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Silent Rage

Understanding and Treating Maladaptive Anger Inhibition

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Till Love och Belle.

Abstract

Maladaptive anger inhibition can be defined as the excessive suppression of angry expressions while remaining intensely angry from within. A growing body of research suggests that maladaptive anger inhibition is associated with various negative outcomes including physical health risks and psychopathology. This thesis aimed to advance our understanding of maladaptive anger inhibition in adults and develop and evaluate novel treatments for this problem. To this end, four studies were conducted.

Study I explored for the presence of anger subgroups in an anger-treatment-seeking sample and identified four groups. Of primary relevance was an "anger-in" profile characterized by low outward anger expression combined with elevated internal anger, consistent with maladaptive anger inhibition. *Study II* used network analysis to examine associations among anger components and regulation strategies in the same sample, identifying anger rumination as a central process potentially involved in maintaining maladaptive anger inhibition. *Study III* experimentally investigated the effects of state anger suppression in combination with subsequent regulation strategies, showing that suppression followed by rumination increased anger, whereas suppression followed by reappraisal or continued suppression was associated with reduced anger and improved physiological regulation. *Study IV* evaluated an emotion regulation-focused treatment for maladaptive anger inhibition, demonstrating feasibility, acceptability, and promising preliminary effects.

Together, findings suggest that maladaptive anger inhibition represents a clinically relevant problem that may be responsive to targeted emotion regulation-focused treatment.

Keywords: Anger, Aggression, Anger Suppression, Anger Inhibition, Anger Rumination, Emotion Regulation, Mindfulness

Tackord på Svenska

Att det blev just *ilska* som jag skulle skriva om känns på sätt och vis ödesbestämt. Även om jag ofta beskrivs som lugn och sansad – åtminstone av människor utanför min familj – har ilska alltid legat mig nära till hands. Inte nödvändigtvis på något destruktivt vis, utan snarare som en inre motor som har drivit mig framåt när det känts tufft.

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List of Papers

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I. Larsson, J., Bjureberg, J., & Hesser, H. (2023). Anger profiles among individuals seeking treatment for maladaptive anger: Associations with emotion regulation. *Clinical Psychology and Psychotherapy*. 30 (3), 599-610. https://doi.org/10.1002/cpp.2821

II. Larsson, J., Bjureberg, J., Zhao, X., & Hesser, H. (2024). The inner workings of anger: A network analysis of anger and emotion regulation. *Journal of Clinical Psychology*, *80*(2), 437-455. https://doi.org/ 10.1002/jclp.23622

III. Larsson, J., Denson, T., Oskarsson, S., Brandt, H., Lang, A., Bjureberg, J., Tuvblad, C., & Hesser, H. When Anger Stays Inside – An Experimental Investigation of the Effects of Anger Suppression and Post-Suppression Regulation on Anger and Heart Rate Variability. [Under Review in *Behaviour Research and Therapy*]

IV. Larsson, J., Ojala, O., Bjureberg, J., Sederström, P., Hvass, O., Björk, P., Lidskog, S., & Hesser, H. (2025). Online emotion regulation treatment for maladaptive anger inhibition–an open-label pilot with a pooled interrupted time series design. *Cognitive Behaviour Therapy*, 1-16. https://doi.org/10.1080/16506073.2025.2542355

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Preface

"*Anger Is a Gift*"— Rage Against the Machine, *Freedom*, 1993

Anger is powerful. Unlike other negative emotions, that often pull us toward withdrawal or passivity, anger mobilizes. It propels us to act. Across history, anger has fueled resistance against injustice, discrimination, racism, and abuses of power, sharpening moral boundaries and motivating efforts to confront what feels wrong. Anger may also feel good. The blood warms, attention narrows, and a surge of physiological activation prepares the body for action. In such moments, anger may indeed feel like a gift.

At the same time, anger may also lead us astray. As my perhaps somewhat romanticized depiction suggests, anger tends to simplify the social world. When angry, we may divide people into heroes and villains, right and wrong, us and them. In doing so, anger may narrow rather than expand the range of possible actions. Instead of fostering change, anger can harden positions, escalate conflicts, and perpetuate cycles of resentment, conflict and, at worst, result in aggression, violence, and even death.

Problems with anger, however, are not always visible. At times, anger unfolds in silence, in the privacy of one's own thoughts. A person may replay an argument long after it has ended, lie awake rehearsing what they should have said, or carry a growing sense of grievance without taking steps to resolve it. At those times, anger can be deeply felt yet remain largely unspoken. When expressed, anger may be misdirected toward a safer but innocent target, or emerge at the wrong moment. Over time, such patterns can create a world experienced as hostile, unjust, and unwelcoming.

This thesis focuses primarily on this less visible form of anger—herein referred to as *maladaptive anger inhibition* or simply *anger-in*, defined as intensely felt anger that is rarely outwardly expressed. Understanding how maladaptive anger inhibition develops, is maintained, and can be effectively treated among adults, represents central aims of the work presented here.

Background

Introduction

To suppress impulses of anger may be crucial for facilitating social interaction and interpersonal wellbeing (Consedine et al., 2002). If left unrestrained, anger may lead to problematic expressions including verbal and physical aggression (Finkel & Hall, 2018; Wilkowski & Robinson, 2010), which in turn may cause harm to others and oneself. However, research suggests that some individuals may excessively suppress external expressions of anger while still remaining intensely angry from within, also known as *maladaptive anger inhibition*. Maladaptive anger inhibition—often referred to as *anger-expression-in*[1] or simply *anger-in* (Spielberger, 1999)—has been consistently associated with a range of adverse outcomes.

For example, anger-in has been linked to increased risk of physical health problems, including cardiovascular disease (e.g., Chida & Steptoe, 2009; Williams, 2010) and heightened sensitivity to acute and chronic pain (Burns et al., 2008a), as well as various emotional disorders such as depression and social anxiety (Novaco, 2010; Versella et al., 2016). Greater usage of anger suppression is also linked with generally lower levels of social well-being, including reduced relationship satisfaction and perceived social support (Chervonsky & Hunt, 2017). Furthermore, although anger-in is generally associated with lower levels of overt aggression, emerging evidence suggests that anger-in may be associated with certain indirect forms of aggression, such as

[1] I use the terms *maladaptive anger inhibition* (Burns et al., 2008a) and *anger-in* (Spielberger, 1999) interchangeably throughout this thesis. However, as will be discussed later in this background, these constructs are not assumed to be fully equivalent. The broader theoretical concept of maladaptive anger inhibition may not be fully captured by the empirical anger-in construct, which mostly focuses on the suppression of behavioral expression. Nevertheless, because most empirical research has operationalized maladaptive anger inhibition using Spielberger's anger-in scale, this term is adopted for pragmatic reasons when referring to such studies.

displaced aggression (Denson et al., 2006), in which suppressed anger is redirected toward safer targets, such as an intimate partner (Eckhardt et al., 2008). A more detailed overview of the intra- and interpersonal consequences of maladaptive anger in general, and maladaptive anger inhibition in particular, is presented later in the thesis (pp. 32–40).

Taken together, these findings suggest that maladaptive anger inhibition may indeed constitute a clinically significant pattern of responding, with negative consequences for psychological and physical health, as well as interpersonal functioning. This raises important questions regarding the mechanisms that contribute to the development and maintenance of maladaptive anger inhibition, as well as the conditions under which it can be effectively treated. Throughout this thesis, I examine these and related questions in greater detail. *Section I* reviews major theories and empirical findings on emotion and emotion regulation before turning to the defining features of anger. *Section II* focuses on the clinical aspects of anger, including when anger becomes maladaptive, its consequences, and proximal and distal factors involved in its development and maintenance. This section concludes with a discussion on psychological treatments for maladaptive anger. *Section III* addresses conceptual challenges related to maladaptive anger inhibition. It begins by examining how the construct has been defined and then introduces a theoretical model of maladaptive anger inhibition. The section concludes by outlining two major research gaps. Finally, *Section IV* presents the aims guiding the empirical studies in this thesis.

Section I: Emotion, Emotion Regulation, and Anger

This section introduces the interrelated processes of *emotion generation* and *emotion regulation* and reviews key concepts in these areas. The section concludes by outlining the key features of anger, including its determinants, phenomenology, and manifestation.

Emotion

Attempts to define and understand emotions, such as anger, can be traced back to the ancient Greeks (Harris, 2009). However, it was not until the mid-19th century that emotions began to be studied systematically, giving rise to the modern scientific study of emotion, also known as affective sciences[2].

While contemporary emotion theorists debate the precise structure and causal nature of emotions, most agree that the generation of an emotional episode (*emotion generation*) involve coordinated changes across multiple interacting components. These components commonly include: (a) *cognitive* components, such as situational evaluations (appraisals) and attention; (b) *affective* components, including physiological arousal and subjective hedonic feelings (e.g., pleasant versus unpleasant); and (c) *behavioral* components, such as facial expressions and action tendencies (e.g., Russell, 2003; Russell & Barrett, 1999; Scarantino, 2016, p. 5).

[2] Emotion theories are often separated into three different overarching traditions, each offering slightly different accounts on *how* emotions are generated: basic-, constructionist- and appraisal theories of emotion. Put simply basic emotion theories propose that a limited set of discrete, biologically grounded emotions (e.g., fear, anger, joy) are elicited by evolutionarily relevant stimuli and supported by dedicated neural and physiological circuitry (e.g., Ekman, 1992; Izard, 2009; Ortony & Turner, 1990; Tomkins & McCarter, 1964). Constructionist theories emphasize the role of core affect and context in the construction of an emotional experience (e.g., Lindquist, 2013). Appraisal theories focus on how cognitive evaluations (i.e., appraisals) give rise to emotion (e.g., Ellsworth, 2013).

The most basic ingredient of an emotion experience, however, is often argued to be *core affect* (e.g., Russell, 2003; Russell & Barrett, 1999). Core affect is defined as a continually varying physiological state with two basic dimensions: valence (pleasant–unpleasant) and arousal (activated–deactivated; Russell, 1980). Core affect is considered an ever-present background state that does not constitute an emotion in itself but provides the basis from which emotions emerge through the integration with other relevant contextual and perceptual information (Barrett, 2006; Lindquist, 2013).

Importantly, the concept of valence can be understood at multiple levels, including the subjective feeling state, the valence of the eliciting event, and the valence of the anticipated consequences of emotional responding (Carver & Harmon-Jones, 2009; Harmon-Jones et al., 2008). This becomes especially important when discussing the valence of anger, which although often defined as a negatively valanced emotion, also oftentimes consist of a positively valenced component (Harmon-Jones et al., 2008). For example, the feeling of anger is typically experienced as unpleasant (Szymaniak et al., 2023), and so is its triggering event (e.g., a provocation), yet the anticipated outcomes may be of positive valence (e.g., resolving conflict; Litvak et al., 2009).

While some argue that meaning-making or appraisal processes are the next important ingredient for an emotion to be constructed (Barrett, 2017; Lindquist, 2013), others argue that appraisals are a necessary determinant or cause of an emotion (Ellsworth, 2013; Frijda, 1986; Lazarus, 1991; Scherer & Moors, 2019). The term *appraisal* was first coined by Magda Arnold (1960) to refer to the cognitive process by which situations are evaluated as either beneficial or harmful, drawing on both automatic bodily reactions and more reflective judgments. In contemporary formulations, appraisal is understood as a process by which individuals assess the significance of an event for their well-being, goals, values, and beliefs. When events are appraised as facilitating or obstructing one's concerns, specific emotional responses are believed to be elicited (Lazarus, 1991). Decades of research have generated a substantial body of empirical work mapping specific appraisal patterns onto specific emotions (for a recent review, see Yeo & Ong, 2024). Anger has, for example, been described as

arising from appraisals of goal obstruction, other-blame, and perceived offense against the self (ego-threat), often accompanied by perceived coping potential that individual can handle the situation through confrontational action (Lazarus, 1991 pp. 222-226).

That emotions are closely linked to events that bears on an individual's well-being and personal goals have long been recognized (Darwin, 1999/1872). As articulated by Ekman & Cordaro (2011, p. 364) emotions "prompts us in a direction that, in the course of our evolution, has done better than other solutions in recurring circumstances that are relevant to our goals.", highlighting the functional value of emotions (Ekman, 1992; Ekman & Cordaro, 2011; Izard, 2009; Ortony & Turner, 1990). Indeed, emotions provide us with important information that guide our behavior and our attention. Fear, for example, promotes detection of danger and withdrawal, whereas anger signals goal obstruction or potential harm and mobilizes approach-oriented responses (Ekman & Cordaro, 2011). Despite their functionality emotions can sometimes lead us astray, especially when emotions are overly intense, prolonged, or simply contextually misaligned, prompting efforts to regulate the emotional response.

Emotion Regulation

Emotion regulation[3] can be broadly understood as the abilities we have, and the processes (i.e., strategies) we use to influence either our own emotional experience (self-regulation) or that of others (other-regulation; Gross, 1999). This thesis will focus on self-regulation. Emotion regulation has been implicated as a central process across various psychological domains, contributing to both adaptive functioning and the development and maintenance of psychopathology (i.e., emotion dysregulation), including various anxiety- and depressive disorders, as well as problems with anger and aggression (e.g., Aldao et al., 2016; Aldao et al., 2010; Pop et al., 2025; Smith et al., 2026).

Early conceptualizations of emotion regulation emerged from the stress and coping literature and the appraisal tradition of emotion (e.g., Lazarus, 1991; Lazarus & Folkman, 1984). Herein emotion-focused coping strategies denoted cognitive and behavioral strategies intended to control, or downregulate, negative emotional responses to stressors (Lazarus & Folkman, 1984). As the field of emotion regulation began to emerge, however, the conceptualization of emotion regulation broadened to include not only a focus on the downregulation of negative emotions but also its upregulation, focusing on both negative and positive emotions (Gross, 1998, 1999, 2015).

Importantly, contemporary accounts of emotion regulation recognize the functional role of emotions in guiding our attention and behavior (e.g., Gross, 1999). As such, whilst emotion regulation indeed may involve a focus on downregulating, avoiding or "getting rid" of an emotion or emotional response it may also involve simply changing the

[3] The extent to which emotion generation and emotion regulation should be conceptualized as distinct has been debated. Some, particularly basic emotion theorists, propose partially separable systems in which automatic, bottom–up emotional responses can be modulated by top–down regulation. Appraisal and psychological constructionist approaches, by contrast, often view generation and regulation as closely intertwined. For a review, see Gross & Feldman Barrett, 2011).

intensity, duration or frequency of emotional experiences across experiential (e.g., thoughts and feelings), physiological, and behavioral components, in the service of a regulatory goal (Gross, 2015). For example, this may involve downregulating sadness with the immediate goal of reducing subjective distress and alleviating low mood in the moment (i.e., feeling less sad), or modulating anxiety in a more instrumental way, such as reducing anxiety-related arousal in order to indirectly improve performance during a public speaking event.

Thus, emotion regulation should not be viewed as inherently adaptive or maladaptive; rather, its adaptiveness depends on how regulatory strategies are flexibly deployed in context (Aldao et al., 2015; Bonanno & Burton, 2013). For instance, chronic avoidance or downregulation of negative emotions may paradoxically contribute to increased emotional distress over time (Hayes et al., 1996), whereas the ability to downregulate the intensity of anger in order to express it in a firm but non-aggressive manner may facilitate more adaptive interpersonal functioning. Moreover, as will be elaborated below, adaptive emotion regulation is also thought to rest on a set of core abilities such as recognizing emotions as functional and informative signals rather than experiences that should be avoided or eliminated.

Emotion Regulation Abilities

Although definitions of adaptive emotion regulation vary, there is broad agreement that it rests on a set of core, trait-like abilities (Bjureberg, 2026; Gratz & Roemer, 2004; Hofmann & Kashdan, 2010). These may, for example, include the capacity to identify and understand emotions, accept emotional experiences, and maintain goal-directed behavior under emotional distress (i.e., distress tolerance; Gratz & Roemer, 2004). Together, these abilities enable flexible, context-sensitive selection and implementation of regulatory strategies, allowing individuals to modulate emotional intensity or expression as needed (Bjureberg, 2026). Conversely, deficits in these domains—such as poor emotional clarity, low acceptance, or limited distress tolerance—are associated with rigid and context-insensitive strategy use (e.g., habitually suppressing emotions), which may contribute to different types of psychopathology, such as anger problems

(Gardner & Moore, 2008; Roberton et al., 2012). Importantly, emotion regulation abilities are shaped by the interplay of biological predispositions (e.g., temperament), environmental influences (e.g., culture, norms, and early events), and learning processes (e.g., classical, instrumental, and social learning; Wright et al., 2025), and are therefore both relatively stable and modifiable over time (Bjureberg, 2026).

Emotion Regulation Strategies

The aforementioned emotion regulation abilities provide the foundation for how specific *emotion regulation strategies* can be used to regulate emotions in the moment. The Extended Process Model of emotion regulation offers a comprehensive account of how regulatory strategies may be employed across various stages of the emotion generation sequence (Gross, 2015). Drawing on appraisal theories of emotion, the model conceptualizes emotion generation as unfolding across four stages—*situation*, *attention*, *appraisal*, and *response*. Specifically, an individual first encounters an emotion-eliciting *situation* (external or internal), directs *attention* toward or away from aspects of the situation, *appraises* its meaning in relation to personal goals, and ultimately produces a multi-component emotional *response* encompassing subjective experience (e.g., feelings), expressive behavior, and physiological changes.

If the emotion generated is not aligned with the person's desired emotional state—that is, "how they want to feel"—an emotion regulation goal is often identified. For example, an individual who feels anger rising during a tense meeting may aim to feel calm to maintain professionalism and avoid conflict. Once a regulation goal is *identified*, one or more strategies are *selected*, *implemented*, and *monitored* to see whether one successfully achieve one's regulatory goal (Gross, 2015)

The extended process model further organizes emotion regulation strategies into five "families", defined by *when* in the generation sequence they "intervene": situation selection, situation modification, attentional deployment, cognitive change, and response modulation. *Situation selection* involves strategies aimed at approaching or avoiding situations based on their anticipated emotional impact; for example, an individual prone to anger might choose to cancel a meeting

with a colleague known to be confrontational. *Situation modification* refers to altering aspects of the environment to reduce its emotional impact, such as rearranging a meeting agenda to prevent anger from escalating. *Attentional deployment* involves shifting focus within a situation, for instance distracting oneself by focusing on non-emotional aspects of the situation. *Cognitive change* includes strategies that focus on how one thinks about an emotion eliciting situation. Most notably this involves the strategy of *cognitive reappraisal*, which can be defined as attempts to reconsider, reframe or gain a new perspective an emotional situation so as to change its emotional meaning (McRae, 2016), such as interpreting a colleague's critical feedback as constructive rather than threatening, thereby reducing anger intensity. Finally, *response modulation* occurs after the emotional response has been generated and includes strategies like *expressive suppression*, where, for example, outward expressions of anger are withheld (e.g., keeping a neutral face).

In what follows, three of the most commonly studied strategies will be described in a bit more detail—emotional suppression, cognitive reappraisal, and rumination—and evidence regarding their adaptiveness and effectiveness.

Suppression, Cognitive Reappraisal, and Rumination

While the suppression of emotional expression (*expressive suppression*) is the most extensively studied form of suppression, experimental typologies (Webb et al., 2012) suggest that emotional suppression can be further differentiated into suppression of emotion-related thoughts (*thought suppression*) and suppression of the internal emotional experience (*experiential suppression*). As an overarching construct, emotion suppression is generally regarded as maladaptive. Indeed, studies suggest that the tendency to habitually use emotion suppression (i.e., trait use) is associated with lower mental health and well-being (Hu et al., 2014), lower levels of positive affect (Fernandes & Tone, 2021), poorer interpersonal functioning (Chervonsky & Hunt, 2017), and higher levels of psychopathology (Aldao et al., 2010).

Experimental research further indicates that the moment-to-moment use of suppression (i.e., state use) may be problematic. For example,

while suppression may effectively reduce outward emotional expression, it appears to have limited effects on subjective emotional experience and may even increase physiological activation (Webb et al., 2012), although its effects on physiological responding are not entirely consistent (Tyra et al., 2024; Zaehringer et al., 2020). In addition, suppression has been shown to impair memory for emotional events (Richards & Gross, 2000; Richards & Gross, 2006).

Importantly, context matters: occasional suppression of emotions such as anger in social interactions can facilitate more favorable interpersonal evaluations, including perceptions of warmth and competence (Green et al., 2025), a topic I will return to in section II.

Cognitive reappraisal, in contrast, is generally regarded as an effective and adaptive strategy. Trait reappraisal is consistently linked to greater well-being, better interpersonal functioning, and lower psychopathology (e.g., Aldao et al., 2010; Hu et al., 2014; McRae, 2016). Experimental research likewise demonstrates that state reappraisal effectively reduces negative affect and attenuates physiological responses to emotional stimuli (Webb et al., 2012). Despite these benefits, individual often report that they use reappraisal relatively infrequently (Suri et al., 2015), which likely reflects that cognitive reappraisal is often a quite cognitively demanding strategy (Milyavsky et al., 2019). Its effectiveness may also be reduced during high-intensity emotions (Ford & Troy, 2019), such as anger, where strategies like distraction can be more effective (Denson et al., 2012; Shafir et al., 2015). These findings suggest that cognitive reappraisal may, in some contexts (e.g., during high intensity emotions), need to be complemented by other strategies or underlying regulatory capacities to work effectively.

Finally, rumination is a regulatory strategy characterized by repetitive and passive focus on the causes, meanings, and consequences of negative emotional experiences (Nolen-Hoeksema et al., 2008; Watkins, 2008; Watkins & Roberts, 2020). Per definition, rumination has often been conceptualized as maladaptive, involving habitual dwelling and thinking on negative information in abstract ways rather than engaging in active problem-solving (Watkins & Roberts, 2020). In line with this, trait rumination shows robust moderate-to-large associations

with a range of psychopathologies, including depression, anxiety, and maladaptive anger and aggression (Aldao et al., 2016; Aldao et al., 2010; Anestis et al., 2009; Denson, 2013; Hu et al., 2014; Watkins & Roberts, 2020). Experimental studies further show that inducing rumination in the lab (i.e., state rumination) intensifies and prolongs negative affect and increases physiological arousal (Kirkegaard Thomsen, 2006; Ottaviani et al., 2016).

However, not all forms of emotional processing of negative content are maladaptive. For instance, experimental studies suggest that adopting a self-distanced perspective when reflecting on negative experiences may reduce emotional reactivity and promote insight, in contrast to repetitive self-immersed rumination (Murdoch et al., 2023)Kross et al., 2005; Kross & Ayduk, 2017). Similarly, related constructs such as emotional processing (Rachman, 1980) emphasize deliberate, constructive engagement with emotions, which instead can confer adaptive benefits (Pennebaker, 1989).

Anger

Turning to anger, the construct has been defined in multiple ways, with related concepts, such as aggression and hostility, sometimes used interchangeably with anger (DiGiuseppe & Tafrate, 2007). In the present thesis, however, these constructs are treated as distinct. Anger is conceptualized as an emotional state, hostility as a cognitive–attitudinal disposition that may predispose individuals to experience anger, and aggression as a potential behavioral consequence of anger. More specifically, to provide a clear conceptual foundation, the present thesis adopts the definition proposed by Kassinove and Sukhodolsky (1995, p. 11).

