociale orde te versterken (Matsumoto et al., 2010), terwijl anderen geloven dat de belangrijkste functie van boosheid is om te voorkomen dat er wandaden tegen zichzelf gericht worden en om onderwerping aan anderen te voorkomen (Potegal & Stemmler, 2010b). Enigszins hieraan gerelateerd zijn enkele onderzoekers van mening dat boosheid voornamelijk dient om zich fysiek en geestelijk voor te bereiden op agressief gedrag (Berkowitz, 2012; Fessler, 2010).

Kort samengevat komt boosheid als emotionele staat universeel bij mensen voor en is boosheid in zekere mate functioneel en adaptief; afhankelijk van hoe het wordt geuit. Boosheid neemt echter disfunctionele vormen aan wanneer de boosheid ongepast en disproportioneel geuit wordt (i.e., te intens, te langdurig, te veelvuldig) en/of schade berokkent voor de persoon in kwestie of zijn of haar omgeving (Fernandez, 2013).

Een begrip dat nauw gerelateerd is aan disfunctionele boosheid (DiGiuseppe & Tafrate, 2004) en dat ingaat op individuele verschillen in hoe en wanneer boosheid ervaren wordt, betreft *trait anger* (Spielberger, 1999). *Trait anger*, in het Nederlands ook wel dispositie[35] tot boosheid geheten (Hovens et al., 2014), kenmerkt zich door de neiging om een verscheidenheid aan situaties als vervelend of frustrerend te ervaren en op deze situaties met boosheid te reageren. Korter omschreven, omvat trait anger de neiging om frequent, langdurig en op een intense manier boosheid te ervaren. Niet verwonderlijk is een dispositie tot woede gekoppeld aan een tal van negatieve uitkomsten. Zo blijkt trait anger geassocieerd te zijn met agressief gedrag en geweld zowel binnen het laboratorium als daarbuiten, waaronder een verhoogde kans tot moord (DiGiuseppe & Tafrate, 2010), kindermishandeling (Nomellini & Katz, 1983), huiselijk geweld (Barbour et al., 1998), woede op de weg (Deffenbacher et al., 2001; Deffenbacher, 2013) en agressief gedrag na een

[35] De term dispositie kan ook begrepen worden als ‘aanleg tot’ en wijst erop dat er sprake is van een persoonlijkheidskenmerk.

provocatie[36] (Bettencourt et al., 2006). Zo blijken mensen met een dispositie tot boosheid wel drie keer zo vaak geneigd te zijn met agressief gedrag te reageren na provocatie in vergelijking tot low trait anger individuen (Tafrate et al., 2002). Naast de associatie met agressief- en gewelddadig gedrag is trait anger ook gerelateerd aan andere nadelige gevolgen, waaronder riskant rijgedrag (Deffenbacher et al., 2001), middelenmisbruik (Leibsohn et al., 1994; Shopshire & Reilly, 2013; Spielberger et al., 1995), suïcidaal[37] gedrag (Daniel et al., 2009; Zhang et al., 2012), problemen in intieme relaties (Baron et al., 2006; Kassinove & Tafrate, 2002; Novaco, 2011), problemen op het werk (Douglas & Martinko, 2001), gezondheidsproblemen (Baron et al., 2006; T. Smith et al., 2004), verlaagd algemeen welzijn (Mahon et al., 2000), comorbide psychopathologie[38] (McDermut et al., 2009) en een verhoogde kans op negatieve behandeluitkomsten (Erwin et al., 2003; Fassino et al., 2003, 2007; Mammen et al., 1997; Rizvi et al., 2009; Rüsch et al., 2008).

Kortweg kan geconcludeerd worden dat mensen met een dispositie tot boosheid een veelvuldigheid aan negatieve consequenties ervaren indien de boosheid onbehandeld blijft. Tot op heden is echter nog onduidelijk waarom sommige mensen gemakkelijk en in toenemende mate woedend en/ of agressief worden bij relatief kleine provocaties, terwijl de meeste mensen niet snel boos te krijgen zijn en hun kalmte weten te bewaren. Derhalve is het van essentieel belang om beter inzicht te krijgen in de factoren die aan deze schadelijke persoonlijkheidstrek ten grondslag liggen.

[36] Provocatie staat voor uitlokken/ tergen.
[37] Suïcide betreft een ander woord voor zelfmoord.
[38] Comorbide psychopathologie staat voor het tegelijkertijd aanwezig zijn van meerdere psychiatrische stoornissen (mentale problemen).

Doel en relevantie van het huidige proefschrift

Het huidige proefschrift heeft als belangrijkste doel om meer inzicht te verschaffen in welke (neuro)cognitieve processen[39] een rol spelen bij (een dispositie tot) boosheid.

Een eerste belangrijke stap in die richting betrof de vertaling en validering[40] van de Nederlandse versie van de State Trait Anger Expression Inventory-2 (STAXI-2; Spielberger, 1999). In de klinische praktijk komen therapeuten veelvuldig in aanraking met boze patiënten waarbij hun boosheid zich veelal op disfunctionele wijze manifesteert (DiGiuseppe & Tafrate, 2004). Gedegen informatie over de vorm van boosheid die bij de patiënt aanwezig is (toestandsboosheid versus dispositieboosheid) en hoe de patiënt zijn boosheid reguleert en/of tot uiting laat komen, is van essentieel belang om de behandeling met succes te kunnen afronden. De STAXI-2 is wereldwijd één van de meest gebruikte meetinstrumenten om boosheid (zowel toestandsboosheid als een dispositie tot boosheid) en de uiting en regulatie hiervan in kaart te brengen. De STAXI-2 wordt beschouwd als een meetinstrument met sterke psychometrische eigenschappen[41] en een sterke wetenschappelijk onderbouwde theoretische achtergrond (Eckhardt et al., 2004; Fernandez et al., 2015). Aan het begin van het promotietraject was de Nederlandse versie van de STAXI-2 echter nog niet beschikbaar. Bovendien was er beperkt informatie beschikbaar over hoe verschillende patiëntengroepen scoren op de STAXI-2 in vergelijking tot elkaar en in vergelijking tot een niet-klinische ("gezonde") populatie. Het doel van het eerste onderzoek van dit promotietraject, zoals beschreven in hoofdstuk 2, betrof derhalve de vertaling en validering van de Nederlandse versie van de State-Trait Anger Expression Inventory-2 (STAXI-2). Als subdoel werden

[39] Aandacht, geheugen en interpretatie zijn voorbeelden van neurocognitieve processen. Verwacht wordt dat de invloed van een gebeurtenis wordt bepaald door hoe deze gebeurtenis in de hersenen verwerkt wordt.

[40] Valideren bekent ongeveer het wetenschappelijk toetsen van de kwaliteiten van een vragenlijst.

[41] Pyschometrische kwaliteiten betreffen statistische gegevens in hoeverre een meetinstrument betrouwbaar is en in welke mate het meetinstrument meet wat het behoort te meten.

daarnaast de scores van enkele klinische en niet-klinische steekproeven op deze vragenlijst met elkaar vergeleken.

Op het moment dat het promotietraject van start ging was er bovendien vrijwel geen onderzoek verricht met behulp van een electro-encephalogram (EEG)[42] bij mensen met een dispositie tot boosheid. In het bijzonder ontbrak er kennis over neurocognitieve correlaten van cognitieve controle in relatie tot een dispositie tot boosheid, terwijl verscheidene theoretische cognitieve modellen suggereren dat cognitieve controle een belangrijke rol speelt bij een dispositie tot boosheid (DeWall et al., 2011; Finkel, 2007; Wilkowski et al., 2010; Wilkowski & Robinson, 2008b). Volgens deze theorieën zou cognitieve controle individuen namelijk in staat stellen hun boze en agressieve neigingen en gedachten te onderdrukken. Cognitieve controle verwijst naar het vermogen om flexibel, vrijwillig en op een adequate manier gedrag te coördineren ten behoeve van doelgericht gedrag (Luna et al., 2004). Verschillende neurobiologische onderzoeken hebben aan de hand van neuroimaging technieken (bijv. een electro-encefalogram en functional magnetic resonance imaging [fMRI]) aangetoond dat meerdere afzonderlijke, doch op elkaar inwerkende, mentale processen van belang zijn bij cognitieve controle (Luna et al., 2004), waaronder, maar niet beperkend tot, impulscontrole en foutverwerking. Impulscontrole is bijvoorbeeld nodig om automatische, ongewenste en/of ongepaste gedragingen die niet in lijn liggen met een persoon zijn of haar lange termijn doelen te onderdrukken (Luijten et al., 2014). Foutverwerking is weer van belang om gedrag te monitoren en waar nodig bij te sturen om toekomstige fouten te verkomen (Luijten et al., 2014). Mensen met een dispositie tot boosheid zouden ofwel over onvoldoende cognitieve controle beschikken dan wel in provocerende situaties minder geneigd zijn deze cognitieve processen aan te spreken, hetgeen resulteert in frequentere en intensere boze buien. Belangrijk hierbij is dat