> "Anger refers to a label given to a constellation of specific uncomfortable subjective experiences and associated cognitions (i.e., thoughts, beliefs, images, etc.) that have various associated verbal, facial, bodily, and autonomic reactions. It is a transient state, in that it eventually passes, and it is a social role, in that our culture or subculture allows for the display of certain kinds of behaviors associated with the internal experience, but punishes others. Thus, anger is felt in people's conscious awareness and is communicated through verbalizations and bodily reactions."

I will now delve deeper into anger by discussing its preceding events (i.e., anger determinants), its unique phenomenology, and potential functions.

Determinants of Anger: Triggers, Appraisals, and Pre-Anger States

According to Deffenbacher's (2011) cognitive model of anger, anger unfolds at the intersection of what happens to us (*anger triggers*), the internal state we bring into the situation (*pre-anger states*), and how the event is interpreted in the moment (*appraisals*). Each of these determinants of anger will now be outlined.

Anger Triggers

Deffenbacher (2011) categories anger triggers into three broad classes of events: *external events*, *internal stimuli*, and a combination of the two. External triggers most commonly arise from other people's behavior (i.e., interpersonal events). Supporting this, Kashdan et al. (2016) found that 63% of anger episodes in university students were interpersonal. Similarly, in Averill's (1983) early survey studies on anger aggression, 72.5% of anger episodes in a community sample were reported as being triggered by another person, with over half involving either a loved one (29%) or someone well known and liked (24%). These interpersonal triggers typically involve provocations, perceived injustices, criticism, or other events of personal significance to the individual, often involving some form of violation against social norms or personal expectancy (Averill, 1982).

Importantly, external triggers are not limited to social interactions and external agents (often termed *provocations*; Miller et al., 2003). They also include non-social events and triggers (often termed *frustrations*; Miller et al., 2003). These trigger may, for example, include one's own behavior (i.e., self-anger; Ellsworth & Tong, 2006), frustrating situational constraints (e.g., being stuck in traffic; Berkowitz, 1989), and interactions with inanimate objects (e.g., malfunctioning technology; Berkowitz, 1990; Berkowitz & Harmon-Jones, 2004).

The second class of events, internal stimuli, is also a common trigger of anger. Kashdan et al. (2016) reported that approximately 14% of anger episodes were triggered by internal psychological or physical distress. Internal stimuli encompass both cognitive and emotional material, including ruminative thoughts, activation of anger-related memories (e.g., replaying a past argument), or other emotions that may elicit anger (e.g., anger emerging in response to shame; Deffenbacher, 2011).

The third class of events reflects situations in which internal and external stimuli interact, creating a heightened vulnerability for anger. For example, receiving a critical remark from a colleague may be

particularly anger-inducing when it resonates with unresolved past conflicts or ongoing self-critical thought.

Pre-Anger States

Anger is also influenced by the state we have when entering an anger-eliciting situation, also known as *pre-anger states.* Such states may include, for example, *past* affective states (e.g., heightened negative affect following intense physical exercise; Zillmann et al., 1972), and *current* affective states (e.g., fatigue, hunger; Berkowitz, 1990). Together, these states affect the likelihood that we will experience anger, and if so, its intensity. In short, states that are affectively similar to anger—such as negative feelings combined with high arousal when hungry ("hangry"; MacCormack & Lindquist, 2019) or feeling sleepy (Krizan & Hisler, 2019)—may increase one's vulnerability to anger triggers.

Beyond transient states, more stable factors such as individual assumptions, values, rules, and cultural norms surrounding anger may further shape how frequently and intensely anger is experienced. For example, Harmon-Jones and colleagues have shown that individuals with more positive attitudes toward anger also report an increased tendency to experience anger (Harmon-Jones et al., 2011; Szymaniak et al., 2023).

Anger Appraisals

With roots in stoic philosophy emotion theorists have long regarded appraisals as the major determinant of anger (Harris, 2009). Worth of note, while often viewed as causal determinants that *precedes* emotions (e.g., Lazarus, 1991; Moors, 2013; Roseman & Evdokas, 2004), appraisals may also occur *with* or *following* an emotional response (Berkowitz & Harmon-Jones, 2004; Frijda, 1993).

One of the most commonly proposed appraisals associated with anger is the appraisal of *goal obstruction*. Goal obstruction encompasses any event appraised as blocking one's attainment of goals, concerns or more basic needs (Lazarus, 1991) that are of *personal significance* (Frijda, 1993). Examples include thwarted autonomy or control, frustrations in the form of omitted rewards, or interruptions to plans and

goal-directed behavior, as well as violations of moral or social standards (Berkowitz, 1989).

Importantly, the appraisal of goal obstruction may also involve more fundamental *threat appraisals* (Novaco, 2014), that are relevant to the individual's well-being, including potential physical or psychological harm. Indeed, in his seminal work on defensive responding, Cannon (1915) early on conceptualized perceived threat as a core antecedent of both anger and fear, mobilizing the organism toward fight (anger) or flight (fear) depending on situational demands (for a review on anger and fear, see Schultz et al., 2009).

Yet, as many negative emotions involve appraisals of goal obstruction, this appraisal alone is often insufficient to classify an emotional episode as anger (Lazarus, 1991). When the goal blockage is also attributed to an external agent (someone or something) that is to be blamed, anger is more likely to arise (*blameworthiness* and *accountability*). For example, Lazarus (1991) suggested that the overarching appraisal (core relational theme) of anger was an attack against the self or those close to me, and that this attack was performed by some blameworthy agent.

If the event is further appraised as *unfair* or *illegitimate*, the likelihood of anger increases even more. This may involve judging the situation as intentional, negligent, or preventable, rather than accidental (Frijda, 1986; Lazarus, 1991). More generally, anger is likely when an event violates one's expectations or the standards of what one believes "ought to happen".

A final, crucial appraisal dimension is *coping potential*—the perceived ability to take action to address or correct the provoking event. As stated by Frijda anger is the reaction to an nonacceptable event that is deemed to be "…amenable to change" (Frijda, 1986, p. 199). Likewise, Lazarus (1991) points out that the capacity to respond is often what separates anger from emotions as fear, which instead may arise when a threat is uncontrollable.

In sum, anger is most likely to arise when a triggering event is appraised as obstructing one's goals, is attributed to a responsible agent,

is perceived as unfair or illegitimate, and is seen as amenable to change through action. However, anger can also occur with only some—or even none—of these appraisals, for instance when elicited directly by negative affect (Berkowitz, 1990; Berkowitz, 2012; Berkowitz & Harmon-Jones, 2004). This illustrates the dual pathway of anger elicitation, either via deliberate cognitive appraisals or more automatic processes.

The Phenomenology of Anger: Anger Intensity, Frequency, and Duration

Anger is said to vary in intensity, from mild irritation to intense rage[4]. The intensity of anger typically reflects the perceived significance of the provocation (at least in many instances of non-clinical anger): small provocations or frustrations elicit, low-levels of anger, whereas more substantial triggers elicit stronger anger (e.g., Vasquez et al., 2013). As anger intensifies, its cognitive, affective, physiological, and behavioral components also amplify—attention narrows physiological arousal rises, angry thoughts increase, and the readiness to confront or remove the source of frustration grows (Berkowitz, 2012).

Most individuals experience anger on a daily or weekly basis. Large-scale daily diary studies suggest that any form of anger may be experienced in approximately 10 % (Trampe et al., 2015) to nearly 16 % (Scherer et al., 2004) of sampled moments in everyday life. Of these anger episodes, however, few appear to be intense and strong. For example, in a sample of over 47,000 Swedish adults Titova et al. (2022) found that 9 % reported experiencing intense anger at least weekly, suggesting that the majority of individuals did not experience intense anger.

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[4] While some authors consider, for example anger and rage, to be distinct emotions—or variants of anger (e.g., Lewis, 2009)—I align with Spielberger & Reheiser, 2009 and Berkowitz (2012) in viewing them as reflections of anger intensity. In this view, anger exists on a continuum, ranging from mild irritation to intense rage.

In terms of duration, anger usually subsides quickly, often within minutes, as the provoking event passes or the individual disengages from it (Potegal, 2010). More intense episodes, however, can linger, particularly when the provocation remains unresolved (e.g., absence of communication in an angry conflict; Fridhandler & Averill, 1982) or when individuals reenact the triggering event via thoughts, and memories (e.g., anger rumination; Potegal, 2010).

For some individuals, anger is not only intense but also frequent and long-lasting. To capture these individual differences, anger is often separated into its state and trait form. *State anger* refers to the temporary experience of anger in response to specific situations, while *trait anger* reflects a stable tendency to become angry across time and contexts (Spielberger & Reheiser, 2010). Individuals high in trait anger tend to experience anger more frequently, intensely, and for longer durations than those low in trait anger (e.g., Alcázar-Olán et al., 2015; Tafrate et al., 2002). In this way, trait anger can be seen as a dispositional vulnerability that shapes the likelihood and course of anger episodes, contributing to what is sometimes referred to as *maladaptive anger,* the focus of the next section.

The Function and Expression of Anger

One of anger's core features, distinguishing it from other negative emotions, is its close relation to approach motivation and aggression. As noted in the introduction, unlike most negative emotions, which typically elicit withdrawal, anger is negatively valenced but triggers approach-related tendencies, often manifesting as aggressive behavior or aggressive inclinations (e.g., Averill, 1982; Berkowitz, 2012; Ekman & Cordaro, 2011; Frijda, 1986; Lazarus, 1991). Frijda (1986), for example, described goal obstruction as a "terminal signal for aggressive behavior" (p. 89), while Ekman and Cordaro (2011) highlights anger's evolved function in *motivating* efforts to remove obstacles and stop perceived harm, often with a wish to aggress against the instigator of one's anger. Consistent with these theoretical accounts, a substantial body of empirical research has linked anger to various correlates of approach-related motivation (for a review, see Carver & Harmon-Jones, 2009).

This action-oriented preparation is indeed reflected in both the physiological and attentional components of anger. When angered, the body enters a high-arousal state, characterized by increases in, for example, heart rate, blood pressure (particularly diastolic), skin conductance, and muscle tension[5] (Stemmler, 2009). Likewise, anger is associated with heightened alertness and determination (Harmon-Jones, 2003), with attention being more readily directed toward perceived threats (Schultz et al., 2009). For example, experimental findings have shown that angered individuals are more likely to detect facial signals of threats, such as angry (Ceccarini & Caudek, 2013) and aggressive faces (Shasteen et al., 2015), and interpret ambiguous cues as potentially threatening (Barazzone & Davey, 2009). Such attentional, interpretational, and physiological changes likely reflect an evolutionarily adaptive, self-protective function of rapidly detecting imminent threat and preparing the individual for aggressive confrontation of the threat (e.g., Öhman et al., 2001).

This is not to suggest that anger always leads to approach behavior or aggression. On the contrary, while most report feeling an urge to aggress when angry (action tendency), most anger episodes do not result in actual aggressive acts (Averill, 1982, pp. 194-199). Rather, aggressive motor impulses are typically regulated and inhibited. However, as Averill (1982) notes, "…inhibition [of anger] is seldom complete…" (p. 194): even when anger is not expressed, aggressive tendencies may persist covertly as thoughts, urges, or fantasies.

Moreover, anger does not only serve aggressive or confrontational functions. Anger has also been posited to fulfill other important social functions (Averill, 1982; Novaco, 1976). Anger can facilitate cooperative social living, support reflective self-awareness, and enable the use of symbolic or indirect means to address problems. In

[5] Although definitive evidence that physiologically separates anger from other negative emotions is lacking, reviews suggest several notable differences between, for example, the emotions of anger and fear (see, Stemmler, 2009). For example, Stemmler (2009, p. 109) notes that, compared to fear, anger elicits greater facial temperature, diastolic blood pressure, total peripheral resistance, and muscle tension.

interpersonal and societal contexts, anger may act as a social signal, communicating perceived norm violations, enforcing moral standards, and motivating corrective or restorative action (Averill, 1982, p. 320), see also Novaco (1976) for a discussion of the diverse functions of anger.

Gender Differences in Anger Expression

Regarding gender differences, a common stereotype in many Western contexts is that men experience and express more anger, particularly through direct aggression, whereas women are more likely to suppress anger or express it indirectly. But to what extent is this stereotype supported by empirical evidence? With respect to the subjective experience of anger, studies generally suggest that men and women report similar levels of anger, indicating limited gender differences (Fischer & Evers, 2009). However, when it comes to the expression of anger, empirical evidence indeed somewhat support the contention that men tend to express anger more directly while women are somewhat more likely to suppress or express it indirectly (Fischer & Evers, 2009). Importantly, these differences appear highly context dependent.

Experimental studies, for example, show that men and women report similar levels of anger following provocation, yet women express anger less in social contexts, a difference that disappears in non-social settings (Evers et al., 2005). Similarly, research comparing relationship contexts suggests that women suppress anger more than men in traditional relationships, whereas this difference disappears in egalitarian relationships (Fischer & Evers, 2011). Such findings suggest that gender differences in anger expression are shaped less by differences in anger experience and more by social norms, interpersonal power dynamics, and contextual expectations regarding appropriate emotional displays.

Section II: Maladaptive Anger

This section begins by defining the construct of maladaptive anger and summarizing its prevalence in clinical and non-clinical populations, before reviewing its consequences, associated risks, and the distal and proximal factors that contribute to its development and maintenance, with a particular focus on emotion regulation. The section concludes with an overview of evidence-based treatments for anger.

Defining Maladaptive Anger

Unlike anxiety, which has given rise to multiple diagnostic categories, anger is the central feature of only one formal diagnosis – *Intermittent Explosive Disorder* (IED). In DSM-5, IED is defined by recurrent, impulsive, and, to the context, grossly disproportionate verbal or physical aggressive outbursts (Coccaro, 2011). While the DSM-5 specifies that these outbursts may be either impulsive or anger-based—a distinction not clearly articulated in DSM-IV—IED captures only one manifestation of maladaptive anger—anger-driven aggression.

Maladaptive anger, however, can emerge in multiple forms. For example, DiGiuseppe and Tafrate (2007, pp.270-272) proposed an anger disorder called *anger-regulation expression disorder* that suggests that maladaptive anger may manifest in two different forms, which may co-occur: (a) internalized anger, characterized by intense, frequent, and enduring anger that manifests through sustained feelings, physiological arousal, or rumination, and/or (b) externalized anger, involving anger-driven aggressive or outward expressions. Other diagnostic proposals (e.g., Eckhardt & Deffenbacher, 1995; Martin, 2020) similarly recognize variations in anger expression and experience.

However, while the introduction of additional anger disorders could increase clinical and research attention, elevations in anger and anger-related problems cut across multiple diagnostic categories, including mood and anxiety disorders, PTSD, and personality disorders, highlighting its *transdiagnostic nature* (see also "Maladaptive Anger and Comorbidity" later in this section). The need to address maladaptive anger therefore persists regardless of the presence of a separate

diagnosis (Gardner & Moore, 2013). Moreover, as others have emphasized, the intensity, frequency, or duration of anger alone is not necessarily maladaptive. Such responses may be contextually appropriate, for example, in chronically toxic, harsh, or punitive environments. Maladaptive anger is better conceptualized in terms of the behavioral consequences it engenders, which can adversely affect personal, vocational, and interpersonal functioning (Deffenbacher, 2011; DiGiuseppe & Tafrate, 2007; Fernandez, 2009; Gardner & Moore, 2013).

Nonetheless, in line with DiGiuseppe and Tafrate's (2007) proposed anger diagnosis, numerous researchers have long recognized at least two subtypes of maladaptive anger that warrant attention: anger directed outward (e.g., through aggression) and anger experienced primarily internally (e.g., as intense angry thoughts and feelings) (Blackburn, 1968; Funkenstein et al., 1954; Harburg et al., 1973; Megargee, 1966; Roberton et al., 2012; Spielberger, 1999).

The most influential operationalization of this distinction comes from Spielberger's State–Trait Anger Expression Inventory (STAXI; Spielberger, 1999; Spielberger et al., 1988), which conceptualizes these tendencies as *anger-out*—the propensity to express anger toward others or objects—and *anger-in*—the propensity to experience anger internally while suppressing its outward expression. Importantly, anger-in and anger-out are orthogonal constructs rather than opposite ends of a single continuum (Eckhardt et al., 2004), meaning that individuals may exhibit both simultaneously (e.g., high anger-in and high anger-out).

Building on the above, I herein define maladaptive anger as intense, frequent, and enduring anger accompanied by dysregulated expression, whether expressed outwardly (anger-out) or inwardly (anger-in), that results in clinically significant distress or functional impairment for the individual or those around them.

Estimating the Prevalence of Maladaptive Anger

As there is no shared, consensus definition of maladaptive anger, estimating its prevalence is challenging. Epidemiological data on anger-driven aggression nevertheless provides some context. For example, Okuda et al. (2015) found that ~8 % of respondents in a U.S. community sample ($N \approx 34{,}000$, U.S. adults) reported having experienced excessive anger leading to temper outbursts or aggression, such as hitting or throwing objects, at least once during their lifetime. Similarly, meta-analytic evidence from 17 countries further suggests a pooled lifetime prevalence of Intermittent Explosive Disorder (IED) of 5.1% and a 12-month prevalence of 4.4%, although estimates vary substantially across countries and populations (Liu & Yin, 2025). In Western countries, lifetime prevalence of IED are somewhat higher, typically in the 7 % range for lifetime prevalence (Kessler et al., 2006). Rates are also generally higher in clinical samples (10.5%; Liu & Yin, 2025). These estimates, however, primarily reflect anger-out tendencies and aggressive outbursts.

By contrast, problems with anger-in, or maladaptive anger inhibition, remain largely unquantified, likely because clear criteria for when anger-in becomes maladaptive are lacking. Nevertheless, observational studies using the anger-in scale (Spielberger, 1999) indicate that anger-in is often elevated in clinical samples (e.g., Lievaart et al., 2016; Pop et al., 2025), suggesting at least a higher prevalence of anger-in in clinical populations.

Consequences and Outcomes of Maladaptive Anger

Anger, Aggression, and Violence

The link between anger and aggression is among the most extensively studied areas in anger research. Accordingly, this section gives particular attention to aggression, situating it as a potential consequence of anger.

Although aggression and violence can be distinguished, they are here conceptualized along a continuum, with violence representing a more extreme form of aggression. Specifically, *aggression* can be defined as any behavior directed at another individual with the proximate intent[6] to cause harm, where the target is motivated to avoid it (Anderson & Bushman, 2002). *Violence*, in turn, refers to severe forms of aggression involving the intent to inflict serious physical harm (e.g., stabbing). The actual occurrence of harm, however, is not necessary; both aggression and violence are defined by the intent to cause harm, even if the attempt fails (Anderson & Bushman, 2002). Acts of aggression or violence may in certain situations be functional and contextually adaptive (e.g., in self-defense), herein however, I focus on when anger leads to contextually inappropriate or excessive aggression or violence

Subtypes of Aggression

As noted in Section I, many episodes of anger are accompanied by an action tendency toward confrontation or attack, indicating a close functional relationship between anger and aggression. Nevertheless, aggression does not always arise from anger. To distinguish aggression driven by affective states from aggression motivated by more strategic goals, researchers commonly differentiate between hostile and instrumental aggression (Anderson & Bushman, 2002).

Note that some researchers, such as Berkowitz (2012), place less emphasis on intent, arguing that many aggressive acts are impulsive and arise from automatic, affect-driven processes rather than deliberate, consciously mediated appraisals.

Hostile aggression (also termed reactive, impulsive, or affective aggression) has traditionally been seen as impulsive and un-planned aggression. Although most often associated with anger, it may also be elicited by other aversive states (Berkowitz, 2012), and emotions such as shame (Elison et al., 2014). In contrast, *instrumental aggression* (also referred to as proactive or goal-directed aggression) involves more deliberate actions in which harm is a means to an end, serving broader goals such as material gain, dominance, or social status (Bushman & Anderson, 2001).

This distinction is particularly relevant for understanding maladaptive anger. Maladaptive anger is most closely linked to hostile forms of aggression[7], where heightened emotional reactivity, biased appraisals processes, hostile attributions and difficulties regulating anger all have been posited to increase the likelihood of disproportionate aggressive responses (e.g., Allen et al., 2018; Berkowitz, 1990; Gardner & Moore, 2008; Roberton et al., 2012; Wilkowski & Robinson, 2010). Aggressive behavior itself can take multiple forms, ranging from direct verbal (e.g., insults) and physical acts (e.g., punching someone) to more indirect expressions (e.g., spreading rumors).

The Link Between Anger Expression and Aggression

Not surprisingly, most instances of anger-driven aggression and violence are associated with high levels of trait anger and anger-out, rather than anger-in (e.g., Deffenbacher, Oetting, Lynch, et al., 1996). For example, meta-analytic findings suggest moderate to large associations between trait anger and intimate partner violence (d = 0.51; Birkley & Eckhardt, 2015), aggressive driving (r = .31; Zhang & Chan, 2016), and violence among offender populations (d = 0.86; Chereji et al., 2012).

[7] As Bushman and Anderson (2001) note, real-world aggression rarely fits neatly into hostile or instrumental categories. For example, individuals may plan an aggressive act instrumentally while simultaneously experiencing intense anger both during the planning phase and during its execution.

However, while limited research exists, there are indications that individuals high in anger-in are not entirely free from aggression. Anger-in is generally negatively associated with overt forms of aggression (e.g., verbal and physical aggression; Deffenbacher, Oetting, Lynch, et al., 1996), but has been linked to more indirect expressions. Early work by Averill (1982, p. 194), for example, found that individuals who strongly inhibited anger reported lower levels of direct aggression (e.g., talking negatively about someone to a third party) but higher levels of indirect aggression compared to those low in inhibition. Moreover, although aggressive impulses are typically suppressed, they may at times be redirected toward safer targets—a phenomenon known as displaced aggression.

Displaced aggression is more likely when the original provocateur is unavailable or perceived as too powerful to confront directly, leading to inhibited expression followed by later redirection toward a less threatening target (Miller et al., 2003). Displaced aggression may be particularly relevant for individuals high in anger-in, who are more likely to suppress immediate reactions than those high in anger-out. Indeed, empirical support remains sparse but suggestive. For example, in developing a measure of trait displaced aggression, Denson and colleagues reported a moderate association between anger-in and displaced aggression (r = .32; Denson et al., 2006). Moreover, across two experiments Rajchert and Winiewski (2016) found that scoring high on avoidance and inhibitory tendencies (similarly to anger-in) predicted lower levels of immediate retaliatory aggression following experimental provocation, but greater aggression at a later occasion (i.e., displaced aggression).

One potential target of displaced aggression is an intimate partner. Indeed, a meta-analysis by Maloney et al. (2023) reported a small positive association between anger-in and IPV perpetration ($r \approx .20$), although this estimate was based on only three studies. Similarly, a recent review of IPV among undergraduate students (Neilson et al., 2023) concluded that emotional suppression and rumination—particularly when combined with high trait anger and alcohol use (Maldonado et al., 2015)—may be associated with greater risk of partner aggression compared to more adaptive strategies (e.g.,

reappraisal). Importantly, it remains unclear to what extent such findings reflect individuals with predominantly anger-in versus mixed anger expression profiles (e.g., high in both anger-in and anger-out).

Providing some clarification to this matter Eckhardt and colleagues identified three subgroups of court-mandated IPV perpetrators, a high anger-expressive group (n = 56), a low anger group (n = 118), and a moderate anger-inexpressive group characterized by elevated anger-in (n = 13). Relative to the low anger group, both the expressive and inexpressive groups showed higher rates of rearrest, with the inexpressive group displaying a particularly elevated risk for assault-related offenses. These findings suggest that even predominantly anger-in tendencies may confer increased IPV risk in specific subpopulations.

Relatedly, in another field of study, clinical observations and case reports suggest that a subgroup of violent offenders may be characterized by excessive tendencies to control and suppress experiences and expressions of anger (i.e., anger overcontrol). For example, Blackburn (1968) described two subtypes of violent offenders: a *conforming type*, who denies experiencing anger, and an *inhibited type*, who suppresses anger expression despite substantial internal anger (e.g., rumination). Both of these subtypes of violent offenders have been suggested to be an increased risk for extreme violent outbursts. For a review of anger overcontrol in violent offending, see Davey et al. (2005).

Overall, anger-in is typically associated with a lower propensity for aggression and violence. However, under certain conditions and for some individuals, habitual suppression combined with sustained internal anger (e.g., rumination) may contribute to elevated risk.

Anger and Physical Health

The relationship between anger and physical health has been studied for several decades, with consistent evidence linking anger to various health risks. The following section reviews two primary lines of research, focusing on cardiovascular disease (CVD) and pain.

Anger and Cardiovascular Disease

Cardiovascular disease (CVD) refers to a heterogeneous set of medical conditions affecting the heart and vascular system, including, for example, coronary heart disease, heart failure, and stroke. CVD is a leading cause of morbidity and mortality worldwide, with well-established risk factors such as age, hypertension (i.e., high blood pressure), smoking, obesity, and diabetes (World Health Organization, 2025). Beyond these traditional risk factors, the general tendency to experience negative emotions including anger, anxiety, and depression-related emotional states, have been recognized as contributors to cardiovascular risk (Suls & Bunde, 2005).

Indeed, a large body of prospective and meta-analytic investigations have largely supported a modest but reliable association between trait anger/hostility and various CVD-related events (Chida & Steptoe, 2009; Miller et al., 1996; Suls, 2013, 2018; Suls & Bunde, 2005; Titova et al., 2022; Williams, 2010). Importantly, anger may function both as a *long-term vulnerability factor* and as an *acute trigger* of cardiovascular events. Specifically, meta-analytic evidence shows that risk is elevated in the two hours following an anger outburst (i.e., an acute trigger; Mostofsky et al., 2014), and that higher trait anger and hostility are associated with approximately 20–25% increased risk of coronary heart disease and poorer prognosis among those with existing disease (Chida & Steptoe, 2009).

However, whether anger confers cardiovascular risk through a distinct pathway remains an open empirical question. Findings suggest substantial overlap with other emotional states, such as anxiety and depression, as well as broader constructs like negative affectivity (Suls & Bunde, 2005). Consistent with this, in a more recent review it was concluded that the predictive role of trait anger and hostility in CVD risk may be limited when accounting for other emotional states (e.g., anxiety and depression; Suls, 2018).

When it comes to the link between the style of how anger is expressed and its link to CVD risk, findings are a bit mixed. Building on early psychodynamic models of the cathartic effects of anger, it was originally proposed that outward expression of anger (anger-out) might be

protective and reduce CVD risk, whereas habitual suppression of anger (anger-in) would increase risk (Linden et al., 2003). Empirical evidence, however, challenges this view: both anger-in and anger-out have been associated with elevated CVD risk (Williams, 2010). For example, in a review it was concluded that Type D personality—characterized by social inhibition and negative affectivity, which closely parallels the anger-in construct—is an established independent risk marker in patients with coronary heart disease (CHD), and is prospectively associated with poorer clinical and patient-reported outcomes (Kupper & Denollet, 2018).

One possibility is that anger-out and anger-in represent extreme styles of anger expression, whereas more constructive or regulated expressions may reduce risk (Linden et al., 2003). Alternatively, as pointed out by Williams (2010, p. 444) it may not be the style of expression per se that determines adverse outcomes, but rather the "…mere experience of intense physiological arousal associated with anger…" , highlighting the importance of effective regulation of anger-related arousal in mitigating CVD risk.

Anger and Pain

The association between anger and pain has long attracted research attention (Fernandez & Turk, 1995). Pain may indeed both elicit anger and be influenced by it. Recent meta-analytic evidence, for example, indicates that anger is associated with both greater pain intensity and pain-related disability (Adachi et al., 2022). These associations have been observed for both experimentally induced state anger—also when controlling for related emotional states such as anxiety and depression (e.g., Burns, 2006)— and self-reported state anger in cross-sectional studies, with similar patterns observed for trait anger (Adachi et al., 2022)

When considering how anger is expressed, the evidence is more mixed. Review findings suggest that anger-out tendencies are associated with increased responsiveness to both experimentally induced and clinical pain, as well as greater chronic pain intensity (i.e., pain that lasts more than 3 months; Bruehl et al., 2006). In contrast, the temporary expression of anger at the state level appears to have short-

term pain-inhibitory effects, possibly by reducing acute physiological tension related with anger (Burns, 2006).

State anger suppression, by contrast, appears to result in increased tension and pain. Experimental studies by Burns and colleagues, for instance, have shown that healthy pain-free participants instructed to suppress anger during provocation subsequently exhibited lower pain tolerance, higher pain ratings in a cold pressor task, and increased accessibility of anger-related thoughts compared to non-suppression control (Quartana & Burns, 2007; Quartana et al., 2007). Similar effects have also been observed among chronic back pain patients, with the largest pain increases in individuals predisposed to anger-out—suggesting that expressing anger contrary to one's habitual style may intensify pain (Burns et al., 2011). Interestingly, these effects also appear to extend beyond laboratory settings. For example, expressive suppression of anger has been associated with both concurrent and lagged increases in pain interference (up to three hours later), even after controlling for concurrent and lagged state anger, suggesting that behavioral suppression of anger, in itself, may have a unique detrimental effect on pain (Burns et al., 2015).

Evidence regarding trait anger-in, measured via Spielberger's Anger-In scale, is less clear. Burns et al. (2008a) argue that associations with pain and anger-in may reflect general negative affectivity rather than a unique effect of suppression. Under this view, individuals high in anger-in may be more likely to attend to and amplify pain due to heightened distress, rather than due to suppression per se.

Anger and Interpersonal Functioning

Elevated levels of trait anger and anger-out are associated with a range of social costs that extend beyond the direct harm caused by aggression or violence. Meta-analytic evidence indicates that individuals prone to expressing anger report poorer social well-being (d = –.14), including lower perceived social support, reduced liking by acquaintances and strangers, and diminished relationship satisfaction and romantic quality (Chervonsky & Hunt, 2017). Similar findings are observed in occupational contexts, where frequent anger expression is often viewed as inappropriate or indicative of poor self-control,

undermining perceived competence, social status, and workplace relationships (Porat & Levy Paluck, 2024).

When it comes to the suppression of anger, the picture appears more complex. Here it becomes important to once again separate between the trait tendency for anger-in, which involves habitual suppression of anger, and state anger suppression. For example, across seven experimental studies (N = 2,613), participants forming impressions of individuals who either suppressed or expressed anger in various contexts (e.g., workplace scenarios) judged those who suppressed anger as warmer and more competent than those who expressed it openly (Green et al., 2025). At the same time, other research points to interpersonal costs of state suppression, such as disrupted communication (Butler et al., 2003), and reduced performance in negotiations tasks (Shao et al., 2015).

More consistently problematic, however, is the habitual tendency to suppress emotions, which is even more strongly associated with poorer social well-being (d = –.20) than tendencies to express anger (d = –.14). Importantly, research suggest that the social effects of suppression may be culturally bound, such that individuals with western-European values may experience stronger negative effects from habitual- and state suppression than, for example, individuals with Asian values, where negative social effects may be reduced (e.g., Butler et al., 2007).

Anger and Comorbidity

Elevated levels of anger and aggression have been associated with a wide range of psychological disorders, ranging from various externalizing (Fernandez & Johnson, 2016) and internalizing disorders (Cassiello-Robbins & Barlow, 2016) to personality-, psychotic- and neurodevelopmental disorders (Novaco, 2010), making it a transdiagnostic problem. In the DSM-5, for example, anger and its variants (e.g., irritability, temper outbursts and hostility) constitute central diagnostic criteria in five disorders (one of which have already been discussed): Intermittent Explosive Disorder (IED), Oppositional Defiant Disorder (ODD), Disruptive Mood Dysregulation Disorder (DMDD), Borderline Personality Disorder (BPD), and the manic phase of

Bipolar Disorder, where disproportionate anger or irritability is explicitly part of the diagnostic criteria (Fernandez & Johnson, 2016).

Elevated anger and irritability are also observed as subsidiary symptoms across a range of emotional disorders (Cassiello-Robbins & Barlow, 2016). This is perhaps unsurprising given the close relationship between anger and fear/anxiety, both of which are linked to heightened threat sensitivity (Novaco, 2010; Schultz et al., 2009). For example, while panic attacks (i.e., sudden episodes of intense fear) are well-known features of several emotional disorders (e.g., panic disorder), so called "*anger attacks*", defined as sudden episodes of intense anger accompanied by marked autonomic arousal (Fava & Rosenbaum, 1999) are also common. Estimates suggest that approximately one-third to two-thirds of individuals with depression report such attacks, with similar rates observed in anxiety disorders (Fava & Rosenbaum, 1999; Gould et al., 1996).

When it comes to expressions of anger, both anger-out and anger-in tendencies are elevated across a range of disorders. For instance, reports of both elevated anger-out and anger-in tendencies have been found among individuals with depression (for a review, see Novaco, 2010), with similar findings also reported for post-traumatic stress disorder (PTSD), although anger-in is more strongly associated with PTSD than anger-out (Orth & Wieland, 2006). A notable exception appears to be social anxiety disorder, where anger-in tendencies may be elevated while anger-out remains low (e.g., Tsekova et al., 2026; Versella et al., 2016), likely reflecting heightened concerns about social evaluation and fear of interpersonal conflict.

Importantly, a large body of evidence suggests that elevations in anger and anger expressions are strongly linked to poorer outcomes across psychological disorders. As noted by Fernandez (2016) and Cassiello-Robbins and Barlow (2016) individuals with elevated anger in the context of a psychiatric disorder tend to experience greater symptom severity, longer disorder duration, higher comorbidity, poorer treatment response, and reduced quality of life—even when controlling for alternative explanations such as demographic factors or pre-existing psychological disorders.

Etiology of Maladaptive anger

Describing the etiology of maladaptive anger is challenging given the lack of a consensus definition. Nevertheless, research on elevated anger and aggression provides important insights. Drawing on this literature and theories of maladaptive anger and aggression (Allen et al., 2018; Gardner & Moore, 2008; Roberton et al., 2012; Wilkowski & Robinson, 2010), the present part outlines key biological and environmental predispositions (*predisposing factors*), as well as within-person *proximal mechanisms* that contribute to the development and maintenance of maladaptive anger.

Predisposing Factors

Individual differences in the propensity to respond with anger-like behaviors have been observed as early as 2–8 months of age (Lewis et al., 1992). Although these differences evolve over time, they show increasing stability across development (Kim et al., 2010). Consistent with this, genetic studies indicate a moderate heritable influence on both state and trait anger, as well as anger-out tendencies (Alia-Klein et al., 2020). Parallel findings have been reported for aggression, which shows substantial stability from childhood into adulthood (Huesmann et al., 1984), and increasing genetic influence over time (Koyama et al., 2024).

Biological Vulnerabilities Contributing to Trait Anger and Aggression

One specific factor that may contribute to heightened anger and aggression is individual differences in approach motivation. Across several studies, Harmon-Jones and colleagues have, for example, shown that state anger is linked to greater relative left anterior cortical activation—a neural pattern typically associated with approach motivation (Carver & Harmon-Jones, 2009). Moreover, individuals high in trait anger exhibit greater left frontal activity both at rest and in response to anger-inducing stimuli suggesting heightened sensitivity within neural systems supporting approach motivation (Harmon-Jones, 2004, 2007). Self-report research converges in showing that anger is associated with self-reported approach tendencies as measured by the

Behavioral Inhibition System/Behavioral Activation System (BIS/BAS) self-report scales (Carver & Harmon-Jones, 2009; Harmon-Jones, 2004, 2007). In contrast, the Behavioral Inhibition System (BIS) shows weak or inconsistent associations with trait anger and aggression once shared variance with fear is accounted for.

Additional vulnerability factors, relevant for trait anger and aggression, include, but are not limited to: reduced effortful control, heightened emotional reactivity and impulsivity, and increased reward sensitivity, which may amplify the reinforcing properties of aggressive responding (Megías-Robles et al., 2022; Romero-Martinez et al., 2022; Wilkowski & Robinson, 2010).

Environmental Vulnerabilities Contributing to Trait Anger and Aggression

Environmental factors, particularly emotion socialization processes and early adverse experiences, also play a central role (e.g., Blair, 2024; Pollak & Kistler, 2002; Smith & Pollak, 2020). How caregivers and close others respond to, model, and regulate emotions helps shape foundational ER abilities (Bjureberg, 2026). For instance, invalidation or punishment of emotional expression may foster low distress tolerance and nonacceptance of emotions such as anger. Alternatively, or in addition, exposure to caregivers who respond to anger with verbal or physical aggression may model and reinforce similar emotion–behavior patterns in the child (Allen et al., 2018). In interaction with biological vulnerabilities (e.g., heightened emotional reactivity and approach motivation), such environments may undermine the development of emotional clarity, acceptance, and adaptive regulation, increasing the likelihood of impulsive or avoidant responses to anger (Gardner & Moore, 2008; Röll et al., 2012).

Early adverse events are of particular concern. Illustrating this, Blair (2024) reviews evidence showing that sufficiently intense or chronic stressors can sensitize threat-responsive neural systems, lowering thresholds for anger and reactive aggression and biasing attention toward threat cues (Schultz et al., 2009). However, in contrast to fear-prone individuals, temperamental differences in approach motivation likely interact with these environmental risk factors, such that the

individuals with higher tendencies for approach respond with anger and aggression rather than fear and avoidance. Importantly, chronic early life stressors—especially those involving witnessing or being a victim to violence (Fazel et al., 2018)—can fundamentally alter the developmental sensitivity of the "threat-circuitry", with effects that persist into adulthood (Blair, 2024).

Differences in Vulnerability Factors Between Anger-out and Anger-In

Whether individuals with primary anger-out versus anger-in problems follow distinct developmental trajectories—and whether they are shaped by differential distal risk factors—remains largely unexplored. Research to date does, however, suggest meaningful differences in dispositional correlates. Whereas anger-out is primarily associated with externalizing traits such as impulsivity, and heightened approach motivation (Carver & Harmon-Jones, 2009; Cooper et al., 2008; Smits & Kuppens, 2005) anger-in is more strongly associated with internalizing traits including neuroticism, negative affect, and withdrawal (Cooper et al., 2008; Martin & Watson, 1997; Smits & Kuppens, 2005; Stewart et al., 2008). Neuroimaging studies further suggest potential differences, where anger-out has been associated with more left-frontal activation, typically reflecting approach-oriented motivation, whereas anger-in has been linked to right-frontal activation, indicating withdrawal tendencies (Consolini et al., 2022). However, other research show that anger-expression style (anger-out versus anger-in) does not moderate the association between trait anger and left-frontal asymmetry during rest (Stewart et al., 2008). This might suggest that anger-in and anger-out profiles show similar neurological profiles (left-frontal activation) when trait anger levels are high.

Taken together, these findings highlight the interplay of biological predispositions and environmental experiences in shaping anger-related tendencies, and underscore the need for further research on factors differentiating anger-in and anger-out pathways.

Proximal Mechanisms

Cognitive Biases

Although the terms anger and hostility at times have been used interchangeably (Eckhardt et al., 2004), *hostility* is now more commonly defined as a cognitive–attitudinal predisposition to evaluate others in negative and antagonistic ways, thereby increasing vulnerability to anger. More specifically, it encompasses tendencies such as cynicism, mistrust, and denigration of other (Miller et al., 1996). According to several influential theories on maladaptive anger and aggression (Allen et al., 2018; Deffenbacher, 2011; Dodge, 2006; Gardner & Moore, 2008; Wilkowski & Robinson, 2010), individuals prone to anger and aggression are posited to have an increased tendency for perceiving, interpreting, and attributing hostile intent to ambiguous or seemingly neutral social cues (also known as *hostility biases*). These hostility biases may include a tendency to attribute hostile intent to others' ambiguous actions (hostile attribution bias), to interpret social interactions as antagonistic (interpretation bias), to perceive ambiguous cues as threatening or provocative (perceptual bias), and to expect others to respond with hostility during conflict (expectancy bias) (Smeijers et al., 2019). While these biases likely arise from interactions between biological and environmental factors, early negative experiences have been identified as a key predisposing factor, where heightened vigilance for threat may predispose the individual to perceive ambiguous cues as hostile (Crick & Dodge, 1994; Dodge, 2006).

Empirical findings across diverse populations—including adults and children in clinical- (e.g., Lobbestael et al., 2013) and non-clinical samples (e.g., Coccaro et al., 2009)—consistently support associations between hostility biases and aggressive behavior, typically of small-to-moderate magnitude (De Castro et al., 2002; Smeijers et al., 2019; Tuente et al., 2019). Importantly, these biases appear to operate within a reciprocal feedback loop. Individuals high in trait anger are more likely to perceive ambiguous social cues as hostile (Wingrove & Bond, 2005), which increases the likelihood of aggressive responding (Tuente et al., 2019). Aggressive responding, in turn, may escalate interpersonal conflict and hostility from others, and as such reinforcing future hostility biases.

Although research on the hostility-anger link has primarily focused on aggression (e.g., anger-out), similar mechanisms are likely to operate for anger-in individuals. Indeed, hostility biases reflect appraisals of others as antagonistic, unfair, or threatening—appraisals that are likely to elicit anger regardless of whether it is expressed outwardly (anger-out) or inwardly (anger-in; Berkowitz, 2009). Consistent with this, correlational work show associations between hostility and anger-in (Vella & Friedman, 2009). Similarly, across four experimental studies, DeWall et al. (2010) found that social anxiety symptoms were associated with heightened feelings of hostility and perceptions of others as hostile, but with lower levels of aggressive behavior—a pattern resembling maladaptive anger inhibition (anger-in).

Anger Dysregulation

Once a situation has been appraised as hostile and anger has been elicited, anger dysregulation constitutes a second proximal mechanism in the development and maintenance of maladaptive anger. According to Roberton et al. (2012) anger dysregulation can be broadly conceptualized as involving *underregulation* and *overregulation.*

Underregulation is thought to occur when an individual fails to sufficiently downregulate an emotional experience such as to be able to continue on with relevant goal-directed behavior or avoid impulsive action (Roberton et al., 2012). This may reflect both limited ability to identify and label emotional states and reduced capacity to modulate their intensity once activated. For example, an individual may not notice rising anger until it becomes highly intense, at which point it becomes increasingly difficult to reappraise the situation, ultimately resulting in a loss of control and an impulsive outburst. Consistent with this account, meta-analytic evidence indicates that emotion regulation difficulties—a construct closely related to underregulation—are associated with increased aggressive behavior, particularly verbal and physical aggression (Smith et al., 2026). Similarly, difficulties in identifying and accepting emotions (i.e., alexithymia) have been linked to heightened aggression, partly through their association with emotion regulation deficits (i.e., underregulation) and impulsivity, in both offender and community samples (Garofalo et al., 2018).

Overregulation, in contrast, involves rigid or excessive attempts to regulate, control or avoid the emotional experience, either behaviorally (e.g., withdrawing from the situation) or cognitively, using strategies such as anger suppression and rumination (Denson, 2013). While anger suppression is addressed in detail in Section III, anger rumination warrants some further consideration here as it is often considered one of the key mechanisms contributing to maladaptive anger.

Anger rumination is defined as the tendency to engage in repetitive thinking about personally meaningful anger-eliciting events (Denson, 2013). More specifically, Sukhodolsky et al. (2001) describe anger rumination as involving recurrent attention to past anger episodes (i.e., angry memories), which can re-evoke state anger in the moment. During such episodes, individuals may dwell on the causes and consequences of the anger experience and mentally re-enact aspects of the event, thereby intensifying and prolonging current anger. Moreover, unlike depressive rumination, which is primarily retrospective, anger rumination may also include a forward-looking component, such as counterfactual thinking of what one would have liked to do differently in the situation (e.g., fantasizing about revenge or retaliation), which potentially may increase current aggressive inclinations (Sukhodolsky et al., 2001).

Indeed, several influential theories on aggression identify anger rumination as a key proximal mechanism (Denson, 2013; Gardner & Moore, 2008; Wilkowski & Robinson, 2010), contributing to aggression by amplifying and sustaining anger and reducing self-regulatory capacity. Experimental evidence supports this account, showing that anger rumination increases both the intensity and duration of anger and impairs subsequent self-control (e.g., Bushman, 2002; Denson et al., 2011b; Fabiansson et al., 2012; Ray et al., 2008). In addition, meta-analyses and reviews on correlational studies of anger rumination as a trait indicates that anger rumination is among the emotion regulation strategies most strongly associated with both anger (Pop et al., 2025) and aggression (Anestis et al., 2009; Fabiansson et al., 2012; Navas-Casado et al., 2023). Similarly, as will be noted in section III, anger rumination has been suggested as a key mechanism in maladaptive anger inhibition (Burns et al., 2008a).

Learning and Habit Formation

A third proximal mechanism involves reinforcement learning and the formation of automatic habits. According to Gardner and Moore's (2008) model of maladaptive anger, the core issue is not anger intensity itself but the chronic and inflexible use of strategies to avoid or escape anger. In individuals prone to reactive aggression, acts of aggression can serve to terminate aversive situations—e.g., by shouting or slamming a door—thereby escaping unpleasant emotions, which negatively reinforces the behavior. Moreover, although aggression may be later followed by guilt or shame, the *immediate* experience of power or control when aggressing may have positively reinforcing qualities, particularly for individuals high in trait anger (Megías-Robles et al., 2022; Terburg et al., 2012). Over time, these reinforcement contingencies may consolidate aggression as a habitual, automatic response to anger.

A parallel process may occur for individuals with anger-in tendencies. State suppression of anger-related expressions or experiences may become habitual when it leads to the avoidance of conflicts and "unwanted" mental content. Similarly, anger rumination may be maintained because it temporarily diverts attention from the immediate emotional discomfort (negative reinforcement) and, for some individuals, provides a transient sense of cognitive control (positive reinforcement). In this way, both suppression and rumination can develop into automatic, habitual strategies for regulating anger.

Together, these three proximal mechanisms—cognitive biases, anger dysregulation, and reinforcement learning—probably interact to sustain and escalate maladaptive anger. Hostility biases increase the likelihood that situations are appraised as threatening and hostile, dysregulated responses undermine adaptive emotion regulation, and reinforcement contingences make maladaptive responses more automatic and habitual over time.

Anger Treatment

As noted by several prominent researchers in the field of anger, the development and empirical evaluation of treatments for maladaptive anger have historically lagged behind that of other emotional disorders, such as anxiety and depression disorders (DiGiuseppe et al., 2014; Gardner & Moore, 2013). To illustrate this disparity, Cuijpers et al. (2023) recently reviewed 118 meta-analyses of psychological treatments for depression from January 2022 and backwards, whereas the most recent review of meta-analyses for anger-treatments identified only 13 meta-analyses (Lee & DiGiuseppe, 2018). Moreover, effect sizes for anxiety and depression disorders are typically large in magnitude in comparison to control (Cuijpers et al., 2025), whereas they tend to be moderate, on average, for anger (Lee & DiGiuseppe, 2018).