[42] Hersencellen communiceren met elkaar door middel van elektrische signalen. EEG is een soort hersenscan, waarbij de elektrische activiteit van de hersenen wordt gemeten. Proefpersonen krijgen een soort badmuts op met elektrodes die de elektrische activiteit van de hersencellen meet.

verondersteld wordt dat beperkingen in cognitieve controle voornamelijk zichtbaar worden in situaties waarin boze gedachten geactiveerd zijn (Wilkowski et al., 2010). Door het EEG te gebruiken in combinatie met cognitieve taken ontstaat een beter beeld van de neurocognitieve processen die betrokken zijn bij een dispositie tot woede. Bovendien hebben EEG technieken het voordeel dat zij een meer directe en zodoende objectievere maat betreffen voor cognitieve processen die betrokken zijn bij een dispositie tot boosheid. Zo zijn deze maten bijvoorbeeld minder afhankelijk van de snelheid waarmee iemand reageert (reactiesnelheid) aangezien de activiteit in de hersenen direct gemeten wordt. Meer inzicht in de neurocognitieve achtergrond van een dispositie tot boosheid kan belangrijke informatie verschaffen over welke processen aangepakt dienen te worden in de behandeling. Een tweede doel van dit proefschrift was zodoende informatie te verschaffen over neurologische en gedragsmatige indicatoren van cognitieve controle (i.e., impulscontrole en foutverwerking) in relatie tot een dispositie tot woede onder condities waarin boosheid geactiveerd werd (zie hoofdstuk 3 & 4).

Tot slot is getracht kennis te verbreden over de invloed van cognitieve processen die betrokken zijn bij boosheid als emotionele staat, alsook hoe deze cognitieve processen met elkaar gerelateerd zijn, met behulp van experimentele onderzoeken (zie hoofdstuk 5 & 6). De meeste onderzoeken hebben tot nu toe cognitieve processen (interpretatie, cognitieve controle, ruminatie) in relatie tot een dispositie tot boosheid in isolatie onderzocht. De kans is echter groot dat risicofactoren op elkaar inwerken en gezamenlijk bepalen in welke mate een persoon geneigd is boosheid te ervaren; ook wel de combined cognitive bias hypothese geheten (Everaert et al., 2012). Derhalve is in dit proefschrift onderzocht in hoeverre cognitieve processen op elkaar inwerken met betrekking tot boosheid.

Cognitieve controle is tot dusverre benadert als een stabiele, vastliggende capaciteit waarbij individuen van elkaar kunnen verschillen wat betreft de mate waarin zij tot cognitieve controle beschikken. Een andere manier om cognitieve controle te benaderen, is door cognitieve controle te zien als een proces dat door de tijd heen kan verschillen en dat afhankelijk is van de staat waarin iemand verkeert. Zo kunnen mensen die in het algemeen over veel cognitieve controle beschikken in sommige situaties hun zelfgeduld verliezen (bijv.

een professor die vlak voor een deadline ontploft van woede wanneer zijn computer vastloopt). Tot op heden is nog onvoldoende kennis beschikbaar over waarom mensen in de meeste situaties hun kalmte weten te bewaren, terwijl zij in andere situaties door het minste geringste kunnen ontploffen. Volgens het *Strenght Model of Self-Control* van Baumeister (2007) kunnen tijdelijke verminderingen in zelfcontrole verklaard worden door eerdere pogingen zelfcontrole uit te oefenen, waardoor bronnen van zelfcontrole uitgeput raken (ook wel *ego depletion* geheten). Net als bij het leeg raken van spierkracht zou het simpelweg uitoefenen van zelfcontrole paradoxaal genoeg kunnen leiden tot minder zelfcontrole. Gebaseerd op de voorgaande theorie voorspeld het *multiple systems model of angry rumination* van Denson (2013) dat boos rumineren eveneens leidt tot minder zelfcontrole over boosheid met agressief gedrag als gevolg. Boos rumineren ontstaat voornamelijk door problemen in selectieve aandacht met het loslaten van woede-gerelateerd materiaal (Wilkowski & Robinson, 2010). Praktisch gezien houdt boos rumineren in dat mensen (a) hun aandacht richten op negatieve boze buien, (b) ze woede-ervaringen uit het verleden herhaaldelijk blijven ophalen uit hun geheugen, (c) wraakfantasieën erop na houden en (d) ze continu nadenken over de oorzaken en gevolgen van de woedebuien (Sukhodolsky et al., 2001). Boos rumineren zou volgens dit model leiden tot dalingen in zelfcontrole, aangezien het inhiberen (remmen) van boze gevoelens, boze gedachten en agressief gedrag (ontstaan door boos rumineren) zelfcontrole zou kosten (Denson et al., 2011). Aan de start van het project was er echter beperkt onderzoek beschikbaar waarin werd aangetoond dat boos rumineren daadwerkelijk tot een afname in zelfcontrole leidt. Ook bestond hevig debat over de mate waarin mislukte pogingen zichzelf te controleren daadwerkelijk verklaard kunnen worden door ego depletion. Sommige onderzoekers beargumenteren bijvoorbeeld dat veranderingen in motivationele processen beter in staat zijn reducties in zelfcontrole te voorspellen dan het leeg raken van zelfcontrole bronnen (Botvinick & Braver, 2015). Andere onderzoekers veronderstellen weer dat mentale vermoeidheid een betere verklaring biedt voor het verliezen van zelfcontrole dan ego depletion (Hagger et al., 2010). Mentale vermoeidheid (geestelijke vermoeidheid) beschrijft een toestand waarin personen een afkeer hebben tegen verdere inspanning, wat tevens

gepaard gaat met veranderingen in stemming, motivatie en de verwerking van informatie (Hopstaken et al., 2015). Een praktisch voorbeeld is bijvoorbeeld wanneer een persoon zich (a) vermoeid voelt, (b) minder concentratievermogen ervaart en (c) geen zin meer heeft in het afmaken van een taak, wanneer hij of zij daarvoor een lange mentaal inspannende vergadering heeft gehad.

Een belangrijke vraag bij de experimenten uit hoofdstuk 5 & 6 betrof zodoende in welke mate cognitieve controle tijdelijk afneemt als gevolg van mentale vermoeidheid en boos rumineren. Beter begrip van de invloed van mentale vermoeidheid en boos rumineren op boosheid en de controle over boosheid kan leiden tot beter inzicht in waarom mensen soms de controle over hun boosheid verliezen en tot agressief gedrag overgaan, terwijl zij in andere situaties de rust kunnen bewaren.

Hieronder volgt een samenvatting en discussie van de hoofdbevindingen, zoals beschreven in hoofdstuk 2 tot en met 6. Eerst wordt echter kort omschreven hoe electro-encephalogram (EEG) is toegepast binnen het huidige proefschrift.

Elektro-encefalogram als methodiek

Ten doeleinde meer zicht te krijgen op de relatie tussen neurale indicatoren van cognitieve controle en een dispositie tot boosheid, is binnen dit proefschrift vooral gebruik gemaakt van EEG als neuroimaging techniek, waarbij gebruik is gemaakt van event-related potentials (ERPs). Een event-related potential houdt een door een stimulus opgeroepen patroon van positieve dan wel negatieve electrofysiologische activiteit in ten tijde van een specifieke gebeurtenis, zoals gemeten met een EEG (Fabiani et al., 2000). Aan verschillende cognitieve functies zijn verschillende ERPs verbonden. Met betrekking tot foutverwerking zijn twee ERPs van belang, te weten de Error-Related Negativity (ERN) en de Error Positivity (Pe). De ERN betreft een elektro-negatieve golf die kort (gemiddeld 50-100ms) na het maken van een fout tijdens de uitvoering van reactietijd taken[43] ontstaat, zoals een GoNoGo taak, en wordt

[43] Reactietijdtaken zijn taken waarop de proefpersonen snel een reactie dient te geven.

verondersteld foutverwerking op onbewust niveau te reflecteren (Bernstein et al., 1995; Luijten et al., 2014; Yeung et al., 2004). De Pe betreft een elektro-positieve golf die wat later optreedt na het maken van een fout (zo'n 200-300 ms) en wordt verondersteld een meer diepere, bewustere verwerking van de fout te reflecteren (Luijten et al., 2014; Overbeek et al., 2005). Met betrekking tot impulscontrole zijn de N2 en de P3 van belang. De N2 betreft een elektro-negatieve golf die omstreeks 200 milliseconden na het vertonen van een stimulus (bijvoorbeeld een plaatje of een woord) ontstaat en wordt verondersteld het detecteren van een responsconflict te reflecteren (Fox et al., 2000; Nieuwenhuis et al., 2003). De P3 betreft een positieve golf die ongeveer 300 milliseconden na het tonen van een stimulus ontstaat en wordt verondersteld een index voor stimulus evaluatietijd te reflecteren (Kok et al., 2004; Verleger et al., 2006). Verscheidene onderzoeken hebben aangetoond dat de N2 en P3 golven versterkt zijn wanneer mensen hun respons dienen te onderdrukken op reactietijdtaken (Falkenstein et al., 1999; Falkenstein, 2006; Luijten et al., 2014), zoals een GoNoGo taak of een Stop-Signal taak.