Anger treatment may include both pharmacological and psychological treatments. Pharmacological treatments have included, for example, the use of serotonin reuptake inhibitors (SSRIs) or so called "antidepressants", such as sertraline and fluoxetine that have been administered to patients with IED, with some evidence for its effectiveness in reducing anger intensity and irritability (Liu et al., 2025). In the present thesis, however, the focus is primarily on psychological treatments for anger.

Cognitive and Behavioral Treatments for Maladaptive Anger

The most established and empirically supported treatment for maladaptive anger is cognitive-behavioral therapy (CBT; Lee & DiGiuseppe, 2018). Most CBT protocols for anger were originally adapted from established treatments for anxiety and depressive disorders. Most notably, in pioneering work, Novaco (1977) applied Stress Inoculation Training (SIT) to anger problems, conceptualizing anger as a stress response that could be modified through skills training. Within this anger protocol, "inoculation" refers to the systematic and graduated exposure to anger-provoking situations combined with the acquisition and rehearsal of coping strategies, including cognitive restructuring (i.e., the identification and modification of maladaptive thought patterns that contribute to anger) and relaxation-based

arousal regulation, to enhance self-control before and during high-risk anger situations.

More specifically, Novaco's (1977) SIT-based anger treatment is structured into three phases. First, a *cognitive preparation phase* where focus is on identifying the client's anger triggers and typical responses to anger as well as providing psychoeducation on anger (e.g., distinguishing between adaptive and maladaptive responses to anger and differentiating it from aggression). Second, a *skill acquisition and rehearsal phase* introduces key anger management techniques, including cognitive restructuring, relaxation techniques (e.g., progressive muscle relaxation, controlled breathing, and relaxation imagery), and behavioral skills such as practicing effective communication of emotions, problem-solving, and assertiveness training (i.e., expressing needs without being aggressive or passive). Third, and last*, an application phase* is initiated which focuses on applying the learnt skills in a structured and safe format, using increasingly challenging scenarios (e.g., via imagery or role-play) to approximate real-life anger-provoking situations.

Subsequent CBT protocols have often followed a similar overall structure as In Novaco's SIT-based anger treatment, typically targeting one or more of these core anger management skills—cognitive restructuring, relaxation training, and behavioral skills training—either in combination (e.g., cognitive restructuring and relaxation training) or, as stand-alone treatments (e.g., relaxation training).

Treatment Efficacy

In their review of meta-analyses on anger and aggression treatments, Lee and DiGiuseppe (2018) identified 13 meta-analyses focusing on treatments for maladaptive anger, of which 8 focused exclusively on different variations of CBT. Studies included in these meta-analyses spanned diverse populations, ranging from children and adolescents to non-clinical and clinical adults (e.g., incarcerated adults or adults with intellectual disabilities), as well as different modalities of treatment (e.g., school-based, group, and individual interventions). Overall, and as previously stated, Lee and DiGiuseppe (2018) found that anger treatments were at least moderately effective across these

various populations and treatment modalities (e.g., d = .71; DiGiuseppe & Tafrate, 2003).

However, there appears to be some variation in efficacy across treatment modalities and anger-related outcomes. For example, some evidence suggests that relaxation training alone may yield particularly strong effects, with large effect sizes (*d* = .82–.90), although these estimates are based on a limited number of studies (Del Vecchio & O'Leary, 2004; Saini, 2009). Moreover, a closer examination of Del Vecchio and O'Leary's (2004) meta-analysis—which focused specifically on adult outpatient populations not previously incarcerated, which a population that most closely resembles the samples targeted in the present thesis—suggests differential effects across anger components. In particular, Del Vecchio & O'Leary (2004) found that cognitive-focused interventions was the most effective treatment type for anger-in (*d* = .64, based on three studies), whereas combined CBT protocols were the most effective treatments for anger-out (*d* = .61; Del Vecchio & O'Leary, 2004). However, as already noted the available treatment studies on anger are scarce and more research and treatment development is clearly needed.

Emotion Regulation Treatment for Maladaptive Anger

While CBT appears at least moderately effective, little is known of which treatment techniques or underlying mechanisms that give rise to positive results (Lee & DiGiuseppe, 2018). One potential mechanism that has recently gained attention in the treatment literature of maladaptive anger is emotion regulation. Indeed, as noted previously, emotion regulation, or more correctly emotion dysregulation, appears to bey a key process involved in the development and maintenance of anger and aggression (e.g., Pop et al., 2025; Smith et al., 2026)

Conversely, the capacity to regulate emotions effectively and adaptively may represent a key pathway for reducing anger-related difficulties (e.g., Gardner & Moore, 2008; Roberton et al., 2012). For example, experimental studies across diverse paradigms indicate that the use of putatively adaptive emotion regulation strategies and abilities—such as cognitive reappraisal and mindfulness-based skills—is associated with reductions in anger and aggression (e.g., Denson et

al., 2011a; O'Dean et al., 2025; Szasz et al., 2011). Corroborating these findings, meta-analytic evidence from cross-sectional studies indicates that habitual use of emotion regulation strategies such as cognitive reappraisal and acceptance is consistently associated with lower levels of anger (Pop et al., 2025) and aggression (Smith et al., 2026).

Building on the notion of emotion regulation as a key mechanism, several treatments protocols have explicitly begun to target emotion regulation skills in treatment (e.g., Eifert et al., 2006; Gardner & Moore, 2013; Kolts, 2022). For example, in Gardner and Moore's Contextual Anger Regulation Therapy (CART; Gardner & Moore, 2013), clients are guided to shift from rigid and inflexible reliance on cognitive and behavioral avoidance strategies toward a more mindful, accepting, and curious engagement with anger. Similarly, therapies such as dialectical behavior therapy (DBT; Linehan, 2014) and emotion-focused treatments (Paivio & Carriere, 2014; Pascual-Leone et al., 2013) have long emphasized emotion regulation as a central mechanism of change. These treatments typically involve helping clients downregulate intense emotional arousal, thereby enabling more adaptive engagement with and acceptance of emotional experiences, rather than impulsive reactions. While studies on emotion-focused therapies are scarce (e.g., Akbari et al., 2025), accumulating evidence suggests that DBT may have beneficial effects on anger (for a review, see Ciesinski et al., 2022), even though anger is rarely the primary target of DBT.

In addition, a small but emerging set of randomized controlled trials provides preliminary support for the that emotion regulation focused treatments can lead to meaningful reductions in anger and aggression (Bjureberg et al., 2023; Cassiello-Robbins et al., 2018; Hesser et al., 2017; Hesser et al., 2026; Neacsiu et al., 2018).

Internet Interventions

In parallel with advances in treatment development, there has been growing interest in internet-delivered psychological interventions across a range of mental health conditions (Hedman-Lagerlöf et al., 2023). Internet-delivered cognitive-behavioral therapy (iCBT) typically adapts core CBT components into structured, often text-based

modules that can be delivered with or without therapist guidance. Such formats offer several advantages, including increased accessibility, scalability, and cost-effectiveness, which may be particularly beneficial for individuals who face barriers to traditional face-to-face therapy (e.g., stigma, geographical constraints, or avoidance of interpersonal contact due to anger or anxiety).

A substantial body of research indicates that therapist supported iCBT is generally effective, with outcomes comparable to face-to-face CBT for several common mental health problems (for a review and meta-analysis, see Hedman-Lagerlöf et al., 2023). However, the application of internet-delivered interventions to maladaptive anger remains relatively underexplored. Existing studies suggest that iCBT may be a promising format for delivering interventions targeting anger (Hesser et al., 2026; Howie & Malouff, 2014) and aggression (e.g., treatments for intimate partner violence; Braithwaite & Fincham, 2009, 2011; Hesser et al., 2017). This also includes emerging work, from our research team, that focuses on emotion regulation focused iCBT for anger (Bjureberg et al., 2023; Hesser et al., 2026) and aggression (Hesser et al., 2017). Nevertheless, the overall evidence base remains limited.

Section III: Maladaptive Anger Inhibition

In this final section, the historical and conceptual foundations of the anger-in construct are examined. A theoretical account of the potential mechanisms underlying maladaptive anger inhibition is then introduced. Finally, two key research gaps relevant to the present thesis are outlined before presenting its aims.

A Conceptual Overview

The concept of maladaptive anger inhibition, along with related constructs such as anger suppression, anger repression, and anger-in, have been defined in multiple ways (Burns et al., 2008), contributing to substantial conceptual confusion. In an attempt to clarify some of these ambiguities, I review key definitions that have appeared over the years.

Suppressed Anger Directed Inward

Anger Repression

Tracing back to the writings of Freud and his notion of the strangulated affect, the suppression of emotions such as anger has long been theorized to lead to adverse health consequences. By suppressing, or more accurately by repressing feelings of anger, Freud hypothesized that the repressed anger would be redirected inward, at the self which would eventually take the form of depressive self-criticism and hate (Freud, 1917). Alternatively, the repressed anger may return in the form of psychosomatic symptoms such as headaches, digestive issues, or even chronic pain (Burns et al., 2008a). Importantly, repression was defined as an *unconscious* defense mechanism that prevents unwanted anger from entering immediate awareness (for a detailed overview of the construct of repression, see Erdelyi, 2006).

Although Freud's theories regarding anger repression have been criticized and the empirical support is limited (Novaco, 2014), these theories were among the first to suggest that suppression of emotions may produce some form of *rebound effect*, whereby the repressed emotions return in some way or form (Wegner, 1994). Early psychosomatic

accounts by Alexander (1939) illustrated some of these ideas through clinical observation, suggesting, for example, that "the early fluctuating phase of essential hypertension is the manifestation of a psychoneurotic condition based on excessive and *inhibited hostile impulses* [italics added]" (p. 178). Similarly, Engel (1959), proposed that inhibited aggressive tendencies may instead be experienced as somatic pain. However, these early attempts to link anger repression to physical symptoms were largely based on clinical observation and case material and generally lacked systematic methodology and clear operational definitions of anger repression.

More contemporary research has operationalized anger repression indirectly by identifying "anger repressors" as individuals high in social desirability and low in self-reported anger or anger-out, but who nevertheless may show indications of anger arousal upon provocation. For example, Burns et al. (1999), who used a similar classification, found that high repressors reported anger levels similar to low anger expressors following provocation, but exhibited heart rate increases comparable to individuals high in anger expression. However, it is still difficult to conclusively assert that this arousal is an actual representation of repressed anger rather some different physiological reaction (e.g., anxiety-related arousal). As DiGiuseppe and Tafrate (2007, p. 280) notes "perhaps true repression does exist but as of yet remains outside the grasp of scientific psychology". Nonetheless, suppression likely varies in automaticity and awareness, ranging from more deliberate acts of suppression to more automatic and unconscious.

Self-Directed Anger

Although the anger repression construct has received little empirical support, the phenomenon of self-directed anger (also known as self-directed hostility), that was implied in Freud´s theory of depression, remains comparably well-documented (Caine et al., 1967; Ellsworth & Tong, 2006; Funkenstein et al., 1954). Early prevalence studies suggest that up to 10 % of anger episodes may be self-directed (Averill, 1982). These episodes of self-directed anger may range from minor episodes of self-blame, such as getting angry at oneself for repeatedly

losing a game, to more severe cases of intense self-criticism and even self-hate (Pascual-Leone et al., 2013).

Whether self-directed anger could be better understood as instances of guilt or shame has been up for discussion (e.g., Silva, 2022). However, in a seminal study by Ellsworth and Tong (2006), self-directed anger was found to differ significantly from guilt and shame. For example, while self-directed anger also entitled a sense of wrongdoing, it lacked the moral violation associated with guilt and shame (Ellsworth & Tong, 2006).

Somewhat confusingly, Spielberger's term *anger-in* suggests that suppressed anger is directed inward, echoing earlier notions of anger repression. However, this is misleading. Items on the anger-in scale (e.g., "I boil inside but do not show it") indicate that individuals high in anger-in often continue to direct anger outwardly, albeit through fantasies or covert thoughts rather than overt behavior (e.g., fantasizing about aggressive retaliation; Berkowitz, 2012). Unfortunately, this has contributed to conceptual confusion, with some authors misusing the anger-in scale to indicate a tendency to suppress anger and turn it against the self (Abi-Habib & Luyten, 2013).

To date, however, I know of no empirical study that has examined the relationship between suppression of angry expression and subsequent self-directed anger. It seems very plausible that individuals who initially suppress an angry impulse toward someone may later experience self-directed anger, thinking, for example, "Why couldn't I stand up for myself? I'm such a failure." This potential link between conscious anger suppression and later self-directed anger (i.e., Suppressed Anger Directed Inward) remains an important area for future research.

Suppressed Anger Directed Outward

Anger-In

One of the earliest empirical investigations of suppressed anger directed toward others (i.e., anger-in) was conducted by Funkenstein et al. (1954). In a laboratory study on the direction of anger,

Funkenstein et al. (1954) examined physiological responses among individuals who were classified as directing their anger toward themselves (i.e., self-directed anger), toward the experimenter without expressing it (i.e., anger-in), or primarily experiencing anxiety following a frustration task. The findings indicated that individuals with self-directed anger showed physiological patterns similar to the anxiety group, whereas "anger-ins" displayed qualitatively distinct responses.

Later on, Harburg et al. (1973) sought to examine the concept of *suppressed hostility* among white and black Americans in areas of high versus low socioecological stress. Suppressed hostility was defined as not overtly expressing anger and feeling that expressions of anger should arouse guilt. Using structured vignette-based assessments (e.g., responses to scenarios involving unfair treatment by authority figures), they indeed identified individuals who reported feeling anger but refrained from expressing it, often accompanied by guilt (i.e., suppressed hostility), who also reported elevated levels of blood pressure (Harburg et al., 1973).

These early findings laid the groundwork for Spielberger's questionnaire-based conceptualization of anger-management styles, including the development of the *anger-in* construct. Although widely used as an index of problematic anger suppression, the construct, and how it is defined within the anger expression inventory (Spielberger, 1999), has notable limitations. Burns et al. (2008), for example, argue that the anger-in scale conflates anger suppression (e.g., "I keep things in") with anger rumination (e.g., "I harbor grudges"), thereby failing to clearly distinguish between these processes. This distinction is important, as suppression may not always be followed, or accompanied, by rumination. Moreover, anger-in focuses primarily on the suppression of anger expression, neglecting other components of the anger experience (e.g., thoughts and feelings; Burns et al., 2008a; Deffenbacher, Oetting, Thwaites, et al., 1996; Linden et al., 2003).

Maladaptive Anger Inhibition

Partly in response to the limitations of the anger-in construct, Burns et al. (2008a, p. 274) proposed a theory of when suppression of anger can become problematic, which I will return to shortly, and defined

maladaptive anger inhibition as the "…suppression of thoughts, feelings and behaviors during an anger-provoking event, followed by post-event processing and the inability to inhibit rumination." This definition usefully distinguishes the immediate regulatory response—suppression—from its potential downstream consequence—rumination, and acknowledges that suppression may involve more than the suppression of overt expression. However, this definition does not address the directedness of anger. As noted in earlier work, suppressed anger may be directed either toward the self or others.

Overall, the literature reflects conceptual inconsistency. Constructs such as anger repression, suppression, suppressed hostility, and anger-in are often conflated, obscuring distinctions between regulatory processes (e.g., suppression) and their outcomes (e.g., rumination). While the present thesis will mostly rely on Spielberger's anger-in scale as a proxy for maladaptive anger inhibition, future work would benefit from more nuanced models that differentiate between types of suppression (thoughts, feelings, behaviors), direction of anger (inward vs. outward), and level of awareness (conscious vs. unconscious/automatic).

Burns' Two-Stage Theory of Maladaptive Anger Inhibition

To date, few theories have explicitly described the processes underlying maladaptive anger inhibition; Burns and colleagues' (2008a) model is a notable exception. Developed in the context of pain research, Burns et al. (2008a) suggests that maladaptive anger inhibition arises from two sequentially interacting regulatory strategies—state anger suppression and subsequent anger rumination. In the first stage, individuals respond to provocation or frustration by suppressing anger, including its expression or experiential components (thoughts and feelings), or both. Suppression is then posited to leave residual cognitive and physiological activation, reflecting incomplete downregulation and increased accessibility of anger-related content. In the second stage, the individual uses rumination, as a second emotion regulation strategy, in efforts to regulate this lingering anger. However, paradoxically, rumination instead prolongs and intensifies anger (Denson, 2013) and biases interpretations of subsequent experiences (e.g., perceiving pain more negatively; Burns et al., 2008a). Thus, the theory describes a maladaptive regulatory sequence in which two strategies, that are each associated with adverse effects, combine to produce a particularly persistent and exacerbated emotional response—a "double dose" of dysregulation (Burns et al., 2008a).

To explain why suppression may lead to lingering anger and thereby facilitate rumination, Burns and colleagues draw on three complementary lines of research: emotion regulation research (Gross, 1999), which suggests that (expressive) suppression is often ineffective in downregulating emotional experience and may even increase physiological arousal (Gross & Levenson, 1993; Richards & Gross, 1999); emotional processing theories, which posit that suppressed or avoided emotions result in "incomplete processing" that sustains emotional activation and promotes rumination (Pennebaker, 1989; Rachman, 1980); and Wegner´s (1994) ironic process theory, which proposes that suppressed thoughts may paradoxically become more accessible following suppression—a theory that Burns and colleagues particularly emphasize.

Specifically, the ironic process theory proposes that thought suppression involves two interacting processes: a deliberate *operating* process that directs attention away from the unwanted thought, and an automatic *monitoring* process that scans for its recurrence (Wegner, 1994; Wenzlaff & Wegner, 2000). Ironically, once suppression efforts cease, the monitoring process may persist, increasing the likelihood of the suppressed thought to re-emerge (i.e., a rebound effect).

Indeed, meta-analytic findings generally support small-to-moderate rebound effects following suppression, and under conditions of cognitive load, such effects may even occur *during* active suppression (so-called immediate enhancement effects; Abramowitz et al., 2001; Wang et al., 2020; Webb et al., 2012), although the robustness of rebound and immediate enhancement effects have been questioned in recent work (Niczyporuk, 2026; Purdon, 2020). In turn, rebound effects may contribute to hyperaccessability of thoughts and increase the likelihood of subsequent rumination (Gold & Wegner, 1995) or other, more adaptive, post-suppression regulation strategies (e.g., reappraisal; Burns et al., 2008b).

Empirical Gaps In the Literature

Gap 1 – Understanding Maladaptive Anger Inhibition: Anger Subtypes, Aggression Links, and Emotion Regulation

A striking feature of the literature reviewed so far is the relative lack of empirical and theoretical work on maladaptive anger inhibition compared to anger driven aggression. As noted above, the account proposed by Burns and colleagues appears to be one of the few theories explicitly describing the phenomenon of maladaptive anger inhibition and its underlying mechanisms. This stands in stark contrast to the many theories developed in relation to anger and anger-out tendencies or aggression. Accordingly, several important research gaps remain, of which I will outline a few below.

Anger Subtypes

First, despite calls to develop empirically grounded taxonomies of anger problems (Lee & DiGiuseppe, 2018), relatively few studies have examined the presence of distinct subtypes of anger problems. Moreover, existing work has been limited in two important respects. First, much of the literature has been primarily theoretical, proposing typologies based on clinical observation rather than empirical data (Megargee, 1966). Second, the small number of empirical studies that do exist have often relied on traditional cluster-analytic techniques (e.g., k-means clustering; Furlong & Smith, 1998), which, for example, are limited by a lack of statistical fit indices to guide selection of the number of subgroups and does not provide an estimates on classification uncertainty.

Maladaptive Anger Inhibition and Aggression

Second, basic descriptive knowledge of maladaptive anger inhibition remains limited. Although anger-in and anger-out are often elevated in clinical populations, little is known about the prevalence and demographic characteristics of individuals with maladaptive anger inhibition tendencies, particularly among treatment-seeking samples. Similarly, little is known of the relation between maladaptive anger inhibition and aggression as a potential consequence. As reviewed in

the previous section there are indications that “anger-ins” may experience elevations in certain forms of aggression, such as displaced aggression (e.g., Denson et al., 2006). However, studies so far have mostly been correlational, highlighting the need for experimental investigation.

Maladaptive Anger Inhibition and Emotion Regulation

Third, and most central to the present thesis is the limited understanding of how individuals with maladaptive anger inhibition regulate their anger. Spielberger´s anger-in scale is often used synonymously with anger suppression, however, as noted by Burns et al. (2008a) maladaptive anger inhibition likely reflects a broader repertoire of regulatory strategies, including, for example, both anger suppression and anger rumination. At the same time, it remains unclear to what extent individuals with maladaptive anger inhibition rely on other regulatory strategies—both putatively maladaptive and adaptive (e.g., cognitive reappraisal)—and how these strategies co-occur. Moreover, most research on emotion regulation, including anger regulation, has examined strategies in isolation both correlationally and in the lab. However, as suggested by Burns (2008b), anger suppression may be especially toxic when interacting with anger rumination, suggesting a sequential interplay between the two.

Emerging studies on the sequential interplay between emotion regulation strategies suggests that that deploying a putatively adaptive strategy following a maladaptive one can partially mitigate the negative effects of the initial strategy (e.g., Peuters et al., 2019; Yoon & Joormann, 2012). For example, Peuters et al. (2019) found that using cognitive reappraisal after rumination reduced some of the adverse effects of the preceding rumination, although the effects were modest and the initial strategy generally exerted the strongest influence. This points to the importance of examining suppression not only in relation to maladaptive strategies such as rumination, but also in relation to potentially adaptive strategies such as distraction and reappraisal.

Gap 2 - Limitations in Current Anger Treatments

The second major research gap concerns limitations in current anger treatments. As noted earlier, the development and evaluation of anger treatments have lagged behind those for other emotional disorders, and their overall efficacy is comparatively modest. Moreover, few, if any, treatments have been developed to specifically target maladaptive anger inhibition.

Translational Issues

These limitations become more apparent when considering how existing treatment approaches have been conceptualized and developed. As pointed out by DiGiuseppe et al. (2014, p. 56) "Anger treatments have not been developed from an understanding of disturbed anger" Instead, most CBT anger management protocols have been created and adapted from existing manuals for anxiety and depression. Despite sharing many features with fear and anxiety, anger differs in important ways. Notably, anger is typically approach-oriented and can involve both negatively and positively valenced experiences (e.g., anticipated relief or reward in resolving conflict). These differences suggest that standard techniques such as emotional exposure may need clear adaptation, particularly in determining whether the focus should be on exposing the patient to the aversive trigger that elicited anger or the anger experience in its whole, which may contain positively valenced and reinforcing qualities (e.g., thoughts of revenge).

Limitations of Cognitive Approaches to Anger

Beyond translational issues, Gardner and Moore (2008) argues that the field has been overly dominated by a cognitive–appraisal perspective, emphasizing the modification of dysfunctional thoughts as the primary mechanism of change. In contrast, their account highlights broader deficits in emotion regulation and avoidance learning—particularly in the understanding, tolerance, and modulation of anger—as central to maladaptive anger.

Importantly, even when targeted, cognitive strategies such as reappraisal may be difficult to implement during intense anger. Although most anger treatments attempt to address this by reducing arousal

first through, for example, relaxation training, there remains a need for treatments that more directly facilitate adaptive regulation under such conditions. One promising ability to focus on is mindful emotion awareness.