Samenvatting onderzoeksbevindingen

Uit de resultaten van **hoofdstuk 2** kwam naar voren dat de STAXI-2 een betrouwbaar en valide meetinstrument is om de ervaring, expressie en regulatie van boosheid vast te stellen. De betrouwbaarheid van de STAXI-2 bleek uit een voldoende interne consistentie (Cronbachs alpha) en test-hertest betrouwbaarheid[44]. De concurrente validiteit[45] werd eveneens ondersteund middels betekenisvolle (inter)correlaties tussen de STAXI-2 schalen en aan boosheid-gerelateerde constructen (agressie, vijandigheid, impulsiviteit) in zowel de

[44] Test-hertest betrouwbaarheid geeft de mate aan waarin een test twee keer hetzelfde resultaat geeft. Indien je een persoonlijkheidskenmerk meet, verwacht je dat de test bij een tweede afname grotendeels dezelfde resultaten geeft.

[45] Concurrente validiteit geeft de mate aan waarin de score op het meetinstrument samenhangt met de score op een ander meetinstrument dat ongeveer hetzelfde meet. Zo verwacht je dat boosheid sterker samenhangt met een meetinstrument voor agressie dan een meetinstrument die de hoeveelheid vreetbuien meet.

klinische als niet-klinische steekproeven. Tevens werd een factorstructuur[46] overeenkomend met Spielberger's (1999) originele conceptualisatie gevonden waarmee zowel de constructvaliditeit als crossculturele validiteit ondersteund werd. Met betrekking tot de criteriumvaliditeit toonde het huidige onderzoek aan dat psychiatrische patiënten vaker geneigd zijn boosheid te ervaren en te uiten in vergelijking tot niet-klinische ("gezonde") populaties. Bovenal werd gevonden dat ambulant behandelde forensische psychiatrische patiënten met verslavingsproblemen meer geneigd zijn hun boosheid fysiek en verbaal te uiten dan de overige psychiatrische patiënten, ondanks dat zij niet verschillen van de overige patiënten wat betreft de ervaring en regulatie van hun boosheid. Ofwel de forensische psychiatrische patiënten verschilden niet van de overige patiënten in de frequentie waarmee ze boosheid ervoeren en waren evenveel geneigd hun boosheid onder controle te krijgen. Deze resultaten impliceren dus dat de manier waarop forensische psychiatrische patiënten hun boosheid uiten van groter belang is dan de frequentie waarmee zij hun boosheid ervaren en dat behandelingen zich vooral moeten richten op het aanleren van meer constructieve (minder agressieve) manieren om boosheid te uiten. Concluderend kan gesteld worden dat de STAXI-2, waaronder de Nederlandse versie, een waardevol, wetenschappelijk onderbouwd meetinstrument is voor clinici en onderzoekers die boosheid in kaart willen brengen.

Het doel van het onderzoek beschreven in **hoofdstuk 3** was te onderzoeken of mensen met een dispositie tot boosheid gekenmerkt worden door een verminderde responsinhibitie en foutverwerking middels gedragsmatige en neurale indicatoren. Hierbij werd tevens gekeken of problemen met impulscontrole sterker aanwezig waren indien boosheid geactiveerd werd. Voor dit doeleinde werd gebruik gemaakt van een affectieve GoNoGo taak waarbij neutrale en aan boosheid gerelateerde plaatjes getoond werden. Een

[46] De factorstructuur geeft aan in welke mate bepaalde items (vragen) onderdeel uitmaken van een overkoepelende term. Zo verwacht je dat wanneer twee vragen over aantrekkelijkheid gaan en twee vragen over eendjes, dat de twee vragen over aantrekkelijkheid sterker met elkaar samenhangen en tot de factor 'aantrekkelijkheid' behoren, terwijl de vragen over eendjes tot de factor 'eendjes' behoren.

GoNoGo taak betreft een taak waarbij proefpersonen vrijwel continu een reactie dienen te geven door een toets in te drukken op Go trials en slechts af en toe bij de vertoning van een NoGo trial hun gedrag dienen te onderdrukken. Doordat er infrequent op onverwachtse momenten een respons onderdrukt dient te worden, maakt de taak sterk aanspraak op impulscontrole. De gedragsmatige data (accuratesse) en ERPs geassocieerd met impulscontrole (N2, P3) en foutverwerking (ERN, en Pe) werden tijdens uitvoering van deze taak gemeten. Groepen met extreme scores op de Trait Anger schaal van de STAXI-2 werden geselecteerd, resulterend in 45 mensen met een lage dispositie tot boosheid (LTA) en 49 mensen met een hoge dispositie tot boosheid (HTA). Er werd ten eerste verwacht dat HTA mensen minder goed in staat zouden zijn hun responsen te onderdrukken, wat zou blijken uit een lagere accuratesse en verlaagde N2 en P3 amplitudes op NoGo trials. Ten tweede werd verwacht dat het vermogen responsen te onderdrukken in grotere mate verminderd zou zijn op het moment dat aan boosheid gerelateerde plaatjes getoond zouden worden. Tot slot was de verwachting dat HTA mensen in mindere mate fouten zouden verwerken, hetgeen zou blijken uit verlaagde ERN en Pe amplitudes tijdens het maken van fouten. In tegenstelling tot onze verwachting presteerden beide groepen even goed op de affectieve GoNoGo taak. Evenmin vonden we groepsverschillen op de N2 en P3 golven ongeacht of er neutrale plaatjes dan wel aan boosheid gerelateerde plaatjes werden getoond. Wel werd gevonden dat de N2 en P3 amplitudes verhoogd waren op NoGo trials ten opzichte van Go trials, waaruit we kunnen opmaken dat de taak heeft gewerkt zoals beoogd. De beschreven resultaten tonen aan dat mensen met een sterke dispositie tot boosheid niet verschillen van mensen met een lage dispositie tot boosheid wat betreft impulscontrole. Met betrekking tot foutverwerking vonden we wel verlaagde Pe amlitudes na het maken van fouten bij HTA individuen in vergelijking tot LTA individuen. De groepen verschilden echter niet van elkaar op de ERN golven. Deze resultaten tonen aan dat HTA individuen gekenmerkt worden door een verminderde verwerking van fouten tijdens de meer bewuste fase van foutverwerking en een minder diepe verwerking van fouten in de hersenen. Deze bevindingen komen overeen met enkele neuroimaging onderzoeken waarin verlaagde activatie van hersengebieden gerelateerd aan de continue evaluatie van gedrag (ofwel de anteriore

cingulate cortex en de orbitofrontale cortex) gevonden wordt in disfunctioneel boze patiëntenpopulaties (Blair, 2012; Coccaro et al., 2007; Davidson, 2000; Fulwiler et al., 2012). Dit onderzoek draagt bij aan de literatuur door een relatie tussen verstoorde foutverwerking en een dispositie tot boosheid eveneens aan te tonen bij een gezonde steekproef.

Waarbij voor het onderzoek beschreven in hoofdstuk 3 gekozen werd voor een design met extreme groepen, is in **hoofdstuk 4** voor een correlationeel design gekozen waarin trait anger als continue maat werd meegenomen. Bovendien werd ditmaal een forensisch psychiatrische steekproef getest in plaats van een steekproef studenten. Het hoofddoel van het onderzoek beschreven in hoofdstuk 4 was om te achterhalen of de scores op de trait anger schaal van forensische psychiatrische patiënten geassocieerd waren met individuele variaties in impulscontrole en foutverwerking gemeten middels gedragsmatige en neurologische indicatoren. Gebruikmakend van dezelfde GoNoGo taak als in hoofdstuk 3 werden accuratesse en ERPs (N2, P3, Pe & ERN) gemeten bij 38 forensisch psychiatrische patiënten met een matig tot hoog recidiverisico[47] op crimineel gedrag. Verwacht werd dat scores op de trait anger schaal bij deze misdadigers negatief zouden samenhangen met het vermogen responsen te onderdrukken op NoGo trials alsook met de ERPs gerelateerd aan impulscontrole (N2 en P3 golven). Net als bij hoofdstuk 3 werd verwacht dat deze effecten sterker aanwezig zouden zijn voor aan boosheid gerelateerde plaatjes dan voor neutrale plaatjes. Tot slot was onze verwachting dat verhoogde scores op de trait anger schaal zouden samengaan met ERPs gerelateerd aan foutverwerking (ofwel verlaagde ERN en Pe golven). In lijn der verwachting vonden we dat hogere scores op de trait anger schaal samen gingen met verlaagde prestaties op NoGo trials; met nog sterkere effecten voor NoGo trials waarbij aan boosheid gerelateerde plaatjes werden getoond. Deze bevindingen zijn in lijn met het cognitieve model van trait anger en reactieve agressie ontwikkeld door Wilkowski en Robinson (2008b, 2010), waarin verondersteld wordt dat impulscontrole ten tijde van vijandige situaties een belangrijke rol speelt in het verklaren van een dispositie tot boosheid.