According to the mindfulness-to-meaning theory proposed by (Garland et al., 2015), mindfulness promotes a metacognitive state in which individuals observe emotional reactions without immediately elaborating on them. This decentered awareness may interrupt automatic appraisal processes, broaden attentional scope, and facilitate the generation of more adaptive reappraisals of stressful events, possibly creating positive synergistic effects between the two.

Building on these ideas, my supervisors and their colleagues (Bjureberg et al., 2023) recently developed and evaluated three emotion-regulation treatments for maladaptive anger (defined as elevated anger-in, anger-out, or both). Specifically, *N* = 234 adults received four weeks of training in either mindful emotion awareness (MEA), cognitive reappraisal (CR), or a combination of the two (MEA + CR). All three treatments led to reductions in anger outcomes relative to a prolonged baseline, but the combined MEA + CR treatment was most effective in reducing anger-out, aggression, and anger rumination, particularly among individuals with higher baseline severity. Interestingly, no differential effects were observed for anger-in, suggesting that anger-in may require distinct or additional treatment components.

Lack of Adapted Treatments for Maladaptive Anger Inhibition

Indeed, to my knowledge, only one treatment study has explicitly targeted processes conceptually related to maladaptive anger inhibition, albeit in a different clinical context (Slavin-Spenny et al., 2013). In this study, young adults (*N* = 147) experiencing recurrent headaches were randomized to either anger awareness and expression training, relaxation training, or a wait-list control condition. Both active treatments improved self-efficacy in managing headaches, but only anger awareness and expression training led to increases in emotional processing and assertiveness. However, anger and anger-in tendencies were not included as outcome variables, and the intervention was

evaluated within a somatic symptom context rather than in populations characterized by maladaptive anger inhibition per se. Accordingly, the extent to which these findings, and this treatment, generalize to maladaptive anger inhibition as a primary clinical phenomenon remains unclear.

Section IV: Thesis Aims

The overarching aim of this thesis is twofold. First, it seeks to advance our understanding of individuals who experience maladaptive anger inhibition: who they are, how they regulate anger, and which processes contribute to the development and maintenance of their difficulties. Second, the thesis aims to develop and evaluate an emotion regulation focused CBT specifically adapted for maladaptive anger inhibition. Study specific aims are outlined below.

Study I

Study I aimed to empirically derive anger subgroups among individuals seeking treatment for maladaptive anger (aim 1), and to examine differences between these subgroups in the use of emotion regulation strategies (aim 2), as well as in key demographic and clinical characteristics (aim 3).

Study II

Study II aimed to examine associations between anger components and emotion regulation strategies using a variable-centered network analytic approach. Specifically, it aimed to identify clusters of tightly interconnected anger components (aim 1) and to extend these networks by including emotion regulation variables to examine their interconnections (aim 2).

Study III

Study III aimed to experimentally study the immediate (aim 1) and delayed (aim 2) effects of anger suppression and how subsequent post-suppression regulation (continued suppression, rumination, or reappraisal) affects anger and heart rate variability (HRV) (aim 3).

Study IV

Study IV aimed to evaluate the feasibility (aim 1), acceptability (aim 2) and preliminary efficacy (aim 3) of a newly developed internet-delivered therapist-supported emotion regulation focused CBT for individuals with maladaptive anger inhibition. Treatment outcomes included anger-in (primary outcome), anger rumination, and anger assertion (secondary outcomes).

Methods

Research Design, Procedures, and Analytical Methods

Study I and II

Research Design - Studies I and II

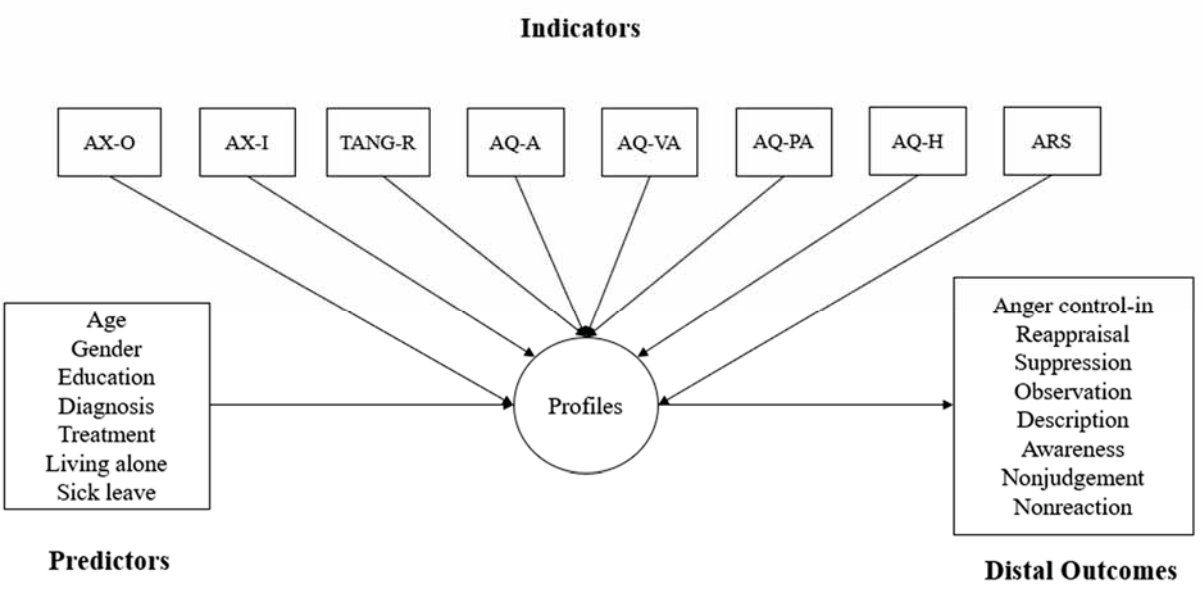
Study I and II both used cross-sectional designs with screening data from Bjureberg et al. (2023) RCT for maladaptive anger. After exclusion of participants with missing data on key measures a final sample of *N* = 538 anger-treatment-seeking adults were included in the studies.

Analytical Methods - Study I

In study I, we used a data-driven, person-centered approach—latent profile analysis (LPA)— in *M*plus version 8.7 (Muthén & Muthén, 1998-2017) to derive subgroups of maladaptive anger (aim 1). Anger components were entered as indicators, and LPA models with 1-7 profiles (i.e., subgroups) were compared to find the best fitting model. In line with recommendations, we determined model fit iteratively by comparing each model with *k* profiles to the model with *k*–1 profiles using information criteria indices (AIC, BIC, SABIC), likelihood ratio tests (e.g., Lo-Mendell-Rubin; LMR), classification quality, and profile-interpretability based on past theory (Nylund-Gibson & Choi, 2018; Nylund et al., 2007).

Subsequently, the best-fitting model was validated against demographic and clinically relevant characteristics, as well as relevant emotion regulation strategies. Demographic and clinical variables were entered as predictors using multinomial regression with the three-step approach (Asparouhov & Muthén, 2014) and emotion regulation strategies were included as distal outcomes using the Lanza method (Lanza et al., 2013). All covariates were included as auxiliary variables to avoid unintended influence on profile estimation (Asparouhov & Muthén, 2014; Nylund-Gibson & Masyn, 2016). See Figure 1 for a schematic overview of the final LPA model.

Figure 1. A Schematic Overview of The LPA Model in Study I



Note. TANG-R = Trait Angry-Reaction; AXO = Anger-Out; AX-I = Anger-In; AQH = Hostility; AQ-A = Trait Anger; AQ-VA = Verbal Aggression; AQ-PA = Physical Aggression; ARS = Anger Rumination

Analytical Methods - Study II

In study II, we used a variable-centered approach by estimating psychometric networks in *R* (version 4.3.1) to explore associations among anger and ER variables. Specifically, we estimated regularized partial correlation networks, following procedures recommended by Epskamp et al. (2018). In these networks, *nodes* represent individual variables (i.e., anger components or emotion regulation strategies), and *edges* represent partial correlations between the nodes (i.e., the correlation between nodes after controlling for all other nodes in the network). To improve interpretability and reduce the risk of identifying spurious associations (i.e., type 1 errors) among our nodes we used a regularization method called Least Absolute Shrinkage and Selection Operator with extended Bayesian information criteria (EBICglasso; Friedman et al., 2008). In short, this regularization method shrinks small partial correlations to zero, resulting in a more parsimonious and interpretable network.

Networks were estimated in three steps. First, an anger network including all anger components (as in Study I), was estimated to examine the overall structure. Exploratory graph analysis (EGA) was then applied to identify clusters of densely connected nodes (aim 1; Golino & Epskamp, 2017). Second, to examine connections between anger components and emotion regulation strategies extended networks were estimated by including the same emotion regulation strategies as in Study I into the identified cluster networks (Aim 2). Finally, an anger-regulation network including all anger components and emotion regulation strategies was estimated to examine their interactions across a combined anger-regulation network.

Centrality indices, primarily node strength, which reflects how strongly a given node is connected to all other nodes in the network, were calculated to assess the relative importance of each node. Moreover, the robustness and accuracy of networks, cluster solutions, edge weights (i.e., partial correlations), and centrality estimates were evaluated using bootstrap procedures and stability analyses (Epskamp et al., 2018).

Study III

Research Design Study - III

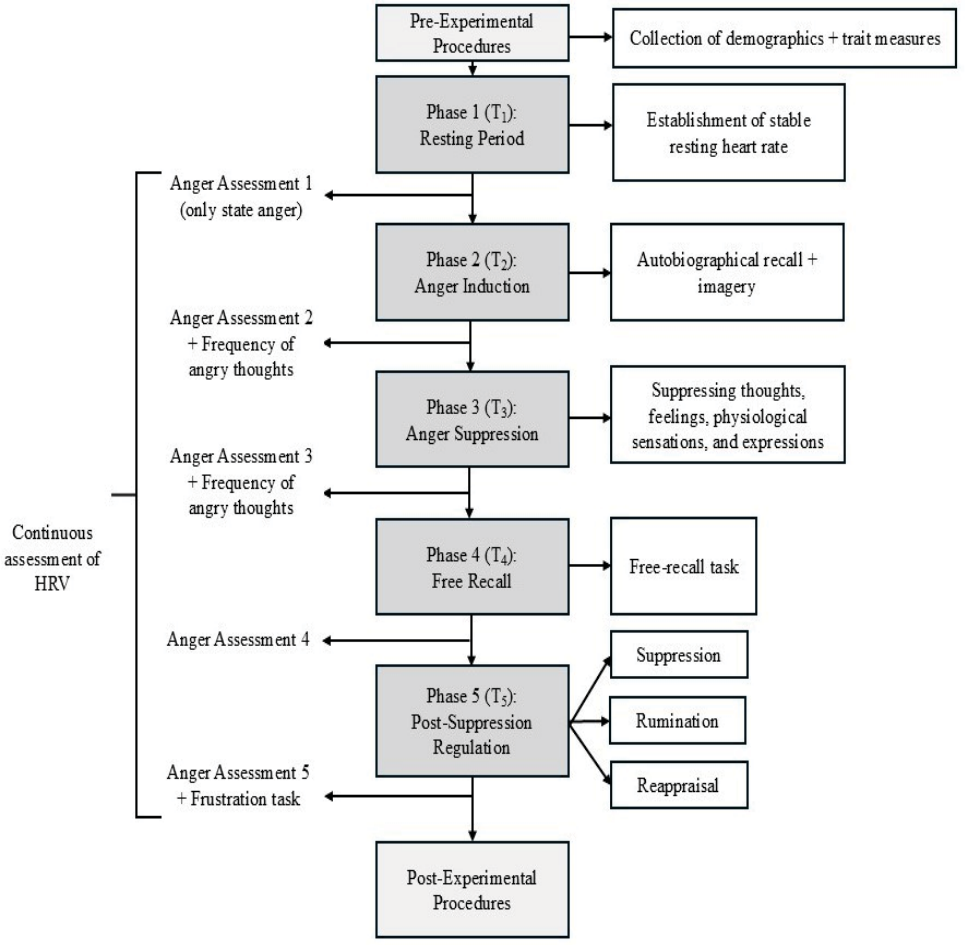
Study III employed a mixed experimental design (four within-subjects phases and final between-subjects phase) to explore the immediate (aim 1) and delayed (aim 2) effects of anger suppression as well as the effects of three post-suppression strategies (continued suppression, rumination, or reappraisal) on anger and heart rate variability (HRV; aim 3). The study sample consisted of an adult university sample. In total, *N* = 203 participants from Örebro University participated in the experiment.

Experimental Procedures - Study III

The experiment was conducted in an experimental laboratory, with all tasks and instructions presented on a local computer. The phases were: (1) a resting baseline phase, during which participants sat quietly for three minutes to establish baseline heart rate; (2) an anger induction phase, in which participants recalled and vividly reimagined a personally anger-eliciting event; (3) an anger suppression phase; (4) a free-recall phase, in which participants were instructed to think about anything, allowing for assessment of potential rebound effects; and (5) a final post-suppression regulation phase, in which participants were randomized to use suppression, rumination, or reappraisal while re-engaging with the anger memory.

Figure 2 (next page) provides an overview of the experimental phases, procedures and measures taken. In short, self-reported measures of anger were collected after each phase, heart rate and HRV were continuously monitored and a behavioral measure of frustration tolerance was collected following phase 5. Additional pre- and post-experimental measures were also collected which will be described in more detail in the coming “Data Collection” section.

Figure 2. Study III: Experimental Design Overview



Note. Experimental phases are shown in dark grey, phase-specific activities in white, and pre- and post-experimental procedures in light grey. Anger assessment = state assessment of anger, angry thoughts and problems with anger control.

Analytical Methods - Study III

Statistical analyses proceeded in several steps. First, paired-samples t-tests evaluated the effectiveness of the anger induction (phase 1 → phase 2). Second, we evaluated within-subject changes in state anger measures and HRV using linear mixed models and planned contrasts to examine immediate suppression (phase 2 → phase 3; aim 1) effects and rebound effects (phase 3 → phase 4; aim2). Between-condition differences in anger, and HRV (aim 3) during phase 5 were analyzed using ANCOVAs with Bonferroni-corrected pairwise comparisons, controlling for each outcome's corresponding Phase 2 (induction) score. Additional analyses included logistic regression for frustration tolerance, sensitivity checks for HRV, paired-sample t-test for frequency measures of angry thoughts, and manipulation checks for adherence to the post-regulation strategies (see manuscript for details).

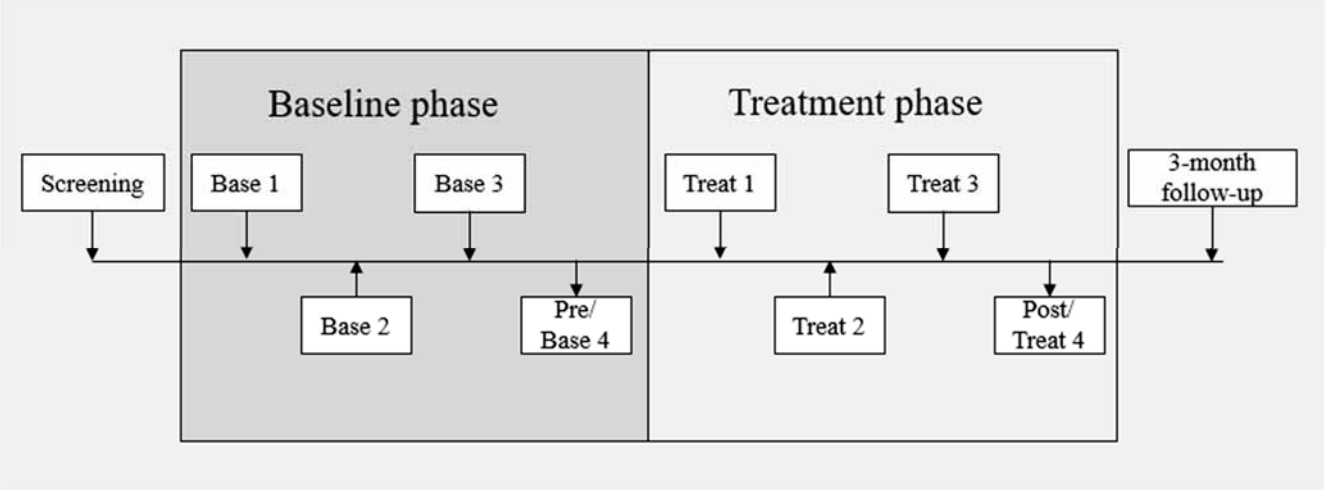
Study IV

Research Design - Study IV

Study IV employed a pooled interrupted time series design. The aim was to develop and evaluate the feasibility, acceptability, and preliminary efficacy of a newly developed internet-delivered emotion-regulation CBT for maladaptive anger inhibition. The sample consisted of *N* = 38 adults who sought treatment for maladaptive anger inhibition, defined as scoring on the upper quartile on the anger-in scale, based on community sample norms (AX-I; ≥18 points; Spielberger, 1999).

Treatment feasibility and acceptability was explored using self-report data along with tracking of study drop-out and completion of number of treatment modules, homeworks, and assessments (see "Data Collection" for details). Preliminary efficacy, were assessed by evaluating changes across primary- (anger-in) and secondary- (anger rumination and anger assertion) treatment outcomes. Specifically, treatment outcomes were assessed during screening and weekly across a baseline phase and a treatment phase, each comprising four assessment points, followed by a 3-month follow-up (10 assessments in total). Analyses were conducted at the group level by pooling observations across participants. See Figure 3 for an overview of the design.

Figure 3. Study IV: Study Design



Note. Primary- (anger-in) and secondary- (anger rumination, and anger assertion) treatment outcomes were assessed at each point of assessment

Treatment Procedures and Content - Study IV

The treatment consisted of a 4-week therapist-guided emotion regulation focused iCBT program, comprising four modules delivered weekly. Participants received continuous text-based support and feedback from an assigned therapist (either a licensed psychologist or a master's-level student under the supervision of a licensed psychologist). Each module included psychoeducation on anger and emotion regulation, audio exercises, worksheets, and both imaginary and real-life practice tasks, accompanied by weekly homework assignments.

The treatment was based on Bjureberg et al. (2023) combined emotion regulation treatment focusing on the two ER skills of mindful emotion awareness and cognitive reappraisal (MEA + CR), however with an enhanced focus on maladaptive anger inhibition, and as such named: e*nhanced Cognitive Reappraisal and Emotion Awareness Training (eCREAT).* Drawing on Burns' conceptualization of maladaptive anger inhibition as a two-stage process involving both suppression and rumination, two major adaptations were made. First, to counteract avoidance and suppression, the treatment placed greater emphasis on facilitating full emotional processing and adaptive expression of anger (e.g., assertive communication of needs) compared to the MEA + CR treatment. Second, additional strategies were incorporated to disrupt rumination, including techniques adapted from interventions targeting depressive rumination (Watkins, 2018), but adapted to an anger context (Denson, 2013). For a detailed overview of treatment content see table 1 on next page.

Table 1. *Treatment Content of the eCREAT intervention (Study IV)*

Module	Submodule	Content
I: Introduction to the Treatment	1. Introduction to internet-delivered CBT	- General treatment information - Technical details about the online treatment platform
	2. Psychoeducation on Anger	- Functions of anger and other emotions - Habitual behaviors and their role in maladaptive anger - Introduction of biopsychosocial model of anger - Identifying triggers and vulnerability factors
	3. Introduction to Mindful Emotion Awareness	- Emotion identification through mindful emotion awareness - Avoidance and counterproductive effects of avoidance (e.g., thought rebound) - Introduction to relaxation exercises - Introduction of crisis strategies (e.g., time-out) to handle extreme anger
II: Mindful Emotional Awareness and Cognitive Reappraisal		- Primary and secondary emotions - The influence of thoughts on emotional experiences - Judgmental thinking and anger - Cognitive defusion to detach from judgmental thoughts - Cultivating Compassion to counteract judgmental thinking - Emotion surfing: experiencing emotions without avoidance
III: Strategies to counteract Rumination and Express Emotional Needs		- Anger rumination: angry thoughts and fantasies directed at others and oneself - Disrupting rumination through anchoring in the present, flow, and radical acceptance - Maladaptive communication styles: avoidant, aggressive, and passive aggression - Assertive communication: a balance between goals, relationships, and self-respect. - Specific assertiveness skills (e.g., boundary setting)
IV: Relapse prevention		- Summary and repetition - Relapse prevention plan

Analytical Methods Study IV

Treatment effects were analyzed using piecewise latent growth models (LGM) in Mplus (version 8.10), modeling separate trajectories for baseline and treatment phases. Differences in level and slope between phases were used to estimate treatment-related change relative to projected baseline trends, and effect sizes were calculated at post-treatment. Missing data were handled using full information maximum likelihood with robust standard errors (MLR) under the assumption of missing at random, and analyses followed an intention-to-treat approach. Power was determined via Monte Carlo simulation, indicating a required sample of approximately 30 participants, with a target of 40 to account for a potential drop-out rate of just below 30 %.

Participants

Three distinct samples were included in the present thesis. Studies I, II, and IV comprised clinical populations of adults who either sought treatment for maladaptive anger (Studies I and II) or participated in a pilot treatment study for maladaptive anger inhibition (Study IV). Study III involved a non-clinical sample of university students and staff. All participants were recruited in Sweden. Below, the recruitment procedures and the demographic and clinical characteristics of each sample are described. Demographic and clinical characteristics for all samples can be found in Table 2 on next page.

Table 2. *Demographic and Clinical Characteristics of Study Samples*

		Study I and II (n = 538)				Study III (n = 203)				Study IV (n = 38)			
Variable		*M*	*SD*	*Range*	*n* (%)	*M*	*SD*	*Range*	*n* (%)	*M*	*SD*	*Range*	*n* (%)
Age		39.78	12.28	18-75		29.42	12.28	18-61		43.8	14.1	18-74	
Gender (female)					298 (55.4)				117 (55.4)				34 (89.5)
University Education					245 (45.5)				N/A				29 (76.3)
Living Alone					77 (14.3)				N/A				9 (23.7)
Psychological Disorder					170 (31.6)				N/A				9 (23.7)
Previous Psychological Treatment					344 (63.9)				N/A				30 (78.9)
Sick Leave					35 (6.5)				N/A				0 (0.0)
STAXI-2	Trait Anger	23.99	5.78	11-40		16.38	3.89	10-31		20.63	4.02	13-28	
	Anger-Out	19.38	4.39	9–32		N/A	N/A	N/A		13.68	3.27	8-19	
	Anger-In	21.15	4.77	8–32		18.51	4.97	9-31		25.50	3.99	18-31	
ARS	Anger Rumination	39.78	12.28	19–76		N/A	N/A	N/A		48.63	11.73	27-73	
AQ	Hostility	11.19	5.92	0-24		N/A	N/A	N/A		12.16	5.39	2-22	
	Verbal Aggression	13.70	3.46	2-21		N/A	N/A	N/A		5.63	3.01	0-13	
	Physical Aggression	9.99	6.20	0-27		N/A	N/A	N/A		5.89	4.34	0-16	
ERQ	Cognitive Reappraisal	22.86	7.33	6-42		30.43	5.39	13-42		23.84	5.63	10-32	
	Expressive Suppression	13.48	5.09	4-27		N/A	N/A	N/A		17.50	3.90	9-25	
FFMQ	Observing	21.51	5.81	7-35		N/A	N/A	N/A		N/A	N/A	N/A	
	Describing	19.56	5.35	6-30		N/A	N/A	N/A		17.74	5-42	7-27	
	Awareness	14.40	4.16	5-25		N/A	N/A	N/A		13.21	4.08	5-23	
	Nonjudging	13.08	4.54	5-25		N/A	N/A	N/A		13.13	3.89	6-23	
	Nonreactivity	15.59	3.92	6-26		N/A	N/A	N/A		16.47	3.78	9-24	

Note. STAXI-2 = State Trait Anger Expression Inventory version 2; ARS = Anger Rumination Scale; DAQ = Displaced Aggression Questionnaire; FFMQ = Five Facets of Mindfulness Questionnaire; N/A = Not Applicable or Not Measured.

Study I and II

Studies I and II used screening data from Bjureberg et al. (2023) trial on maladaptive anger. Participants were recruited between March and November 2019 through social media advertisements, national press articles, and a website for CBT studies. In total, 730 individuals applied and completed parts of the online screening questionnaires via the secure platform BASS (where the data was stored) used to determine eligibility for the trial (see Bjureberg et al., 2023, for full criteria). Of these, 192 participants were excluded due to missing data on key variables, resulting in a final analytical sample of N = 538 treatment-seeking adults.

The study I and II sample was middle-aged (M = 39.78, SD = 12.28) and included both women and men of roughly equal proportions (55.4% females), with a relatively high educational level (45.5% with university education). Approximately one-third met criteria for a psychological disorder (31.6%), and the majority reported prior psychological treatment (63.9%). Relative to community norms (Spielberger, 1999), the average participant scored around the 85th percentile on trait anger, anger-in, and anger-out, supporting the characterization of the sample as a clinically relevant anger population.

Study III

Participants in Study III were recruited between September and December 2025 through online advertisements, posters at Örebro University, and brief classroom presentations. The study targeted a non-clinical university population of Swedish adults (18-64 years old). Recruitment materials recommended that participants should be generally healthy and not currently experiencing significant mental health or anger-related problems, or medical conditions affecting cardiovascular functioning. No formal exclusion criteria were applied; participation was based on self-selection. In total 203 university adults provided written informed consent and enrolled in the experiment.

The study III sample was younger than those in Studies I, II, and IV (M = 29.42, SD = 9.81) and included a roughly equal gender distribution (53.7% female). Interestingly, although mean trait anger scores were relatively low (35th percentile), anger-in levels averaged around the 70th percentile, indicating that many participants would meet the inclusion threshold for the treatment study (study IV). Scores, however, ranged widely (2nd – 99th percentile), reflecting substantial heterogeneity within the sample.

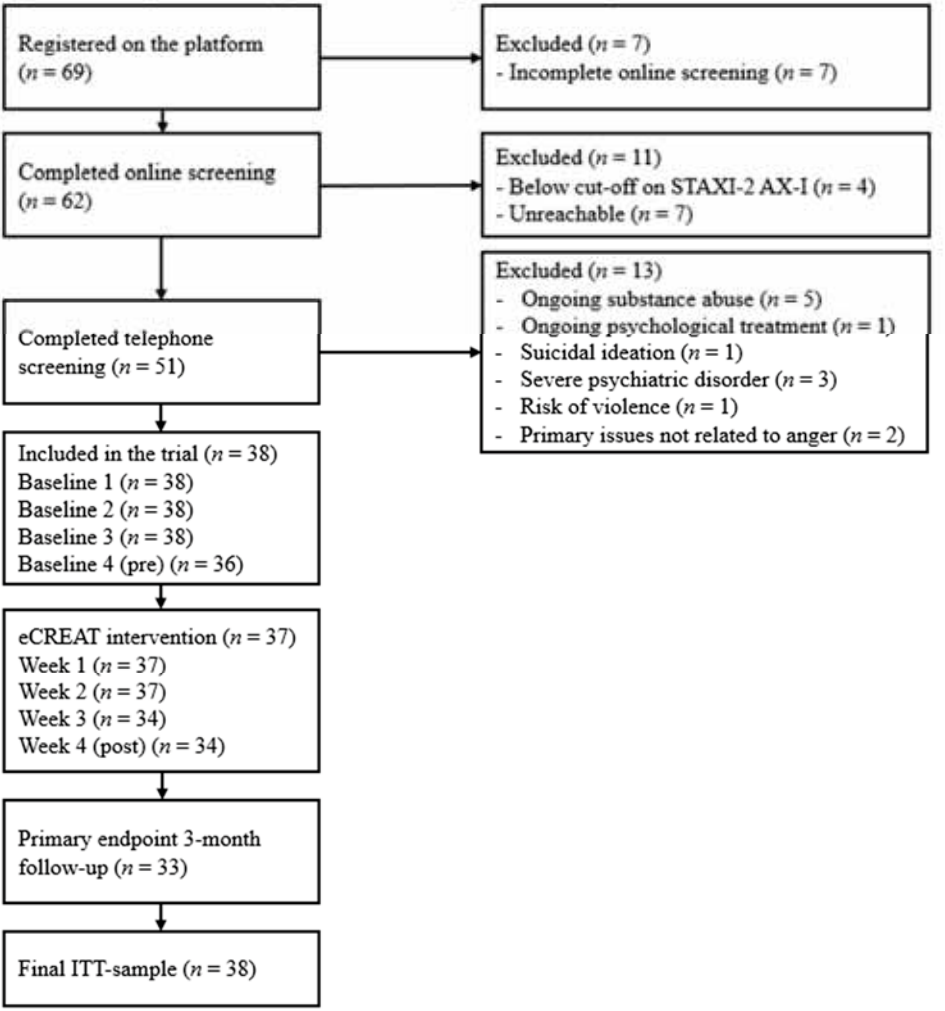
Study IV

Participant recruitment and treatment occurred between January and July 2024. Participants were recruited nationally across Sweden via online advertisements, university postings, and physical flyers. To be eligible, participants had to: a) high anger-in (upper quartile on the STAXI-2 AX-I scale; ≥ 18), and b) age 18-75 years. Exclusion criteria were: c) risk of violence, d) prior convictions of violence or abuse, e) ongoing psychological treatment, f) recent changes in psychotropic medication, g) severe psychiatric conditions (e.g., severe depression or psychosis), h) ongoing substance abuse, i) suicidal ideation, or k) insufficient Swedish language skills.

In total, 69 participants registered interest and provided informed consent via the online platform (BASS). Following online and telephone screening, 31 participants were excluded, resulting in a final intention-to-treat sample of $N = 38$. See Figure 4 next page for a flowchart of participant progression through the study.

The Study IV sample was predominantly female (89.5%) and well-educated (76.3% with a university degree), with a middle-aged profile ($M = 43.8$, SD = 14.1). Similar to the clinical samples in Studies I and II, a subset had a diagnosed psychological disorder (23.7%) and most reported prior psychological treatment (78.9%). Trait anger was slightly lower than in Studies I and II but remained above average, with the mean participant around the 70th percentile. Anger-out tendencies, on the other hand, were well below average, around the 30th percentile of the community norms whereas anger-in levels were considerably elevated (98th percentile), reflecting the intended recruitment of a sample primarily characterized by anger-in.

Figure 4. Study IV: Participant Flow Throughout the Study



Note. AX-I = Anger Expression-In; STAXI-2 = State-Trait Anger Expression Inventory; ITT = Intention-To-Treat

Data Collection

Online Data Collection – BASS (Study I, II, and IV)

In Studies I and II, data were collected using the BASS Online Data Collection platform, a secure system with two-factor authentication that supports both self-report data collection and administration of online treatment materials. In Study IV, the BASS platform was used not only for data collection but also for delivering the online treatment.

Experimental Hardware and Software (Study III)

In Study III, experimental procedures were run locally on a computer using PsychoPy version 2024.2.4 (Peirce et al., 2019), which controlled task presentation, timing, and the collection of anger ratings and frustration tolerance data. Demographic information, self-report questionnaires, and manipulation checks were completed on paper before and after the experiment.

Physiological data (HR and HRV) were collected using three disposable ECG electrodes placed on the lower right and left ribs and the right collarbone. Signals were recorded via a Biopac® MP36 system with an ECG100C amplifier, digitized at 2000 Hz, and logged in real time using AcqKnowledge software (version 5.0). Experimental phase onsets were marked with electronic triggers sent from PsychoPy through a StimTracker (ST-100).

Measures

Below, the measures employed across the studies in this thesis are briefly described. All measures have been previously validated and have demonstrated acceptable to excellent psychometric properties; detailed information is provided in the appended articles. An overview of the measures used across the studies is presented in Table 3.

Anger (trait) Measures

The *State Trait Anger Expression Inventory-2* (STAXI-2; Spielberger, 1999) was used across all four studies. STAXI-2 includes six scales and five subscales (57 items), with items rated on a 4-point scale (1–4, higher = greater anger). In Studies I and II, the anger-out (AX-O; 8 items), anger-in (AX-I; 8 items), and Trait Angry-Reaction (TANG-R; 4 items) scales were used as profile indicators (Study I) or network nodes (Study II). In Study III, the anger-in and total trait anger (TANG; 10 items) scales were used as pre-experimental descriptive variables, and in Study IV, the anger-in scale served as the primary treatment-outcome.

The *Anger Rumination Scale* (ARS; Sukhodolsky et al., 2001) was used in study I, II and IV. ARS assesses tendencies to ruminate on anger via 19 items rated on a 4-point scale (1-4; higher = greater anger rumination). The total scale score (ARS) was used as a profile indicator (Study I), a network node (Study II), or a secondary outcome (Study IV).

The *Aggression Questionnaire* (AQ; Buss & Perry, 1992; Ågren, 2009) was used in study I and II. AQ assesses four aggression-related constructs across 29 items rated on a 4-point scale (0-3; higher = greater aggression): trait anger (AQ-A; 7 items), verbal aggression (AQ-VA; 5 items), physical aggression (AQ-PA; 9 items), and hostility (AQ-H; 8 items). All four subscales were used either as profile indicators (Study I) or network nodes (Study II) in the first two studies.

Additional trait anger measures were used in specific studies for supplementary assessment. In Study IV, the *Behavioral Anger Response Questionnaire* (BARQ; Linden et al., 2003) assertion scale (6 items; 1-5;

higher = greater assertion) was used as a secondary treatment outcome to measure anger assertion. In Study III, pre-experimental trait measures included the anger rumination subscale from the *Displaced Aggression Questionnaire* (10 items; 1-7; higher = greater rumination; Denson et al., 2006), and a 5-item scale assessing positive attitudes toward anger from the *Attitudes Toward Emotion* scale (1-5; higher = more positive attitudes; Harmon-Jones et al., 2011).

Emotion Regulation (trait) Measures

The *Emotion Regulation Questionnaire* (ERQ; Gross & John, 2003) was used in Study I, II, and III. ERQ is a 10-item measure comprising two scales—cognitive reappraisal (6 items) and expressive suppression (4 items)—with items rated on a 7-point scale (1–7, higher = greater use of the strategy). In Studies I and II, both scales were used as profile outcomes (Study I) or as extended network nodes (Study II). In Study III, the cognitive reappraisal scale was additionally included as a pre-experimental trait measure of emotion regulation.

The *Five Facet Mindfulness Questionnaire* (FFMQ; Baer et al., 2004; Lilja et al., 2011) was used in study I, and II to assess various facets of mindful emotion awareness. The Swedish version consists of 29 items rated on a 5-point scale (1–5, higher = greater mindfulness) and comprises five subscales: observing (FFMQ-O; 7 items), describing (FFMQ-D; 6 items), acting with awareness (FFMQ-A; 5 items), nonjudging (FFMQ-NJ; 5 items), and nonreactivity (FFMQ-NR; 6 items), all scales were used either as profile outcomes (study I) or extended network nodes (study II).

Moreover, as an additional measure of emotion regulation we used the Anger-Control-In (AC-I; 8 items) scale from STAXI-2, as a measure of trait anger relaxation skills. Specifically, AC-I measure tendencies to regulate anger through calming and relaxation-based methods (e.g., “I try to relax,” “I take a deep breath and relax”) and was used as a profile outcome in study I and as an extended network node in study II.

Experimental Measures (Study III)

In study III state anger was assessed after each experimental phase using a single-item measure ("How angry do you feel right now?") rated on a 0–10 scale (0 = not at all, 10 = furious). From the anger induction phase onward (phase 2), participants also rated angry thoughts and perceived problems with anger control using similar 0–10 scales. To complement retrospective ratings of angry thoughts, participants also recorded the frequency of anger-related thoughts during suppression (Phase 3) and free recall (Phase 4) by pressing a key on the keyboard each time they experienced an angry thought.

Frustration tolerance was assessed using a computerized version of the Mirror Tracing Persistence Task (MTPT), in which participants traced a diamond-shaped figure on the screen using the mouse cursor. However, to induce frustration the mouse cursor moved in a mirror-reversed direction. Participants could terminate the task at any time, with task going on for a maximum of 7 minutes. Due to ceiling effects (37.4% completed the full task), frustration tolerance was operationalized as task completion (1) versus non-completion (0).

Heart rate (HR) was continuously recorded throughout the experiment. Mean HR and HRV, indexed by the time-domain measure Root Mean Square of Successive Differences (rMSSD), were calculated for each experimental phase. HRV served as a physiological marker of emotion regulation, with lower values indicating less adaptive regulation and higher values indicating more adaptive regulation.

A manipulation check was administered following experimental termination (post-experiment). Specifically, adherence to the assigned post-suppression regulation strategy during phase 5 (post-regulation phase) were evaluated using nine items (three per strategy: suppression, rumination, reappraisal) rated on a 1–7 scale, with higher scores reflecting greater endorsement of that strategy (e.g., "To what extent did you re-consider the event from an objective perspective?").

Diagnostic Assessments and Treatment Feasibility and Acceptability Measures (Study IV)

In Study IV, potential participants underwent a semi-structured diagnostic assessment via telephone using the *Mini International Neuropsychiatric Interview* (MINI; version 7) to assess for study eligibility and collect clinical background information. Interviews were conducted by licensed psychologists or master's-level students under supervision of a licensed psychologist.

Treatment feasibility (Aim 1) was evaluated via assessing treatment retention, adherence, and adverse events. Retention was defined as the number of participants who either dropped out or did not engage in treatment (completed <2 modules, <50%). Adherence was measured by the number of completed modules, homework assignments, and assessments. Adverse events were assessed using a self-report measure (Ljótsson et al., 2014) at post-treatment and 3-month follow-up, with participants indicating whether an adverse event occurred and rating its associated discomfort and lasting impact on a 4-point scale (0–3, higher = greater negative impact).

Treatment acceptability (aim 2) was evaluated using measures of credibility and satisfaction. The *Credibility/Expectancy Questionnaire* (CEQ; Devilly & Borkovec, 2000) was administered at week 2 to assess perceived treatment credibility and expected improvement. Specifically, CEQ comprises six items forming two subscales: credibility, rated on a 9-point scale (1–9; higher scores indicate greater perceived credibility), and expectancy, rated on a 0–100% scale reflecting the anticipated degree of symptom improvement (e.g., 50% indicating an expected 50% reduction in symptoms following treatment termination). Treatment satisfaction was assessed post-treatment using the *Client Satisfaction Questionnaire* (CSQ-8; Attkisson & Greenfield, 2004), an 8-item measure rated on a 4-point scale (higher = greater satisfaction).

Table 3. Measures Used Across Studies

		Study I	Study II	Study III	Study IV
Trait Variables	**Subscales**				
STAXI-2	Trait Anger	Indicator	Node	Pre-Experimental Measure	N/A
	Anger-Out	Indicator	Node	N/A	N/A
	Anger-In	Indicator	Node	Pre-Experimental Measure	Primary Outcome
	Trait Angry Reaction	Indicator	Node	N/A	N/A
ARS	Anger Rumination	Indicator	Node	N/A	Secondary Outcome
AQ	Hostility	Indicator	Node	N/A	N/A
	Verbal Aggression	Indicator	Node	N/A	N/A
	Physical Aggression	Indicator	Node	N/A	N/A
	Trait Anger	N/A	N/A	N/A	N/A
BARQ	Anger Assertion	N/A	N/A	N/A	Secondary Outcome
DAQ	Anger Rumination	N/A	N/A	Pre-Experimental Measure	N/A
ATE	Attitudes Toward Anger	N/A	N/A	Pre-Experimental Measure	N/A
ERQ	Cognitive Reappraisal	Outcome	Node	Pre-Experimental Measure	N/A
	Expressive Suppression	Outcome	Node	N/A	N/A
FFMQ	Observing	Outcome	Node	N/A	N/A
	Describing	Outcome	Node	N/A	N/A
	Awareness	Outcome	Node	N/A	N/A
	Nonjudging	Outcome	Node	N/A	N/A
	Nonreactivity	Outcome	Node	N/A	N/A
STAXI-2	Anger-Control-In	Outcome	Node	N/A	N/A
Experimental State Variables					
Anger		N/A	N/A	Phase 1-5 Outcome	N/A
Angry Thoughts		N/A	N/A	Phase 2-5 Outcome	N/A
Problems with Anger Control		N/A	N/A	Phase 2-5 Outcome	N/A
HR		N/A	N/A	Phase 1-5 Outcome	N/A
HRV		N/A	N/A	Phase 1-5 Outcome	N/A
Frequency of Angry Thoughts		N/A	N/A	Phase 3-4 Outcome	N/A
Frustration Tolerance*		N/A	N/A	Phase 5 Outcome	N/A
Manipulation Check		N/A	N/A	Post experimental Measure	N/A
Diagnostic Assessments, Treatment Feasibility, and Acceptability					
MINI version-7		N/A	N/A	N/A	Screening
Adverse events		N/A	N/A	N/A	Feasibility Outcome (post + fu)
CEQ		N/A	N/A	N/A	Acceptability Outcome (t2)
CSQ		N/A	N/A	N/A	Acceptability Outcome (post)

Note. STAXI-2 = State Trait Anger Expression Inventory version 2; ARS = Anger Rumination Scale; AQ = Aggression Questionnaire; BARQ = Behavioral Anger Response Questionnaire; ATE = Attitude Toward Emotions; DAQ = Displaced Aggression Questionnaire; FFMQ = Five Facets of Mindfulness Questionnaire; HR = Heart Rate; HRV = Heart Rate Variability; CEQ = Credibility/Expectancy Questionnaire; CSQ = Client Satisfaction Questionnaire; N/A = Not Measured or Not Used; * Measured by number of participants who completed the frustration task (1) versus not (0); fu = 3-month follow-up; t2 = treatment week 2.

Ethical Considerations

Ethical Approvals and Preregistrations

All four studies were approved by the Swedish Ethical Review Authority (Study I and II: EPN Dnr 2018/2253-31/5: 2020-05379: 2022-01484-02; Study III: EPN Dnr 2025-03741-01; Study IV: EPN Dnr 2023-04176-01). Moreover, study III was preregistered at OSF (doi; 10.17605/OSF.IO/NKGCT) and study IV at Clinical-Trials.gov (identifier: NCT06885866).

Personal Integrity

Studies I, II, and IV followed similar recruitment and data storage procedures. Participants provided informed electronic consent via the BASS platform after receiving written and verbal information about the respective study and handling of data, and were given the opportunity to ask questions during a screening interview with a member of the research team. All data (self-reports, demographic information, and interview material) were pseudonymized and stored in accordance with the General Data Protection Regulation (GDPR). Online data were managed via the BASS platform using two-factor authentication, in line with recommendations from the European Union Agency for Cybersecurity (ENISA).

In Study III, participants received study information both in writing and orally from the experimenter prior to providing written informed consent. Data (demographics, self-reports, and physiological measures) were collected using paper forms and PsychoPy, and were subsequently transferred to password-protected digital files stored on encrypted devices. Physical materials were securely stored in a locked, fire-resistant cabinet at the university.

Participant Safety and Stress Burden

Studies I, II, and IV all aimed to recruit samples with clinically elevated levels of anger-expressive problems (i.e., anger-out and anger-in). Participants in Studies I and II were drawn from applicants to Bjureberg et al. (2023), meaning that some only completed screening

procedures and did not proceed to treatment. Those who did enter treatment, however, were managed according to the same safety procedures as in the pilot trial in Study IV. Specifically, a structured risk management protocol was followed. First, before entering the treatment participants were screened via telephone interviews and excluded if they were assessed as having (a) an imminent risk of violence, (b) a history of violent or abusive behavior, or (c) high levels of externalizing aggression based on established cut-offs. Second, ongoing risk was monitored through repeated assessments of anger and aggression and therapist contact, with participants being able to reach therapists as needed. Excluded participants were provided with information that they could seek help elsewhere if needed. Third, a medical doctor was available for consultation both during screening and treatment if any medical adverse events would occur.

In Study IV, participant burden was somewhat higher due to intensive assessment, including weekly measures and two periods of daily assessments, combined with intensive treatment procedures. To avoid recruiting participants who deemed this as too demanding, study information clearly specified the expected time commitment. Moreover, ongoing therapist support, the brief format (four weeks), and the option to contact the research team at any time point were partly intended to mitigate potential stress-related effects.

In Study III, a non-clinical university sample was recruited. Risks to participant safety were deemed minimal but included potential discomfort from recalling an anger-inducing memory, completing a frustrating task, and wearing electrodes for physiological recording. Participants were informed of these procedures in advance and could withdraw at any time.

Results

Study I

Study I examined the presence of subgroups in a sample of treatment-seeking adults with anger problems (N = 538) using latent profile analysis (LPA; aim 1). Model comparisons (one to seven profiles) supported a four-profile solution, with substantial improvements in model fit up and log-likelihood ratio tests (LMR and ALMR) up to four profiles and only marginal gains thereafter. Classification quality and theoretical interpretability further supported the four-profile solution

Three of the four profiles were primarily distinguished by overall severity across anger indicators and were labelled the low, moderate, and high anger profiles. Participants in the Low Anger profile (Profile 1; n = 153) scored below the sample mean, those in the Moderate Anger profile (Profile 3; n = 193) around the mean, and those in the High Anger profile (Profile 4; n = 101) above the mean across most indicators. In contrast, the fourth profile (Anger-In; Profile 2; n = 91) showed a qualitatively distinct pattern, characterized by low outward expression of anger (e.g., aggression) alongside elevated anger-in, hostility, and anger rumination (see Table 4).

When validating the profiles against emotion regulation strategies (aim 2), membership in the High Anger profile was associated with the lowest levels of most putatively adaptive emotion regulation strategies and elevated use of expressive suppression. Membership in the Anger-In profile was associated with the highest levels of expressive suppression and the second lowest levels of mindful nonjudging. Surprisingly, the anger-in profile had the second highest levels of cognitive reappraisal (just below the low anger-profile).

Finally, demographic differences emerged across profiles (aim 3). Younger participants were more likely to belong to the High Anger profile compared to the Low and Moderate Anger profiles, and women were more likely to belong to the Anger-In and High Anger profiles. No significant differences were observed across profiles in other clinical or background variables.