[47] Recidiverisico staat voor de kans dat een persoon terugvalt in bepaald (veelal ongewenst) gedrag.

In tegenstelling tot onze verwachtingen vonden we echter geen samenhang tussen scores op de trait anger schaal en de neurologische indices van impulscontrole (N2 & P3).

Verlaagde N2 en P3 amplitudes worden herhaaldelijk aangetoond in steekproeven met impulsieve agressieve misdadigers (Chen et al., 2005; Guan et al., 2015; Munro et al., 2007b; Vilà-Balló et al., 2014). Onze steekproef was vrij heterogeen (i.e. misdadigers met verschillende vormen van onderliggende psychopathologie), hetgeen deze onverwachte tegenstrijdige bevinding met eerder onderzoek mogelijk kan verklaren. Een andere verklaring voor deze onverwachtse bevinding is dat neurale indicatoren van impulscontrole mogelijk wel geassocieerd zijn met impulsieve agressieve gedragingen, maar niet zozeer met een dispositie tot boosheid. Met betrekking tot de foutverwerking vonden we in dit onderzoek geen associatie tussen een dispositie tot boosheid met Pe golven, maar wel een negatieve samenhang tussen een dispositie tot boosheid en ERN amplitudes. Anders omschreven vonden we dat hogere scores op de trait anger schaal samengingen met verlaagde ERN golven, hetgeen impliceert dat wanneer de patiënt hoger scoort op een dispositie tot boosheid hij of zij fouten minder goed detecteert op onbewust niveau.

Kort samenvattend kunnen we uit de bevindingen van **hoofdstuk 4** opmaken dat in een forensische psychiatrische populatie een dispositie tot boosheid geassocieerd is met een verminderd vermogen gedrag te onderdrukken op gedragsmatig niveau en een verminderde foutverwerking op onbewust niveau in de hersenen.

In het kort kan men uit de resultaten van **hoofdstuk 3 en 4** concluderen dat een dispositie tot boosheid gerelateerd is aan een verminderde verwerking van fouten. De verminderde foutverwerking speelt vermoedelijk een rol bij mensen met een dispositie tot boosheid in de voortzetting van impulsief gedrag ondanks de negatieve gevolgen. Resultaten met betrekking tot impulscontrole waren gemengd, waarbij een dispositie tot boosheid binnen de forensisch psychiatrische steekproef enkel op gedragsmatig niveau geassocieerd werd met impulscontrole. Er werd geen bewijs gevonden voor een relatie tussen een dispositie tot boosheid en impulscontrole op neurofysiologisch gebied; zelfs niet in de klinische steekproef. Er is duidelijk behoefte aan meer onderzoek waarin de

randvoorwaarden wat betreft de relatie tussen trait anger en impulscontrole verhelderd worden.

Hoofdstuk 5 beschrijft een experimenteel onderzoek waarin getracht werd de invloed van boos rumineren op boosheid en zelfcontrole te meten in vergelijking tot afleiding. Boosheid werd gemeten aan de hand van zelfrapportage middels visueel analoge schalen. Zelfcontrole werd gemeten met behulp van een affectieve GoNoGo taak. Verwacht werd dat proefpersonen die geïnstrueerd werden om te rumineren (herhaaldelijk terugdenken aan de boze situatie) meer boosheid zouden rapporteren en meer fouten zouden maken op NoGo trials op de affectieve NoGo taak in vergelijking met proefpersonen die via instructies werden afgeleid. Allereerst werden alle proefpersonen uit beide groepen geïnstrueerd een autobiografische herinnering waarin zij ontzettend boos waren op te roepen en te bespreken met de proefleider met als doel bij de proefpersonen boosheid op te wekken. Zowel voor als na de woede-inductie werd de stemming van de proefpersonen gemeten. Vervolgens werden proefpersonen verdeeld over ofwel een ruminatie conditie (n = 37) dan wel een afleidingsconditie (n = 35). Proefpersonen in de ruminatie conditie werden gevraagd de boze herinnering wederom terug te halen in hun gedachten en zich te richten op de emotionele aspecten van de gebeurtenis. Proefpersonen in de afleidingsconditie werden geïnstrueerd in gedachten te visualiseren hoe de campus eruit ziet. Tot slot indiceerden de proefpersonen nogmaals hun stemming en voltooiden zij de affectieve GoNOGo taak. In overeenstemming met voorgaand onderzoek toonden de resultaten aan dat boos rumineren leidde tot een toename in boosheid, terwijl afleiding zorgde voor een daling in boosheid (Bushman, 2002; Denson, Moulds, et al., 2012; Denson, 2013; Fabiansson et al., 2012; Konecni, 1974; Mischkowski et al., 2012; Ray et al., 2008; Rusting & Nolen-Hoeksema, 1998). Echter, in tegenstelling tot onze verwachting, presteerden beiden groepen even goed op de GoNoGo taak, hetgeen betekent dat zelfcontrole niet anders beïnvloed werd door boos rumineren dan door afleiding. De meest logische verklaring voor de huidige bevindingen is dat boze ruminatie leidt tot een toename in boosheid, maar geen invloed uitoefent op zelfcontrole; althans niet op impulscontrole zoals gemeten met een affectieve GoNoGo taak.

In **hoofdstuk 6** wordt tot slot een experimenteel onderzoek beschreven waarin de invloed van een mentaal vermoeiende taak (n-back taak) op boosheid en controle over boosheid in reactie op provocerend gedrag van de proefleider werd onderzocht. Er werd verwacht dat mentaal vermoeide proefpersonen meer boosheid zouden ervaren en minder geneigd zouden zijn om met de proefleider samen te werken na provocerend gedrag van de proefleider dan niet-vermoeide proefpersonen. Na het verrichten van een nulmeting werd de ene helft van de proefpersonen gevraagd een uur lang durende neutrale documentaire te bekijken (*n* = 53), terwijl de andere helft geïnstrueerd werd een mentaal vermoeiende 2-back taak te voltooien (*n* = 51) gedurende een uur lang. Vervolgens verrichtten de proefpersonen een frustrerende taak terwijl zij door de proefleider lastig gevallen werden. Nadat de proefpersonen dachten dat het experiment was afgelopen, deed de proefleider tot slot een klein verzoek aan de proefpersonen. Tussentijds werd de stemming van de proefpersonen middels visuele analoge schalen gemeten. Uit de resultaten kwam naar voren dat de proefpersonen die de 2-back taak hadden uitgevoerd meer vermoeidheid rapporteerden dan de proefpersonen die de neutrale documentaire hadden bekeken. Tevens rapporteerden alle proefpersonen een toename in boosheid na de woede-inductie. Hieruit blijkt dat de manipulaties succesvol waren verlopen. Echter, tegenstrijdig met onze verwachtingen, bleken de mentaal vermoeide proefpersonen niet bozer te reageren in reactie op de provocatie dan de niet vermoeide proefpersonen. Wellicht belangrijker is de bevinding dat een groot deel (meer dan 80%) van de proefpersonen, ongeacht in welke conditie zij zaten, wilde gehoorzamen aan het verzoek van de proefleider. Deze bevindingen impliceren dat het verrichten van een mentaal vermoeiende taak niet leid tot hogere prikkelbaarheid en minder controle over boosheid in reactie op provocatie. De resultaten lijken eerder aan te tonen dat zelfs onder stressvolle situaties (mentale vermoeidheid en provocatie) mensen goed in staat zijn hun boosheid te reguleren; indien zij hiertoe gemotiveerd zijn. Deze resultaten komen overeen met dagboekonderzoeken waaruit blijkt dat mensen regelmatig agressieve neigingen en fantasieën ervaren tijdens provocerende situaties, maar desondanks goed in staat zijn hun boosheid te reguleren (bijvoorbeeld Averill, 1983).

Indien we de resultaten uit **hoofdstuk 5 en 6** tezamen nemen, kunnen we concluderen dat hoewel boos rumineren en mentale vermoeidheid gepaard gaan met toenames in boosheid, maar desondanks niet zozeer leiden tot ego depletion (tijdelijke verlagingen in zelfcontrole). Zodoende is er binnen het huidige proefschrift geen empirische ondersteuning gevonden voor associaties tussen cognitieve processen die betrokken zijn bij een dispositie tot boosheid en agressief gedrag.

Beperkingen van de huidige onderzoeken

De onderzoeken uitgevoerd in dit proefschrift zijn niet vrij van beperkingen. Ten eerste kan vanwege het cross-sectionele aard van de onderzoeken beschreven in hoofdstuk 3 & 4 geen conclusie woorden getrokken over oorzaak en gevolg. Ondanks dat een samenhang is aangetoond, blijft het zodoende onduidelijk of de neurocognitieve processen geassocieerd met een dispositie tot boosheid risicofactoren dan wel bijverschijnselen betreffen. Zo is het net zo goed mogelijk dat een derde onbekende variabele de samenhang tussen neurocognitieve processen en een dispositie tot boosheid verklaart. Gezien de heterogene groep die is onderzocht in hoofdstuk 4 is deze verklaring niet uitgesloten. Zo bestaat de mogelijkheid dat een dispositie tot boosheid een symptoom betreft van een onderliggende externaliserende stoornis die gekenmerkt wordt door een lagere ERN (Olvet & Hajcak, 2008), zoals een antisociale persoonlijkheidsstoornis. Een tweede beperking is dat experimentele onderzoeken enkel een oorzakelijke relatie kunnen aantonen tussen neurocognitieve processen en boosheid als emotionele staat, maar niet tussen neurocognitieve processen en een dispositie tot boosheid. Longitudinaal onderzoek (gedurende een langere periode met herhaaldelijke metingen) is zodoende van belang om aan te kunnen tonen dat verstoorde neurocognitieve processen leiden tot een dispositie tot boosheid. Een andere belangrijke beperking is dat de boosheid die is opgewekt in de huidige experimenten mogelijk niet intens genoeg is geweest om effecten te vinden op impulscontrole (H3 & H4) en zelfcontrole (H5 & H6). Mogelijk dient boosheid in hevige mate aanwezig te zijn alvorens het zelfcontrole beïnvloed (Potegal & Qiu, 2010). Een vierde beperking is gekoppeld aan het feit dat een dispositie tot boosheid in dit proefschrift is gemeten met behulp van zelfrapportage.

Aangezien zelfrapportage afhankelijk is van sociale wenselijkheid en zelfinzicht (Hornsveld, 2007; McEwan et al., 2009) is replicatie van belang waarbij gebruik wordt gemaakt van objectieve maten. Een andere beperking heeft te maken met de GoNoGo taak die is gebruikt in dit proefschrift. Aangezien de neutrale foto's altijd als eerst werden getoond, kan een verminderde prestatie op vijandige plaatjes ook te wijten zijn aan volgorde-effecten (bijv. vermoeidheid). Andere resultaten waren wellicht verkregen indien een random design (type plaatje variërend per plaatje) werd gekozen in plaats van een blocked design (dezelfde types foto's per blok). Ook had counterbalancing (waarbij sommige proefpersonen beginnen met de neutrale foto's terwijl anderen beginnen met de vijandige foto's) tot andere resultaten kunnen leiden. Tot slot bestaat er een mogelijkheid dat effecten met een kleine effectgrootte niet zijn opgemerkt vanwege een gebrekkige power (te weinig proefpersonen).

Een belangrijke vraag betreft tot slot in welke mate de effecten die zijn gevonden bij subklinisch boze populaties (ofwel relatief gezonde, hoog opgeleide studenten die kampen met een dispositie tot boosheid) te generaliseren vallen naar groepen patiënten die voor hun boosheid behandeld worden. De bevinding dat een verlaagde impulscontrole wel bij patiënten werd gevonden, maar niet bij studenten onderstreept het belang van deze vraag.

Klinische toepassing en suggesties voor vervolgonderzoek

De STAXI-2 blijkt een klinisch relevant meetinstrument waarmee de ervaring, expressie en regulatie van boosheid in kaart kan worden gebracht. Gelijkend aan de Engelstalige versie, kan de Nederlandse versie van de STAXI-2 gebruikt worden om de aanwezigheid van boosheid en de manier hoe patiënten met hun boosheid omgaan te onderzoeken. De Nederlandse versie van de STAXI-2 kan eveneens ingezet worden om de effectiviteit van behandelingen te evalueren. Een interessante onderzoeksvraag betreft hoe impulsieve gewelddadige misdadigers zouden scoren op de STAXI-2 en maten voor cognitieve controle in vergelijking met overgecontroleerde gewelddadige misdadigers die gekenmerkt worden door een plotselinge boze uitbarsting nadat zij hun boosheid gedurende een langer periode hebben ingehouden (Davey et al., 2005). Hoewel vragenlijsten van toegevoegde waarde zijn, dient toekomstig onderzoek zich eveneens te richten op het ontwikkelen van verschillende

meer objectieve maten waarmee een dispositie tot boosheid in kaart kan worden gebracht (Hornsveld, 2007). Zo kan gebruik gemaakt worden van *ecological momentary asessments* om de aanwezigheid van een dispositie tot boosheid en de mate waarin cognitieve processen hieraan ten grondslag liggen te onderzoeken (R. C. Martin & Vieaux, 2013). In het huidige proefschrift is vooral gekeken naar de relatie tussen een dispositie tot boosheid en cognitieve controle. Toekomstig onderzoek zou zich kunnen richten op de relatie tussen cognitieve controle en de expressie en regulatieschalen van de STAXI-2.

De correlationele en experimentele onderzoeken beschreven in het huidige proefschrift zijn van dusdanig fundamentele aard dat de directe toepasbaarheid in de klinische praktijk nog op zich laat wachten. Ondanks dat het mogelijk wat zal tijd vergen voordat deze fundamentele bevindingen vertaald kunnen worden naar de klinische praktijk, hebben de bevindingen belangrijke theoretische implicaties en bieden ze een aantal aanknopingspunten voor toekomstig onderzoek. Zo kan het EEG bijvoorbeeld in de toekomst ingezet worden als diagnostisch instrument in de behandeling van disfunctionele boosheid. Voornamelijk de ERN en de Pe amplitudes bezitten goede psychometrische kwaliteiten (Hofmann et al., 2012; Olvet & Hajcak, 2009; Rietdijk et al., 2014) en zouden ingezet kunnen worden om te informeren welke individuen een verhoogd risico lopen op een terugval in ongewenst (agressief) gedrag. De ERPs zouden eveneens gebruikt kunnen worden om de effectiviteit en/of uitval van agressietherapie en woedebeheersing te voorspellen. Voorgaand onderzoek op het gebied van middelenafhankelijkheid heeft bijvoorbeeld aangetoond dat de aanwezigheid van gebrekkige cognitieve controle voorspellend is voor (a) een verminderd vermogen om het eigen gedrag als problematisch in te zien, (b) een verlaagde motivatie voor behandeling en (c) een verhoogde kans op vroegtijdige staking van de behandeling (Ersche & Sahakian, 2007; Severtson et al., 2010). Soortgelijke resultaten zijn te verwachten voor mensen met een dispositie tot boosheid. Echter, voordat het EEG als diagnostisch instrument ingezet kan worden, zal toekomstig onderzoek de predictieve validiteit (ofwel de sensitiviteit en specificiteit) van de ERN en de Pe dienen aan te tonen met betrekking tot een dispositie tot woede en een recidiverisico voor gewelddadig gedrag.

Hoewel EEG onderzoeken belangrijke informatie verschaffen over de timing van cognitieve processen in de hersenen, verschaffen zij weinig inzicht over welke hersengebieden geactiveerd zijn tijdens deze neurocognitieve processen. Derhalve, zijn andere imaging technieken, zoals Functional Magnetic Resonance Imgaging, Structual Magnetic Resonance Imaging en Diffusion Tensor Imganing, van belang om meer inzicht te krijgen over neurocognitieve aspecten die betrokken zijn bij een dispositie tot boosheid. Ook dient vervolgonderzoek zich te richten op hoe de hersengebieden die onderliggend zijn aan de cognitieve processen met elkaar verbonden zijn door te kijken naar de functionele connectiviteit (zie bijvoorbeeld Fulwiler et al., 2012). Tot slot kan de invloed van cognitieve controle onderzocht dan wel versterkt worden bij mensen met een dispositie tot boosheid met behulp van neurofeedback, deep brain stimulation en transcraniële magnetische stimulatie. Recente onderzoeken hebben eveneens aangetoond dat cognitieve bias modificatie paradigma's effectief kunnen zijn in het verminderen van boosheid en agressie (Wilkowski et al., 2015).