Table 4. *Study I: Descriptive Statistics of the 4-Profile Solution*

Anger Indicators	Profiles			
	Profile 1: Low Anger (*n* = 153)	Profile 2: Anger In (*n* = 91)	Profile 3: Moderate An er (*n* = 193)	Profile 4: High An er (*n* = 101)
	M (*SE*)	*M* (*SE*)	M (*SE*)	*M* (*SE*)
STAXI-2				
Trait-angry reaction	6.67 (0.40)	7.83 (0.61)	9.29 (0.56)	12.03 (0.77)
Anger-out	16.82 (0.62)	15.82 (0.83)	20.77 (0.74)	23.79 (0.85)
Anger-in	17.17 (0.64)	23.77 (0.87)	20.71 (0.85)	25.44 (0.69)
AQ				
Hostility	5.47 (0.65)	10.95 (0.83)	11.84 (0.91)	18.60 (0.79)
Anger	12.23 (0.58)	10.47 (0.97)	14.83 (0.58)	16.67 (0.56)
Verbal aggression	5.96 (0.51)	5.00 (0.68)	8.67 (0.61)	10.14 (0.65)
Physical aggression	6.92 (0.65)	6.10 (0.88)	11.66 (0.89)	14.92 (0.93)
ARS	35.12 (1.1)	51.17 (1.12)	49.69 (1.34)	63.44 (1.13)

Note. STAXI-2 = State Trait Anger Expression Inventory-2; AQ = Aggression Questionnaire; ARS = Anger Rumination Scale

Study II

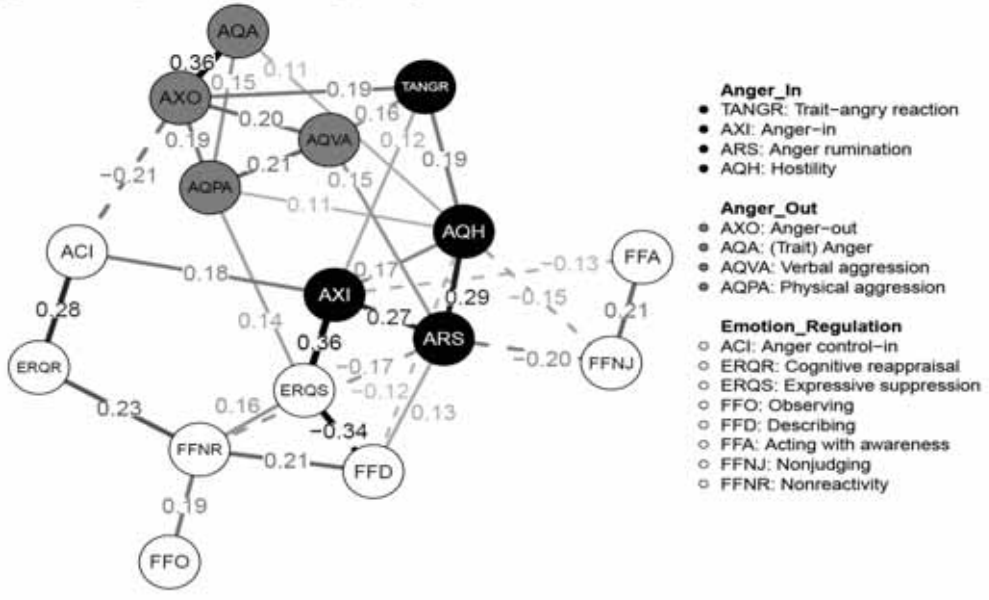
Study II examined the interplay between anger components and emotion regulation strategies using the same sample (N = 538) and variables as in Study I. In this study we used a variable-centered, rather than person-centered, approach through network analysis. Similarly to study I, two clusters of densely connected anger nodes were identified (aim 1): the *Anger-In cluster* and the *Anger-Out cluster*. The Anger-In cluster included anger rumination (ARS), anger-in (AX-I), hostility (AQ-H), and trait angry reaction (TANG-R), reflecting cognitive aspects of anger. The Anger-Out cluster included anger expression (AX-O), trait anger (AQ-A), verbal aggression (AQ-VA), and physical aggression (AQ-PA), reflecting mostly behavioral aspects.

Based on these two clusters, three extended networks were estimated (aim 2), including all emotion regulation strategies: the *Anger-Out Regulation network*, *Anger-In Regulation network*, and the *Anger-Regulation network*. The combined anger-regulation network, shown in Figure 5, provides a visual illustration of the full pattern of connections between anger components and regulation strategies (similar patterns were also observed in the cluster-specific networks).

Focusing on the most relevant associations and centrality findings, the anger-regulation network showed that neither mindfulness nor cognitive reappraisal nodes were directly connected to the Anger-Out cluster but instead were linked indirectly via Anger-In-cluster nodes, expressive suppression, or relaxation (AC-I). Moreover, the three most influential nodes in the combined network were anger-in (AX-I), anger rumination (ARS), and anger-out (AX-O), in falling order.

Overall network stability tests, including the stability of the derived clusters, the accuracy of the partial correlations (edge weights), and centrality indices, indicated that the networks were generally stable (see original article for details).

Figure 5. Study II: Combined Anger-Regulation Network



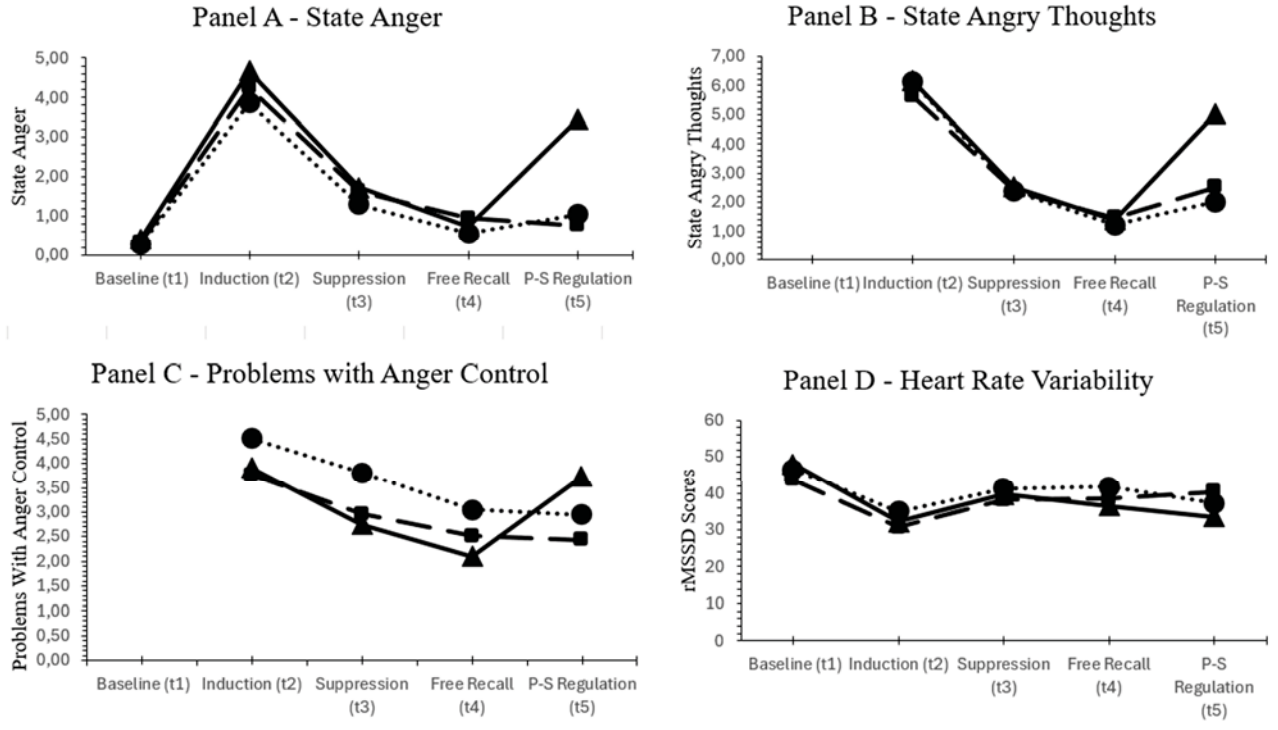
Note. Nodes are categorized into three groups: Anger-In cluster nodes (black), Anger-Out cluster nodes (grey), and emotion regulation nodes (white). Partial correlation coefficients are shown on the edges. Dashed lines represent negative partial correlations, and solid lines represent positive partial correlations.

Study III

Study III experimentally explored the immediate and delayed effects of manipulated anger suppression and subsequent use of three post-suppression regulation strategies (continued suppression, rumination and reappraisal) on anger and HRV. Within-subject results indicated that anger suppression led to immediate reductions in self-reported anger, angry thoughts, and (to a lesser extent) problems with anger control from anger induction to suppression (phase 2→phase 3). Contrary to expectations, anger continued to decline from the suppression phase to the free-recall phase (phase 3→phase 4), providing limited evidence of any rebound effects. Similarly, HRV continuously increased from induction to suppression (phase 2→phase 3) and from suppression to free-recall (phase 3→phase 4), indicating improved physiological regulation.

In the final phase, clear between-condition differences emerged: participants assigned to the suppression-rumination condition reported significantly higher anger, angry thoughts and problems with anger control than those in both the suppression-suppression and suppression-reappraisal conditions, whereas no differences were observed between the suppression-suppression and suppression-reappraisal conditions. Suppression-rumination was also associated with lower HRV compared to suppression-reappraisal. Finally, participants in the suppression-suppression condition were less likely to finish the frustration tolerance task than those in the other two conditions. See Figure 6 for estimated mean changes for anger and HRV outcomes per phase.

Figure 6. Study III: Estimated Mean Trajectories of Self-Reported Anger and HRV Across Phases and Conditions



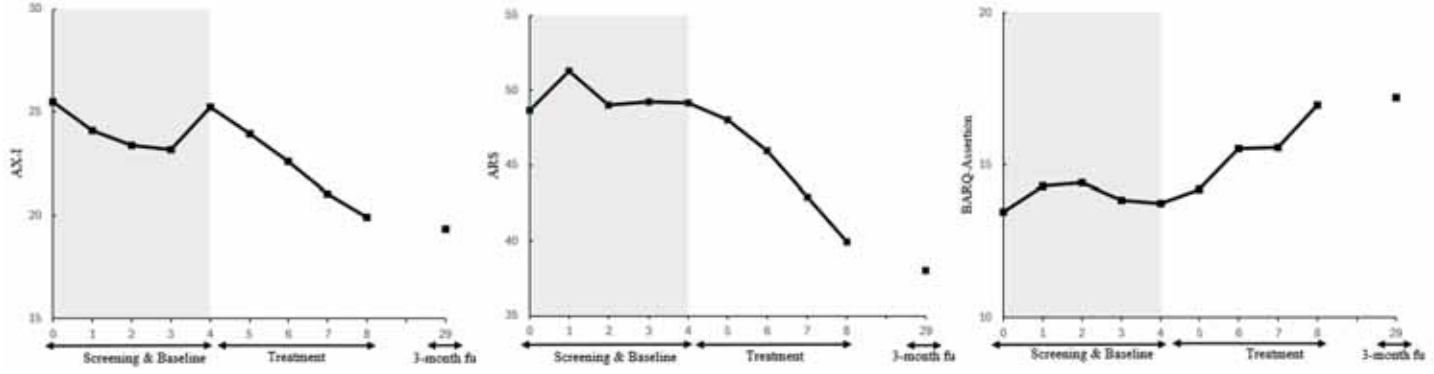
Note. P-S Regulation = Post-Suppression Regulation. Values represent estimated marginal means derived from the statistical models; phase 5 estimates are adjusted for induction-phase (phase 2) levels of the corresponding outcome.

Study IV

Study IV evaluated the feasibility, acceptability, and preliminary efficacy of a four-week internet-delivered Enhanced Cognitive Reappraisal and Emotion Awareness Training (eCREAT) internvetion for maladaptive anger inhibition. Treatment feasibility was supported by low attrition (15.8%), high treatment engagement (participants completed on average 3.21 of 4 modules and 2.61 of 4 homework assignments), and high post-treatment data completion (89.5%), comparable to our previous emotion regulation focused trial for maladaptive anger (Bjureberg et al., 2023). Moreover, we also found support for the acceptability of the treatment. Participants reported high treatment credibility, with an expected symptom reduction of 51.6% mid-treatment (i.e., expecting that symptoms of maladaptive anger inhibition would reduce by approximately 50 % post-treatment). Moreover, treatment satisfaction was rated as high post-treatment (M = 27.62, SD = 3.35), and although 21.1% reported adverse events post-treatment (7.9% at follow-up), these events were predominantly mild to moderately mild, transient, and associated with limited lasting impact (e.g., transient stress or irritability)

Regarding preliminary efficacy, piecewise latent growth modeling demonstrated significant improvements across all outcomes, with distinct phase-related changes over time. Large reductions were observed in anger-in (d = 1.26, 95% CI [0.83, 1.69]) and anger rumination (d = 0.80, 95% CI [0.41, 1.20]), alongside a large increase in anger assertion (d = 0.79, 95% CI [0.35, 1.23]), indicating improvements across both inhibitory and expressive dimensions of anger regulation. See Figure 7 for observed mean changes throughout the baseline, treatment and follow-up phases.

Figure 7. Observed Mean Changes of Treatment Outcomes



Note. Weeks from screening (0) are depicted in the x-axis and observed means of outcomes on y-axis. Screening and baseline assessments (week 0-4) are shaded in grey, and treatment and 3-month follow-up (week 5-29) assessments in white. AX-I = Anger-In; ARS = Anger rumination; BARQ-Assertion = Anger assertion; fu = Follow-up.

General Discussion

The overall aims of this thesis were to advance understanding of maladaptive anger inhibition in adults and to support the development of effective psychological treatments. To this end, four studies were conducted. Study I examined the presence of distinct anger subgroups within a treatment-seeking sample. Study II applied network analysis to investigate the interplay between anger components and emotion regulation. Study III experimentally examined how anger suppression interacts sequentially with subsequent regulation strategies. Finally, Study IV evaluated the feasibility, acceptability, and preliminary efficacy of a newly developed emotion regulation–focused CBT intervention for maladaptive anger inhibition.

Study I identified four anger profiles, of which two were particularly clinically relevant: a high anger profile characterized by combined anger-out and anger-in tendencies, and an anger-in profile marked by elevated anger-in, rumination, and hostility. Extending this, Study II identified anger rumination and anger-in as central network nodes, indicating their potential role in maintaining maladaptive anger. Study III demonstrated that the effects of suppression depend on subsequent strategies: suppression followed by rumination was most maladaptive, whereas suppression followed by reappraisal—or even continued suppression—was associated with reduced anger and increased HRV. Finally, Study IV indicated that the enhanced emotion awareness and cognitive reappraisal training (eCREAT) intervention was feasible, acceptable, and potentially effective in treating maladaptive anger inhibition.

The following sections provide a more detailed discussion of the study results. First, findings are considered in relation to advancing the understanding of maladaptive anger inhibition, including its manifestation and how anger can be regulated. Next, implications for treatment are addressed, drawing on both the pilot study results (Study IV) and literature on emotional processing and expression.

Finally, methodological strengths and limitations are discussed, followed by concluding remarks.

Understanding Maladaptive Anger Inhibition

In this section, findings from Study I, and to some extent Study IV, are discussed. The first part focuses on describing possible defining characteristics of individuals with maladaptive anger inhibition and contrasting these to individuals high in anger-out. This is followed by a discussion of conceptual issues related to the anger-in scale, which was used across all studies in the thesis. Concluding the section, a proposed typology of maladaptive anger inhibition is presented.

Empirically Derived Subtypes of Maladaptive Anger

When empirically exploring the presence of anger subgroups within an anger-treatment seeking sample (N = 534) in Study I, four distinct profiles emerged (low, moderate, high anger, and an anger-in profiles). Of particular clinical relevance were the high anger (n = 101) and anger-in (n = 91) profiles. The high anger profile was characterized by the highest levels on all anger indicators, including both behavioral (e.g., verbal and physical aggression) and cognitive-affective (e.g., hostility and anger rumination) components of anger, likely representing a mixed anger-out/anger-in group. In contrast, the anger-in profile was characterized by relatively low levels of verbal and physical aggression (lowest across all groups), alongside elevated anger-in, and anger rumination, potentially representing a group of individuals with primary problems with maladaptive anger inhibition.

These findings support the accumulating evidence that anger-in and anger-out tendencies are better conceptualized as orthogonal dimensions rather than opposite ends of a continuum (Deffenbacher, Oetting, Thwaites, et al., 1996; Eckhardt et al., 2004; Spielberger, 1999). That is, although individuals may show elevated levels of *either* anger-out tendencies *or* anger-in tendencies, these may also co-occur, as we saw for individuals in the high anger profile.

More broadly, the results also align both with previous anger diagnostic proposals (DiGiuseppe & Tafrate, 2007; Martin, 2020) and emerging empirical work that has identified distinct anger subgroups characterized by either anger-out, anger-in tendencies, or both, across both adolescent (Furlong & Smith, 1998) and adult samples

(DiGiuseppe & Tafrate, 2007; Romero, 2022). For example, using latent profile analysis in a sample of 1,170 individuals with maladaptive anger, Romero (2022) recently identified eight distinct profiles, including a subgroup characterized by moderate levels of angry cognitions and low levels of overt aggression—closely resembling the Study I Anger-In profile.

Differences in Emotion Regulation Across Anger Subgroups

Interestingly, and consistent with theoretical accounts of maladaptive anger and anger-driven aggression (Roberton et al., 2012), we found evidence of under- and overregulation in the high anger and anger-in profiles. The high anger profile, in particular, showed a clear pattern of underregulation, reflected in the lowest levels of putatively adaptive strategies such as cognitive reappraisal and relatively low relaxation skills (i.e. anger-control in), alongside lower levels in more dispositional emotion regulation abilities, including mindful- awareness, describing, nonjudging, and nonreactivity. At the same time, this group reported relatively elevated levels of expressive suppression, suggesting co-occurring overregulatory tendencies, which was also reflected by the highest levels of anger rumination.

Individuals in the anger-in profile, by contrast, showed a more mixed emotion regulation pattern. Although they, like the high anger group, were characterized by high expressive suppression and low nonjudging, they also reported relatively high cognitive reappraisal and the highest levels of relaxation skills, suggesting access to multiple potentially adaptive regulatory strategies. However, the combination of low aggression with elevated cognitive activation (e.g., rumination and hostility) indicates that these individuals may have been better at regulating behavioral rather than experiential components of anger. Moreover, although not directly examined in Study I, the combination of low nonjudging (i.e., a more critical stance toward internal experiences) and high expressive suppression may suggest that these strategies were primarily avoidance-driven, reflecting attempts to control or avoid unwanted internal experiences such as anger (Gardner & Moore, 2008; Roberton et al., 2012). Notably, a highly similar pattern of anger and emotion regulation emerged in the treatment-seeking sample for maladaptive anger inhibition in Study IV.

Differences In Demographic and Clinical Characteristics Across Anger Subgroups

Across both Study I and Study IV, individuals with anger-in tendencies—and particularly those seeking treatment for maladaptive anger inhibition—were predominantly female, with women comprising up to nearly 90% of the clinical sample in Study IV. This aligns with research on gender differences in anger expression, suggesting that women are more likely to suppress or express anger indirectly compared to men in social contexts (though not necessarily in non-social contexts; Evers et al., 2005) as well as within certain relationship constellations (e.g., egalitarian relationships; Fischer & Evers, 2011).

Moreover, when comparing the maladaptive anger inhibition treatment sample in Study IV with the broader treatment-seeking sample in Study I (consisting of a mix of anger-in and anger-out participants), several notable differences emerged. First, although the Study I sample was relatively well educated (45.5% holding a university degree), the Study IV sample was even more highly educated, with 76.3% holding a university degree. In addition, participants in Study IV reported significantly lower levels of trait anger (M = 20.63, SD = 4.02) than those in Study I (M = 23.99, SD = 5.78), $t(49) = 4.81$, $p < .001$, $d = 0.59$.

Taken together these findings suggest that anger-out and anger-in tendencies may be partly differentiated by their regulatory patterns, with anger-out reflecting primarily underregulation and anger-in overregulation. Moreover, findings suggest that, at least in the context of self-referred, internet-delivered treatment, individuals reporting primary problems with maladaptive anger inhibition are predominantly highly educated women, who may exhibit comparatively lower levels of trait anger than broader anger samples.

Conceptual and Measurement Issues in Maladaptive Anger Inhibition

While findings from Studies I and IV suggest that maladaptive anger inhibition may constitute a distinct anger-related problem, there are also indications, from past studies, that maladaptive anger inhibition

may overlap with general negative affectivity (Burns et al., 2008a; Jasinski et al., 2016). What, then, might account for the close association between maladaptive anger inhibition and negative affect? Burns et al. (2008a) argues that this overlap is likely driven, at least in part, by how maladaptive anger inhibition has been operationalized—most commonly through the anger-in scale (Spielberger, 1999). This issue is particularly relevant in the present thesis, where the anger-in scale was used across all four studies to index maladaptive anger inhibition, and thus any measurement limitations directly inform the interpretation of the findings. Specifically, Burns et al. (2008a) highlights two key concerns with the anger-in scale and its application.

First, the anger-in scale shows moderate associations with several indices of negative affectivity, including depressed mood, anxiety, anxious apprehension, and neuroticism (Cooper et al., 2008; Martin & Watson, 1997; Smits & Kuppens, 2005; Stewart et al., 2008). This may partly reflect that the scale taps two related but distinct processes: the experience of internal anger and the suppression of its expression (Burns et al., 2008a; Jasinski et al., 2016). This overlap complicates interpretation, as it becomes difficult to determine whether observed effects—such as associations with pain or cardiovascular disease—reflect maladaptive anger inhibition per se or shared variance with general negative affect (i.e., limited discriminant validity). In contrast, experimental operationalizations of state anger have shown more differentiated effects, with, for example, Burns and colleagues more directly isolating the causal role of state anger suppression on subsequent anger and pain responses (e.g., Burns et al., 2011; Quartana & Burns, 2007).

Second, research on anger expression has often examined anger-in and anger-out tendencies in isolation. As Burns et al. (2008a) notes, this approach may obscure the presence of at least two "implicit" anger-in subgroups (e.g., high anger-in/low anger-out and high anger-in/high anger-out), which may differ meaningfully in their

associations with negative affect, underlying vulnerability factors, and clinical outcomes[8].

One way to overcome the problem with excessive overlap with negative affect is for future research to develop more precise and discriminant measures of maladaptive anger inhibition, whether via refined self-report instruments or other assessment methods (e.g., experimental paradigms). As outlined in the background, this could involve distinguishing between key dimensions of anger inhibition, such as which aspects of the anger experience are suppressed (e.g., thoughts, feelings, behaviors), the direction of the suppressed anger (e.g., toward the self vs. toward others), and the degree of awareness or automaticity (ranging from implicit/automatic to explicit/deliberate). At the same time, further theoretical clarification of how maladaptive anger inhibition manifests may be needed to inform the development of such measures. Before turning to the next section, a few tentative proposals along these lines are outlined below.

Proposed Subtypes of Maladaptive Anger Inhibition

In addition to measurement issues related to the anger-in scale, I suggest that the observed overlap between negative affect and maladaptive anger inhibition may also partly reflect heterogeneity in how maladaptive anger inhibition manifests. Specifically, building on Burns et al.'s (2008a) suggestion of implicit subgroups, as well as prior theoretical work on anger suppression (Blackburn, 1968; DiGiuseppe & Tafrate, 2007; Megargee, 1966), I propose two

[8] In contrast to Burns et al. (2008a), I argue that any subtype of maladaptive anger inhibition should primarily be defined by excessive suppression of anger, rather than by a combination of high anger-in and high anger-out, as observed in the high anger profile in Study I. At the same time, Burns et al. (2008a) raise an important point: some individuals with maladaptive anger inhibition may potentially be at increased risk for certain forms of aggression and anger-out behaviors (e.g., passive aggression) and may therefore show slightly elevated anger-out tendencies.

potential subtypes of maladaptive anger inhibition, that could be used as hypotheses for future research.