Het is eveneens van belang dat vervolgonderzoek zich richt op de rol die neurocognitieve processen spelen bij een dispositie tot boosheid onder condities waarin sprake is van emotionele opwinding en hevige provocatie. Zo zou men eerst een woede-inductie of stressinductie kunnen toepassen alvorens de activiteit en invloed van neurocognitieve functies bij mensen met een dispositie tot boosheid te onderzoeken. Binnen gesloten instituten biedt virtual reality technologie hiertoe een uitkomst. Door gebruik te maken van deze relatief nieuwe technieken kunnen patiënten blootgesteld worden aan situaties die boosheid ontlokken onder gecontroleerde omstandigheden.

Verder zou het interessant zijn om te onderzoeken in welke mate gefaalde pogingen boosheid en agressie te controleren, worden beïnvloed door motivationele processen. Uit de resultaten van het huidige proefschrift komen geen objectieve verminderingen in cognitieve controle capaciteiten naar voren als gevolg van toenames in boosheid of vermoeidheid. Mogelijk speelt de perceptie die een persoon heeft over zijn vermogen zichzelf te controleren een grotere rol dan zijn of haar daadwerkelijke capaciteit zichzelf te controleren. Mensen met een dispositie tot boosheid dienen zich bewust gemaakt te worden van het feit

dat zij hoogstwaarschijnlijk voldoende capaciteiten beschikken om zichzelf te reguleren ondanks dat zij vermoeid of in een geagiteerde staat verkeren. Door deze informatie kenbaar te maken voelen mensen met een dispositie tot boosheid zich mogelijk meer verantwoordelijk voor hun acties dan wel vaardiger, waardoor zij wellicht meer controle zullen vertonen over hun boosheid en agressie in provocerende situaties. Eveneens zou het interessant zijn om te onderzoeken hoe verstoringen in de beschreven (neuro)cognitieve processen zich ontwikkelen. Er zijn bijvoorbeeld indicaties dat verstoringen in informatieverwerking die gerelateerd zijn aan een dispositie tot boosheid ontwikkelen als gevolg van een hardhandige aanpak van ouders (Dodge, 2006; Pollak et al., 2000). Tot slot is er behoefte aan meer onderzoek waarin de wisselwerking tussen meerdere neurocognitieve processen betrokken bij een dispositie tot boosheid onderzocht wordt.

Slotconclusie

Gebaseerd op de onderzoeken beschreven in dit proefschrift kunnen enkele belangrijke conclusies worden getrokken. Allereerst kan geconcludeerd worden dat de STAXI-2 een betrouwbaar meetinstrument is die door clinici en onderzoekers gebruikt kan worden om de ervaring, expressie en regulatie van boosheid te meten. Ten tweede blijkt een dispositie tot boosheid gerelateerd aan verminderde cognitieve controle in zowel neutrale als vijandige condities, waarbij verstoringen in de foutverwerking het meest consistent gerelateerd zijn aan een dispositie tot boosheid. Verminderde impulscontrole in relatie tot een dispositie tot boosheid kon niet consistent aangetoond worden; ook niet onder condities waarin boosheid werd opgeroepen. Tot slot toont dit proefschrift aan dat ondanks dat boos rumineren en mentale vermoeidheid samenhangen met toenames in boosheid, deze processen geen tot een beperkte invloed hebben op cognitieve controle. Over het geheel genomen worden belangrijke neurocognitieve inzichten geboden met betrekking tot een dispositie tot boosheid, waarmee beter begrip is ontstaan voor waarom mensen met een dispositie tot boosheid frequent boos worden en impulsieve gedragingen voortzetten ondanks de negatieve gevolgen op lange termijn.

Dankwoord

Acknowledgements in Dutch

Bij de term “onderzoeker” ontstaat al snel het beeld van een ongeschoren, geïsoleerde in zichzelf mompelende man die uit het niets EUREKA noemt. Niets bleek echter minder waar: onderzoek is teamwerk en onderzoekers blijken ook nog eens vlotte, interessante, leuke mensen te zijn waarmee je prettig kan samenwerken. Ik wil graag mijn dank en waardering uitspreken voor de mensen die met hun steun, kennis, wijsheid, bemoedigende woorden en raad en daad hebben bijgedragen aan dit proefschrift.

Allereerst wil ik mijn dank betuigen aan mijn (co)promoteren zonder wiens hulp dit proefschrift er niet had gelegen.

Professor Hovens, beste Hans, er zijn weinig mensen die me keer op keer weten te boeien met hun kennis en verhalen, maar u is dat gelukt. Ik heb veel geleerd van uw wijsheid, praktische oplossingen en uw klinische, nuchtere kijk op zaken. Ik heb zowel de inhoudelijke alsook de wat meer luchtige gesprekken over zaken als reizen, films en het leven an sich, zeer gewaardeerd. Bedankt voor het vertrouwen en de betrokkenheid die u gedurende het hele project in mij heeft getoond. Of u nu gewoon thuis was, dan wel ver van huis in onder andere Californië, Israël, Rusland en Malawi verkeerde, u stond altijd vrijwel direct voor mij klaar en wist boven verwachting snel te reageren met waardevolle tips.

Ook mijn promotor professor Ingmar Franken ben ik erg dankbaar. Beste Ingmar, ik heb ontzettende waardering voor jouw integriteit. Ik vind het bijzonder knap hoe jij keer op keer de persoonlijke ontwikkeling van je promovendi voorop stelt. Ik voelde me door jou gesteund in mijn groeiproces als onderzoeker en ik ben het persoonlijke contact met de tijd meer en meer gaan waarderen. Ik kijk met veel plezier terug naar de borrelmomenten waarbij ik naast je bescheiden kant ook je meer feestelijke kant heb mogen ontdekken. Ik heb daarnaast ontzettende waardering voor hoe jij met soms kleine tips en subtiele opmerkingen artikelen substantieel weet te verbeteren. Ik kijk er naar uit verder aan de Erasmus Universiteit verbonden te blijven, wetende dat we elkaar daar nog vaak zullen treffen.

Dr. van der Veen, beste Freddy, als dagelijks begeleider (copromotor) stond jij eveneens dagelijks voor me klaar met wijze raad en daad. Jij wist ondanks de hectiek van alle dag frequent tijd voor mijn vragen vrij te maken om mij te adviseren, waardoor ik meestal

direct weer verder kon. Jouw gedegen commentaar op mijn papers, oprechte interesse en droge humor maakte de samenwerking voor mij meer dan prettig. Dank hiervoor!

Dr. Huijding, beste Jorg, de brainstormsessies met jou zal ik niet snel vergeten. Je vragen stimuleerde mij iedere keer weer tot nadenken en je enthousiaste pogingen om de theorie volledig te doorgronden werkte aanstekelijk. Ook bleken je tekstuele wijzigingen vaak van essentieel belang. Toen de moed mij langzaam in de schoenen begon te zakken, wist jij mij direct weer op te peppen en te motiveren. Dat was een leerzame ervaring en hiervoor ben ik je zeer dankbaar!

Ook mijn oud begeleider dr. Elke Geraerts neemt voor mij een belangrijke plaats in. Elke, sinds het eerste contact had jij vertrouwen in mij en konden we goed met elkaar overweg. Jij stimuleerde mij om de kans om te promoveren met beide handen aan te pakken en hebt mij zodoende gebracht tot waar ik nu ben. Ik heb ontzettende waardering voor hoe jij de wetenschap voor een breed publiek weet te vertalen naar de praktijk. Ik hoop nog vaak met je te samenwerken en je in de toekomst te blijven zien.

Zonder mijn kamergenoten, Jesper Hopstaken en Vincent Hoogerheide had dit project nooit zo prettig, leerzaam en lollig geweest als dat het was. De combinatie van enerzijds waardevolle, praktische en handige tips en anderzijds ontzettend veel lol en humor, maakte dat ik elk dag met plezier naar mijn werk ging. Wellicht is het *power hour* hier wel het beste voorbeeld van: enerzijds stimuleerde dit om iedere dag gericht aan mooie publicaties te werken, anderzijds leidde dit op slechte dagen (of goede?) dagen ertoe dat het soms bij dat ene uurtje bleef…..Doordat we allen hetzelfde proces doorliepen, wisten we elkaar competitief uit te dagen (alle papers op het prikbord, trakteren bij een publicatie, kamer met de hoogste Impact factor?), maar ook mental support te bieden. Ik kan oprecht zeggen dat ik er goede vrienden aan heb over gehouden! Mannen, bedankt voor de mooie tijd!

Mijn collega's bij het Instituut voor Psychologie hebben mij altijd een warm gevoel gegeven en steun en gezelligheid geboden; bedankt hiervoor. In het bijzonder wil ik de C3-ers (Lydia Arends, Kim Bul, Linda Dekker, Sanne van Dongen, Ingmar Franken, Colin van der Heiden, Pauline Jansen, Marlies Marissen, Birgit Mayer, Marieke van Meggelen, Martijn

Oostra, Marike Polak, Daniëlle Remmerswaal, Susan van Rijen, Guus Smeets, Freddy van der Veen, Ilse Verveer, Marjolein Wals) bedanken voor hun betrokkenheid en waardevolle feedback op mijn onderzoek. Mijn oud-kamergenoten Anita Eerland, Marijntje Zeijen, Mario de Jonge, Maartje Luijten en Ali Mohd Bin MD Salim [die jammer genoeg te vroeg is heengegaan] wil ik bedanken voor de gesprekken en ondersteuning. Oud-promovendi Angela Nederlof, Reshmi Marhe, Maartje Luijten, Anita Euser en Marianne Littel wil graag bedanken voor de adviezen en tips die mij zeer zeker geholpen hebben het traject op te starten. Jullie hebben mij vaak verder op weg geholpen met adviezen. Tevens waren jullie bereid onderzoekstaken met mij te delen, waar ik veel baat bij heb gehad. Ook alle medepromovendi, waarmee ik mijn onderzoek en lief en leed heb mogen delen tijdens borrels, “PhDinners” en lunchpauzes, ben ik dankbaar.

Een onderzoeksproject is niet mogelijk zonder instanties die hun hulp verlenen en zonder mensen die bereid zijn zich voor de wetenschap in te zetten. Alle proefpersonen, studenten, coördinatoren, ondersteunende personeelsleden en overige mensen die direct of indirect bij de onderzoeken betrokken zijn geweest: dank voor jullie hulp! In het bijzonder wil ik Ben van de Wetering, Ron van Outsem, Mike de Clercq en Els Bourgonjon bedanken. Zonder jullie hulp en inzet bij de werving van de proefpersonen afkomstig van Bouman, Delta en Sint-Jan Baptist Zelzate had ik nooit zulke interessante doelgroepen kunnen testen. Ook is een speciale dankbetuiging aan Gerrit Jan de Bie, Christiaan Tieman en Marcel Boom op zijn plaats. Jullie ondersteuning bij de ontwikkeling van de taken en de dataverwerking was van groot belang. Ook mijn masterstudenten en onderzoeksassistenten die soms met bloed, zweet en tranen hebben geholpen bij de dataverzameling verdienen het om in het zonnetje gezet te worden, te weten Alexandros Solos, Ahmet Yazir, Joy Heersma, Joost van Vierzen, Pryska Maas, Jeroen de Jong, Jolien van der Haar, Melissa van Rekom, Emel Heybeli, Desiree van Winden, Linda van der Kaaij, Margot van Dalen, Lucia den Hertog en Bart Kögeler. Lilian Naeije, jou wil ik in het bijzonder bedanken voor je hulp. Knap hoe jij je doel om onderzoeker te worden achterna streeft. Dat er maar vele publicaties mogen volgen!

Zowel voordat ik als promovendus aan dit PhD project begon als tijdens het project heb ik bij verschillende mensen het vak als scientist practitioner mogen leren. Ook zij

verdienen een plaats in dit dankwoord. Peter Muris, als onderzoeksassistent heb ik bij jou mijn eerste taak ontwikkeld en *informed consent* geschreven (die typerend met vele rode letters en track changes werd teruggestuurd). Hierbij heb ik ook mogen leren dat er een beest bestaat dat een Cuscus heet (al weet ik nog steeds niet of hij nu bijt of niet…). Mijn interesse voor de wetenschap is bij jou begonnen. Ontzettend leerzaam was ook het onderzoek dat ik mocht verrichten bij TBS patiënten tijdens mijn stage onder leiding van Ruud Hornsveld, Thijs Kanters en Almar Zwets. Jullie hebben mijn interesse in woede aangewakkerd alsook aangetoond dat onderzoek van praktisch nut kan zijn. Colin van der Heiden, samen met Elke Geraerts, schreef ik met jou mijn eerste artikel. Ook buiten het schrijven van artikelen om, heb ik veel kunnen leren van jouw praktische vaardigheden als clinicus waar ik nu de vruchten van pluk. Jan Rodenburg bedankt voor je praktische tips omtrent het STAXI-2 onderzoek alsook dat je mij de gelegenheid hebt geboden onderzoek te vervolgen naar morele beschadiging bij veteranen. Ik kijk uit naar een prettige samenwerking. Martijn Oostra, met veel plezier heb ik met je mogen sparren over je onderzoek naar Inquiry Based Stress Reduction bij depressieve patiënten. Daarnaast heb ik de persoonlijke gesprekken en je adviezen over mijn terugkeer naar de praktijk zeer gewaardeerd. Ook mijn huidige collega's van HSK wil ik bedanken voor de mogelijkheid die zij mij hebben geboden om de geleerde kennis in praktijk te mogen brengen.

Tevens wil ik de leden van de promotiecommissie (Eric Rassin, Jan van Strien, Henk Nijman, Matthias Wieser, Anton Loonen en Ben van de Wetering) bedanken voor hun bereidwilligheid mijn proefschrift te lezen en mij hierover kritisch te bevragen tijdens de verdediging van mijn proefschrift.

Onderzoeksschool Experimental Psychopatholgy (EPP) verdient eveneens lof. Ik heb veel mogen leren van de symposia die door het EPP georganiseerd werden. Daarnaast heb ik tijdens de vele overnachtingen in de congreshotels in Utrecht en Heeze mooie contacten op kunnen doen met overige EPP leden. De samenwerking met Lieke Nentjes die na mijn promotietraject is gestart is hier een goed voorbeeld van. Lieke Nentjes, bedankt voor de mogelijkheid bij de UvA samen onderzoek te verrichten. Laten we hopen dat er een mooie publicatie uit volgt.

Benjamin Wilkowski, Tom Denson, and Raymond Novaco: special thanks for sharing some inspiring articles and advices that helped me to progress my career as a researcher in the field of anger. As leading researchers in the field of anger, I have immensely appreciated your advice.

Ten tijden van inspanning is ook ontspanning van belang. De digitale (PSP 4) en real-life voetbalwedstrijden met de Port of Rotterdam Killer Whales (Mario de Jonge, Jan Engelen, Vincent Hoogerheide, Jesper Hopstaken, Wim Pouw, Steven Raaijmakers, Tim van Marlen, Ali Mohd Bin MD Salim en Gertjan Rop) waren onvergetelijk mooi. Ook waren de Clinical Cognition Lab meetings, borrels en etentjes met Marieke van Meggelen, Kevin van Schie, Daniëlle Remmerswaal, Sabine Wanmaker, Elke Geraerts zowel inspirerend als vreugdevol. Sabine Wanmaker, wij waren destijds als klinische promovendi ongeveer tegelijkertijd begonnen. Vaak liepen we bij elkaar binnen voor vragen en ondersteuning en we hebben vele reizen gemaakt met alle workshops, symposia en congressen die we samen bezocht hebben. Jouw optimisme was inspirerend; zelf een verloren koffer tijdens een week congres in Peru kreeg jou niet klein. Thanks voor de leuke tijd! Marieke van Meggelen, ook met jou heb ik vele persoonlijke en prikkelende gesprekken mogen voeren (dien je een depressieve patiënt nu wel of niet te pamperen?). Ontzettend leuk om jou als collega te hebben mogen leren kennen. Maryse Kruithof, wij hebben elkaar leren kennen tijdens het Honours Program en sindsdien met groot plezier contact gehouden. Ik zal de lunches in het T-gebouw niet snel vergeten.

“Bedankt” is een te klein woord voor de mensen die zo’n centrale plek in mijn leven innemen als mijn vrouw, vrienden en familie. Met jullie hulp, vertrouwen en steun is geen uitdaging te groot. Lieve ouders, jullie staan werkelijk waar altijd voor mij klaar. Jullie onvoorwaardelijke liefde is iedere dag voelbaar en maakt dat ik vol vertrouwen in de wereld sta. Ik voel me ontzettend dankbaar met jullie in mijn leven en kan me geen betere ouders wensen. Kim, grote zus van me, met soms kleine adviezen, heb jij een groot effect op mijn leven. Robin, buiten het feit dat je een topzwager bent, ben je ook een uitstekende coach! Jij weet altijd met een korte doortastende zin, precies het juiste te zeggen. Ook mijn schoonfamilie heeft mij met open armen ontvangen en bood de welnodige ontspanning ten

tijde van dit project, waarbij de kerstvieringen eruit springen (Samuel Sumter, over 20 jaar verwacht ik een nog mooier boekje van jou, kleine professor). Jan-Willem, Damir, Laurens, Paul, Jeroen, jullie zijn ware vrienden voor het leven; een half gebaar is genoeg! Jullie staan zowel in goede tijden als mindere tijden altijd voor mij klaar, waarvoor dank. Special thanks to JW en Damir die met hun handige IT skills mijn leven als onderzoeker hebben vereenvoudigd. Damir, ook onze Starbucks momenten op de EUR gaven mij vaak weer energie om met goede moed aan het project verder te werken! Paul, als ik even niet meer wist hoe ik iets mooi en adequaat kon vertalen, kwam jij met adequate vertalingen op de proppen. Ook bedankt voor de mooie filosofische gesprekken die we hebben gehad over dit onderwerp en vele andere onderwerpen.

Lieve Hannah, zelfs tijdens onze eerste date heb je mij moeten missen vanwege mijn onderzoek. Ik diende nog wat brieven te posten en kwam te laat…Bedankt dat je mij altijd de ruimte en vrijheid hebt gegeven om mezelf te ontwikkelen; dat je me hebt helpen ontspannen met je maffe grapjes in tijden van stress; dat je me stimuleert mijn dromen achterna te jagen en …. dat je me soms ook even een schop onder mijn kont geeft wanneer dat nodig is. Een man is zo sterk als de vrouw die achter hem staat, luidt de uitdrukking. We hebben in de korte tijd dat we samen zijn al vele uitdagen mogen trotseren en mooie momenten mogen meemaken. Ik houd van jou en hoop nog vele mooie momenten met je te delen.

Publications

2016

Lievaart, D.M., Veen, F.M. van der, Huijding, J., Naeije, L, Hovens, J.E. & Franken, I.H.A. (2016). Trait Anger in Relation to Neural and Behavioral Correlates of Response Inhibition and Error-processing. *International Journal of Psychophysiology, 99*, 40-47. doi: 10.1016/j.ijpsycho.2015.12.001

Lievaart, D.M., Franken, I.H.A. & Hovens, J.E. (2016). Anger Assessment in Clinical and Nonclinical Populations: Further Validation of the State–Trait Anger Expression Inventory-2. *Journal of Clinical Psychology, 72* (3), 263-278. doi: 10.1002/jclp.22253

Lievaart, D.M., Huijding, J., Veen, F.M. van der, Franken, I.H.A., & Hovens, J.E (In Press). The impact of angry rumination on anger-primed inhibitory control. *Journal of Behavioral Therapy & Experimental Psychiatry*.

2015

Lievaart, D.M., Erciyes, F., Veen, F.M. van der, Wetering van de, B. J. M., Muris, P. & Franken, I.H.A. (2015). Validation of the Cocaine Versions of the Obsessive Compulsive Drug Use Scale and the Desires for Drug Questionnaire. *American Journal of Drug and Alcohol Abuse, 41* (1), 358-365. doi: 10.3109/00952990.2015.1043210

2014

Hovens, J.E., **Lievaart, D.M.** & Rodenburg, J.J. (2014). STAXI-2: Vragenlijst over boosheid. Amsterdam: Hogrefe.

2013

Lievaart, D.M., Heiden, C. van der & Geraerts, E.G. (2013). Associations between Depressive Symptoms, Rumination, Overgeneral Autobiographical Memory and Interpretation Bias within a Clinically Depressed Sample. *Journal of psychology and psychotherapy, S7-004*, 1-6. doi: 10.4172/2161-0487.S7-004

Submitted

Lievaart, D.M., Veen, F.M. van der, Huijding, J., Franken, I.H.A., & Hovens, J.E. The Relation between Trait Anger and Impulse Control in Forensic Psychiatric Patients: an EEG Study. Manuscript submitted for publication.

Lievaart, D.M., Huijding, J., Veen, F.M. van der,. Hovens, J.E., & Franken, I.H.A. Does Mental Fatigue Have an Impact on Anger and Prosocial Behavior Following Provocation? Manuscript submitted for publication.

Presentations

Rodenburg, J.J. & **Lievaart, D.M.** (2016, January)**.** *Moral Injury: Van theorieontwikkeling tot RCT.* Landelijk Zorgsysteem voor Veteranen, Doorn, The Netherlands.

Lievaart, D.M. (2015, October). *Short Fuses & Bad Tempers: Testing the (Neuro)Cognitive Aspect of Trait Anger.* Experimental Psychopathology Symposium, Heeze, The Netherlands.

Lievaart, D.M., Veen, F.M. van der, Huijding, J., Franken, I.H.A., & Hovens, J.E. (2015, August). *The Relation between Trait Anger and Impulse Control in Forensic Psychiatric Patients: An EEG Study.* Insri Meeting: Amsterdam, The Netherlands.

Lievaart, D.M., (2015, March). *Does Mental Fatigue Have an Impact on Anger and Prosocial Behavior Following Provocation?* Battle of the Brains, Graduate School Experimental Psychopathology, Utrecht, The Netherlands

Lievaart, D.M., I.H.A. Franken, & Hovens, J.E. (2014, October). *Trait Anger and Anger in Psychiatric Patients.* Graduate Research Day, The Institute of Psychology, Erasmus University Rotterdam, Rotterdam, The Netherlands.

Lievaart, D.M. (2014, September). *The (Neuro)Cognitive Aspects of Trait Anger.* Honours Science Conference, Rotterdam, The Netherlands.

Lievaart, D.M., Rodenburg, J.J., Franken, I.H.A. & Hovens, J.E. (2014, July). *Anger and Anger Expression Strategies among Different Groups of Psychiatric Outpatients.* XXIst World Meeting ISRA, Atlanta, Georgia, The United States of America.

Lievaart, D.M., I.H.A. Franken, & Hovens, J.E. (2013, November). *Psychometric properties of the Dutch version of the State-Trait Anger Expression Inventory-2.* Graduate Research Day, The Institute of Psychology, Erasmus University Rotterdam, Rotterdam, The Netherlands.

Lievaart, D.M., Franken, I.H.A. & Hovens, J.E. (2013, October). *The Prevalence of Trait Anger and its Relationship with Aggression, Hostility, and Psychological Distress within Psychiatric Outpatients.* The 8th European Congress on Violence in Clinical Psychiatry, Ghent, Belgium.

Lievaart, D.M., Geraerts, E.G., Franken, I.H.A. & Hovens, J.E. (2013, July). *The Prevalence of State Anger and Trait Anger Within Psychiatric Outpatients.* World Congress of Behavioral and Cognitive Therapies, Lima, Peru.

Lievaart, D.M., Heiden, C. van der & Geraerts, E.G. (2013, July). *The Combined Cognitive Bias Hypothesis Tested within a Clinically Depressed Sample: Associations between Interpretation Bias, Rumination, Overgeneral Memory and Depressive Symptoms.* World Congress of Cognitive Behavioral Therapies, Lima, Peru.

Lievaart, D.M., I.H.A. Franken, & Hovens, J.E. (2012, October). *The Cognitive Aspects of Trait Anger and Anger in Psychiatric Patients.* Graduate Research Day, The Institute of Psychology, Erasmus University Rotterdam, Rotterdam, The Netherlands.

Curriculum Vitae

Marien Lievaart was born on April 2nd, 1988, in Maassluis, the Netherlands. After completing his secondary education (VWO) at Accent College in Maassluis, he started studying Psychology at the Erasmus University of Rotterdam in 2006. He obtained his Bachelor's degree in 2009 and his Master's degree in Clinical Psychology in 2011. During his Bachelor's degree, Marien Lievaart also worked as a research assistant for prof. dr. Peter Muris (2008-2009) conducting research on the role of information processing biases in the onset and maintenance of anxiety and started working as a practitioner for Inzowijs (2008-2013) where he guided children suffering from developmental disorders (mostly Attention Deficit Hyperactivity Disorder and Autism). During his Master's degree, Marien took on both a practical healthcare internship (GZ-stage) at the forensic psychiatric hospital the Kijvelanden, and an extensive, research-based internship at the Clinical Cognition Lab under the supervision of Elke Geraerts. His master thesis concerned a cognitive bias modification intervention for depression. Immediately after obtaining his degree, Marien started working as a tutor in general psychology at the Institute of Psychology, Erasmus University Rotterdam. In January 2012, Marien Lievaart started working here as a PhD candidate, in collaboration with Antes Mental Health Care, under the supervision of prof. dr. Ingmar Franken and prof. dr. Hans Hovens. The studies in this PhD project focused on the neurocognitive processes involved in trait anger, resulting in the present thesis. During his PhD, he participated in the education program of the Dutch-Flemish post-graduate research school 'Experimental Psychopathology'. Furthermore, he co-authored the manual of the Dutch version of the State-Trait Anger Expression Inventory-2, taught and coordinated several psychology courses (Depression and Psychosis, Highlights of Psychology, Forensic Psychology), (guest) lectured on clinical subjects, reviewed empirical articles for international journals, and supervised the theses of bachelor and master psychology students. Currently, Marien Lievaart works as a clinical psychologist for HSK Groep B.V., treating patients suffering from axis 1 psychopathology, such as anxiety, depression, fatigue and somatic symptoms. He also works as an academic teacher for the Erasmus University of Rotterdam and started a collaboration with several researchers affiliated with *Het Landelijk Zorgsysteem voor Veteranen* conducting research on moral injury in war veterans.