The first subtype, which may tentatively be termed *suppressed anger turned inward subtype*, is characterized by the suppression of experiential (i.e., thoughts and feelings) and expressive components of anger. Individuals in this group may occasionally acknowledge feelings of anger but tend to rapidly remove any angry thoughts or feelings from conscious experience. In social contexts, they may even deny experiencing anger when questioned by others. Despite this, they may show signs of heightened physiological arousal when provoked, suggesting underlying anger that is actively suppressed (e.g., Burns et al., 1999). This subtype is further characterized by strong negative beliefs about anger, and internalized rules against experiencing or expressing anger. Importantly, when experiences of anger linger, they may redirect the anger inward toward the self, often in the form of self-blaming and self-critical thoughts (i.e., self-focused anger rumination; Denson, 2013), such as blaming oneself for being angry.

The second subtype, which may be tentatively termed *suppressed anger turned outward subtype*, is characterized by the suppression of overt anger expression, while anger-related thoughts and evaluations remain directed toward others. This subtype more closely resembles the content captured by Spielberger´s (1999) anger-in scale (e.g., "I boil inside but I don´t show it"). Individuals in this subtype are likely to experience relatively higher levels of trait anger compared to the suppressed anger turned inward subtype. As such, although they primarily suppress outward expression, they are not totally free from aggression. Instead, they may engage in passive aggressive acts (e.g., spreading negative rumors) or occasional outbursts of displaced aggression (e.g., expressing anger toward a safer target). Thus, despite elevated withdrawal-related tendencies, the underlying action tendency toward aggression is likely still present and may also manifest covertly in the form of aggressive fantasies and thoughts of retaliation. These individuals are probably fully aware of their anger and similar to the inward subtype, may adhere to strict (social) rules against expressing anger outwardly, while experiencing fewer restrictions on anger-related thoughts and feelings.

Using these subtypes, I suggest that the suppressed anger directed inward subtype to be more strongly associated with negative affectivity and internalizing problems, such as depression. One may even speculate whether this subtype might be more appropriately conceptualized within existing models of other internalizing disorders. In contrast, the suppressed anger directed outward subtype is proposed to represent a more distinct anger-related problem[9], with latent aggressive tendencies. Importantly, these subtypes are not proposed to be mutually exclusive but can co-exist such that one may shift between, for example, directing angry thoughts against others and then turn anger against oneself (i.e., a combined presentation).

Tentative Evidence for the Proposed Subtypes

While evidence is limited, some preliminary empirical support for the above suggested typology of maladaptive anger inhibition can be identified. For example, as noted previously, Romero (2022) identified a subgroup characterized by moderate levels of anger-related cognitions, low levels of overt aggression, and moderate levels of passive-aggressive behavior, which aligns with the proposed suppressed anger directed outward subtype. Similarly, and as touched upon in the background, in the context of intimate partner violence, Eckhardt et al. (2008) identified a subgroup characterized by elevated anger-in and relatively low anger-out that nonetheless showed an increased risk of reoffending, including assault-related outcomes (i.e., aggression), compared to low-anger groups.

Evidence for a suppressed anger directed inward subtype is more limited. Ahmed et al. (2012) identified a subgroup characterized by high anger control, low impulsivity, and low overt aggression, which they

[9] As noted in the background, a defining feature of anger is its association with approach motivation and, and aggressive action tendencies or impulses (e.g., Carver & Harmon-Jones, 2009). Accordingly, clinically meaningful anger subtypes should be expected to show some linkage to aggressive inclinations; profiles lacking such features, such as the proposed suppressed anger directed inward type, may be more difficult to interpret as specific anger phenomena but may still warrant investigation.

classified within DiGiuseppe & Tafrate's (2007) taxonomy as a dysfunctional "suppressed-organized" anger subtype. While this may reflect a more inwardly directed form of maladaptive anger inhibition, it remains unclear whether it represents a true clinical subtype or simply a relatively low-anger profile. More fine-grained assessments of this potential subtype, including, for example, physiological indices of arousal (e.g., Burns et al., 1999), may be needed to clarify this distinction.

From a theoretical point, it has also been argued that it is important to distinguish between the suppression of experiential versus expressive components of emotion, as these may serve distinct functions (Greenberg & Bischkopf, 2014). Experientially, anger may function to signal threat or goal obstruction, whereas its expression may serve interpersonal functions and prepare the individual for confrontation (e.g., Novaco, 1976).

Anger Regulation

While the previous section also touched on anger regulation, this section places it in more explicit focus. Drawing on findings from Studies II and III, it examines the interplay between putatively maladaptive and adaptive regulation strategies and how these shape different components of anger. The role of anger suppression in maladaptive anger inhibition is also critically reviewed.

Anger Regulation Dynamics

To more directly examine how different emotion regulation strategies interact in the context of anger, Study II adopted a variable-centered approach using network analysis. The primary aim was to explore how behavioral and cognitive components of anger relate to one another and to different emotion regulation strategies.

The network structure in Study II revealed two relatively distinct clusters. One cluster was characterized primarily by behavioral expressions of anger, including nodes measuring verbal and physical aggression, anger-out, and trait anger (i.e., the anger-out cluster). The other cluster comprised cognitive-affective components, including nodes measuring anger-in, anger rumination, hostility, and heightened reactivity to provocation (i.e., the anger-in cluster).

Notably, nodes within each cluster were strongly interconnected. In network terms, such tightly coupled structures may reflect self-reinforcing loops, or so called *pathological attractor states*, in which activation of one node increases the likelihood of activating others (Hayes & Andrews, 2020). Within the anger-in cluster, anger-in and anger rumination emerged as the most influential nodes, showing the highest strength centrality scores. This suggests that anger-in and anger rumination may play a particularly important role in sustaining potential self-reinforcing loops. For example, anger-in may reinforce tendencies to ruminate (Burns et al., 2008a), while rumination in turn may increase reactivity to provocation and aggression (Denson, 2013). Notably, reactivity to provocation indeed appeared to function as a bridge node between the anger-in and anger-out clusters, through its

associations with anger-out and verbal aggression (however, we did not formally test for the presence of any bridge nodes).

That said, although to my knowledge no study has tested the full aforementioned hypothesized sequence (suppression → rumination → reactivity to provocation → aggression), evidence from different fields provides some tentative support. As noted in the background, Burns and colleagues have shown across several experiments that suppressing anger can increase the cognitive accessibility of anger-related thoughts (i.e., anger rebound; Burns et al., 2008b; Quartana & Burns, 2007), potentially setting the stage for subsequent rumination (Burns et al., 2008a), although the rebound effect may not be present for all individuals (Burns et al., 2011), a point I will return to. In turn, Denson and colleagues have demonstrated across a series of experiments that anger rumination may reduce self-control and increase reactivity to provocation, which in turn increase the risk of aggressive behavior (Denson et al., 2011b). Importantly, though, we do not know to which extent these hypothesized effects apply primarily to individuals high in anger-out or high in anger-in, as Study II used the full mixed sample of anger-treatment seeking individuals (i.e., both anger-out and anger-in individuals).

Providing further support for a potential link between the anger-in cluster nodes and aggression, we observed a direct positive association between trait expressive suppression and trait physical aggression. This finding is consistent with other cross-sectional research linking trait suppression of anger to both elevated anger (Pop et al., 2025) and aggressive behavior (Smith et al., 2026). One hypothesis is that expressive suppression reduces outward expression while leaving internal anger and ruminative processes intact (Burns et al., 2008a; Sukhodolsky et al., 2001), thereby increasing the likelihood of aggressive responses following later provocation (Denson et al., 2011b). In line with this, research on triggered displaced aggression has shown that when individuals are prevented from responding to an initial provocation—paralleling expressive suppression—and given opportunity to ruminate, they subsequently display elevated aggression in response to a minor second provocation (Bushman et al., 2005). More broadly, both rumination and suppres-sion have been proposed as mechanisms contributing to displaced aggression (Lagios et al., 2025; Miller et al., 2003). However, it would be

interesting to further test whether the effects of initial suppression attempts (time 1 provocation) on displaced aggression (time 2 provocation) are moderated by individuals' habitual anger-regulation style (i.e., anger-in versus anger-out).

Sequential Regulation Effects (Study III)

In response to findings from Study II and Burns' two-stage theory suggesting a sequential link between anger suppression and anger rumination, Study III experimentally tested this pathway alongside two alternative anger-regulation sequences (suppression–suppression and suppression–reappraisal). In line with Burns et al., (2008a) two-stage theory, anger suppression followed by rumination turned out as the most detrimental sequence, with elevated anger and reduced HRV following phase 5 (the post-suppression regulation phase). However, as will soon be discussed, we found little evidence that suppression sets the stage for subsequent rumination via an anger rebound effect, calling into question suppression as the primary underlying mechanism of maladaptive anger inhibition (Burns et al., 2008a).

In contrast, the suppression–reappraisal sequence produced the most favorable outcomes. Specifically, participants in this condition showed the lowest levels of all state anger outcomes (state anger, angry thoughts, and difficulties with anger control) and the highest HRV. This adds to the growing body of research suggesting cognitive reappraisal as a particularly effective anger regulation strategy (Denson et al., 2011a; Pop et al., 2025; Szasz et al., 2011). More importantly, these results extend Peuters et al. (2019) work on sequential regulation, suggesting that a putatively maladaptive strategy (suppression) followed by a putatively adaptive (reappraisal) reduces anger and increase HRV, whereas pairing two putatively maladaptive strategies (suppression-rumination) exacerbates anger and HRV.

Contrary to expectations, continued suppression and suppression followed by reappraisal did not differ significantly in terms of subjective anger or HRV, suggesting that the two strategies were similarly effective in downregulating emotional and physiological responses. One potential negative effect of continued suppression, however, emerged. Fewer participants in the continued suppression condition completed

the frustration task relative to the other conditions. This finding is consistent with resource depletion accounts suggesting that suppression is cognitively and physiologically taxing, potentially reducing persistence on effortful tasks (e.g., Dang, 2018). At the same time, alternative explanations—such as reduced motivation due to repeated instructions (i.e., experimental fatigue)—cannot be ruled out.

Rethinking the Role of Anger Suppression

Turning to the within-subject findings of Study III, in which we examined both immediate and delayed effects of state anger suppression, we found no evidence of either an immediate or delayed rebound effect following suppression. Instead, suppression was associated with immediate reductions in anger, increases in HRV, and corresponding decreases in heart rate following the anger suppression phase (phase 3). Moreover, contrary to our expectations, these regulatory benefits persisted, with further decreases in anger and continued increases in HRV during the free-recall phase (phase 4).

One possibility, which I will return to in the limitations section, is that the absence of a non-suppression control condition constrained our ability to detect rebound effects. However, anger levels remained very low during the free-recall phase, suggesting limited possible rebound. An alternative explanation is that rebound effects may be more likely in clinical populations, such as those with maladaptive anger inhibition. Yet, this interpretation is not strongly supported by prior research.

First, in the broader thought suppression literature, meta-analytic evidence indicates no reliable differences in rebound effects between clinical and non-clinical samples (Abramowitz et al., 2001; Magee et al., 2012). Second, findings specific to anger suggest the opposite pattern: individuals with stronger habitual tendencies to suppress may be more effective at doing so. For example, Germain and Kangas (2015) observed reductions in both anger and systolic blood pressure following suppression in a high trait anger sample, with even larger effects among those high in trait expressive suppression. Similarly, Burns et al. (2011) reported that individuals high in anger-in showed

reductions—rather than increases—in anger and pain following suppression (i.e., a reversed rebound effect).

In contrast, clearer indications of rebound effects appear among individuals high in anger-out. Indeed, across several experimental studies in the pain field, high trait anger-out individuals have shown elevated levels of anger and pain following anger suppression in comparison to those with lower scores (Burns et al., 2008c; Burns et al., 2011; Burns et al., 2007). One proposed explanation for these findings is the so called *match–mismatch* hypothesis, whereby the effects of a given regulation strategy depend on its fit with individuals' habitual regulatory tendencies; mismatches appear to yield more detrimental outcomes (Burns et al., 2011).

In conclusion, findings from study III together with Burns et al. (2011) and Germain and Kangas (2015), suggest that suppression may not be the primary driver of anger rumination (at least not for high anger-ins), as proposed in Burns et al., (2008a), although suppression may still contribute to other negative outcomes (e.g., social costs; Chervonsky & Hunt, 2017). This aligns with broader critiques of the thought suppression literature, which highlights substantial heterogeneity in findings. Reviews of rebound effects indicate that paradoxical increases are far from consistent (Niczyporuk, 2026; Purdon, 2020), with reversed effects (i.e., decreases rather than increases) observed in 18% (Abramowitz et al., 2001) to 46% (Wang et al., 2020) of analyzed studies, suggesting that not all individuals experience a rebound effect following suppression.

Building on the possibility that individuals with maladaptive anger inhibition may be less likely to experience anger rebound effects following suppression (relative to anger-out individuals), I encourage future research to consider alternative processes that may account for persistent anger rumination in this group. For example, in the context of depressive rumination, Watkins and Roberts (2020) conceptualizes rumination as a learned habitual response that becomes automatically triggered by negative affect through reinforcement processes (e.g., perceived problem-solving or emotional relief), implying that disrupting rumination may require the development of competing, non-

ruminative habits (e.g., mindful observation of thoughts). Alternatively, metacognitive models in the anxiety and depression literature (e.g., Wells, 2002) suggest that rumination is maintained by both positive beliefs (e.g., usefulness) and negative beliefs (e.g., uncontrollability), with emerging evidence indicating similar processes in anger rumination (Caselli et al., 2017; Salguero et al., 2020; Simpson & Papageorgiou, 2003).

Anger Regulation Abilities and Cognitive Reappraisal

In relation to more adaptive regulation, results from Study II indicated that cognitive reappraisal and mindfulness abilities did not operate in isolation but rather interdependently. Cognitive reappraisal was not directly connected to any of the anger nodes within the anger-regulation network; instead, only indirectly through mindfulness nodes. This is in line with broader accounts that mindfulness may facilitate other forms of emotion regulation (Roemer et al., 2015; Sedlmeier et al., 2012), and more specifically with Garland et al. (2015) Mindfulness-to-Meaning Theory, introduced in the background. To reiterate, this theory proposes that mindfulness facilitates reappraisal by fostering a metacognitive, decentered, and nonreactive stance toward emotional experience, which in turn broadens attention and enables more flexible reinterpretations of emotional events (Garland et al., 2015).

Interestingly, the association between mindfulness nonreactivity and cognitive reappraisal was among the strongest in the anger-regulation network. This aligns with theoretical accounts proposing that mindfulness nonreactivity specifically may facilitate reappraisal by decoupling emotional stimuli from their habitual appraisal value (e.g., as unwanted experiences; Lutz et al., 2015)

Mindfulness interventions have increasingly been emphasized as a complement to traditional CBT for anger, particularly for enhancing the ability to accept, label, and describe emotional experiences without reacting to them, thereby a larger array of different appraisals (Beames et al., 2019; Denson, 2015; Wright et al., 2009). In line with this, we recently reanalyzed Bjureberg et al.'s (2023) trial on maladaptive anger and found that combined mindful emotion awareness and

cognitive reappraisal training (MEA + CR) produced greater improvements in these respective mechanisms than either intervention delivered in isolation. This suggests potentially interactive and mutually reinforcing effects of integrating mindfulness and reappraisal training with eachother (Hesser et al., 2026).

Inspecting the anger-regulation network results in Study II at a more granular level, mindfulness nonjudgment and nonreactivity were found to be directly negatively associated with anger rumination—the most central node in the networks. Indeed, mindful nonjudgment and nonreactivity are thought to attenuate rumination by altering how individuals relate to their internal experiences. Rather than evaluating thoughts and emotions as harmful or threatening, individuals learn to accept them as transient states, and may thus disrupt repetitive thought patterns (Frewen et al., 2008). Illustrating this, across two daily diary studies Blanke et al. (2020) showed that nonjudgmental acceptance was linked to not only lower levels of rumination but also higher levels of reflection—defined as curiosity-driven, non-evaluative self-focus, which further accords with the proposition that mindfulness may promote more adaptive appraisals.

More specific to the context of maladaptive anger, both nonjudgment and nonreactivity have been linked to reductions in aggressive behavior, with anger rumination identified as a key mediating mechanism (Eisenlohr-Moul et al., 2016; Kim et al., 2022; Peters et al., 2015). In line with these findings, a recent meta-analysis by (O'Dean et al., 2025) reported that both dispositional mindfulness and experimentally induced mindfulness are associated with small-to-moderate reductions in anger-related outcomes and aggression, with similar findings observed for mindfulness-based interventions targeting aggression (Gillions et al., 2019).

Treatment Implications

This section focuses primarily on findings from the pilot treatment study (Study IV) to consider potential treatment implications. It begins by examining anger expression as a potential mechanism for counteracting excessive suppression tendencies. It then turns to anger rumination, describing how it was targeted in the pilot treatment and outlining possible directions for shifting from maladaptive ruminative toward more adaptive forms of emotional processing.

From Suppression to Expression – Finding Adaptive Ways to Express Anger

Results from Study IV provided preliminary support for the efficacy of the newly developed emotion regulation treatment, eCREAT, yielding large phase effects from baseline to treatment termination for anger-in (d = 1.26), anger rumination (d = 0.80), and anger assertion (d = 0.79). Notably, the reduction in anger-in exceeded that observed in Bjureberg et al.'s (2023) combined MEA + CR treatment (d = 0.74). This tentatively indicates that eCREAT may be particularly well-suited for individuals characterized by maladaptive anger inhibition. However, alternative explanations should be considered. For example, participants in Study IV exhibited higher baseline levels of anger-in (M = 24.11) than those in Bjureberg et al. (2023) (M = 20.86), which may have allowed for greater absolute change and a relatively larger effect (Nunes et al., 2011). As such, a direct head-to-head comparison between eCREAT and MEA + CR would be needed to determine any superiority in effect.

Nevertheless, one hypothesis for the potentially greater efficacy of eCREAT is its stronger emphasis on emotional expression as a means of counteracting habitual suppression. Although the MEA + CR treatment placed substantial emphasis on cultivating nonjudgmental awareness and acceptance of emotions (Bjureberg et al., 2023)—thereby, in part, targeting avoidance processes such as suppression—it provided comparatively limited guidance on how to translate emotional awareness into adaptive, goal-directed expression of anger.

In contrast, eCREAT, from module 3 and onwards, explicitly thought participants to approach, experience, and, if deemed necessary, constructively express anger. This emphasis is consistent with multiple theoretical traditions, including emotion-focused therapy (Greenberg & Bolger, 2001; Paivio & Carriere, 2014), emotional processing theory (Pennebaker, 1995; Rachman, 1980), and research on emotional assertiveness (Linehan et al., 1979; Speed et al., 2018), all of which underscore the role of adaptive emotional processing and expression in mitigating the adverse effects of chronic avoidance and suppression.

Constructive Anger Expression as a Mechanism of Change

The question, then, is whether constructive anger expression constitutes a key mechanism of change for individuals with maladaptive anger inhibition—as suggested by Burns et al. (2008a) in their notion of "anti-suppression expression"—and, if so, what such constructive expression entails? Unfortunately, as noted by many before me (e.g., Greenberg & Bischkopf, 2014; Linden et al., 2003) limited empirical research exists on constructive modes of anger expression. Instead, contemporary research has largely focused on two extreme modes of expressing anger—excessive aggression and suppression (e.g., using Spielberger´s anger-out and anger-in scales)—thereby reinforcing an implicit dichotomy in which anger is construed as harmful both when expressed and when suppressed (Greenberg & Bischkopf, 2014).

In contrast, Linden and colleagues (2003) propose a third, potentially more adaptive mode of responding: anger assertion. Anger assertion is by Linden et al. (2003) defined as a delayed (rather than impulsive), controlled, and tempered form of anger expression. When expressing anger assertively, anger is expressed in a firm yet not non-aggressive manner, thus representing a more constructive "middle path" between excessive aggression and excessive passivity and avoidance (Speed et al., 2018). Importantly, the way anger is expressed assertively, and its adaptiveness and effectiveness, likely varies as a function of an individual's preferred expressive and regulatory style.

Potential Counterproductive Effects of Expressing Anger

The idea that expressing anger may be beneficial has deep historical roots in the Aristotelian and Freudian theories of emotional *catharsis.* Put simply, the catharsis theory of emotions posits that expression of pent-up emotions leads to a discharge of emotional tension (i.e., an emotional release). Within the frustration–aggression theory, Dollard et al. (1939) formalized this view by suggesting that aggression itself can serve a cathartic function, stating that "the occurrence of any act of aggression is assumed to reduce the instigation to aggression" (p. 50). However, their cathartic hypothesis also included important caveats, noting that any reduction in anger arousal or aggression is likely transient and that catharsis may paradoxically strengthen aggressive responding via negative reinforcement processes, whereby temporary relief following aggression increases the likelihood of its recurrence (i.e., "aggression breeds aggression").

Subsequent experimental work, most notably by Bushman and colleagues, has indeed provided little support for the long-term cathartic effects of expressing anger aggressively. In a seminal study, angered participants instructed to engage in "anger venting" (hitting a punching bag while focusing on the provocateur) exhibited higher subsequent anger and aggression than controls (Bushman, 2002). These findings suggest that venting does not reduce anger long-term and has contributed to the view that anger expression techniques are therapeutically counterproductive. Or as stated by Lohr et al. (2007, p. 62) in their review of the research on anger venting: "Catharsis theory is harmful because it justifies and perpetuates the myth that venting anger is good, despite the fact that virtually all of the available scientific evidence indicates that venting anger increases aggression and violence."

However, this view may be somewhat overly critical (Greenberg & Bischkopf, 2014; Novaco, 2014). While classical venting activities such as punching a bag, screaming, or other forms of undirected emotional discharge clearly have limited relevance in psychological treatment, as they may reinforce such behaviors, alternative modes of expressing anger, such as anger assertion, may have its place. Unfortunately, to my knowledge, few experimental studies have

explored the behavioral and emotional effects of these potentially more adaptive modes of expression.

Moreover, a relevant critique to the current anger venting and anger expression literature is the limited ecological validity of the experimental paradigms used to test the effects of anger expression. For example, both Greenberg and Bischkopf (2014) and Novaco (2014) notes that many experimental paradigms in this area (e.g., Bushman, 2002) rely on tasks in which participants express anger toward arbitrary objects (e.g., punching a punching bag), rather than directing it toward the actual source of provocation. Developing paradigms that capture anger expression in interpersonal contexts, rather than displaced onto objects, may therefore be an important direction for future research. In addition, future work would benefit from more clearly differentiating between at least three forms of anger expression: suppression, anger-driven aggression (within which many venting exercises seem to fall), and assertive expression, and examining their potentially distinct effects. As noted by Novaco (2014), constructive anger expression differs from simple venting in that its primary aim is not emotional discharge (i.e., catharsis), but rather the communication of needs and the resolution of interpersonal conflict.

From Rumination to Adaptive Processing

Moving on to the second proposed key mechanism in maladaptive anger inhibition: rumination. eCREAT was associated with a large phase effect for anger rumination (d = 0.80). Although promising, this effect was smaller than that observed for the primary outcome of anger-in (d = 1.26) and comparable to effects reported for the MEA + CR treatment (d = 0.80; Bjureberg et al., 2023). This is somewhat unexpected, given that a substantial portion of eCREAT—particularly from module 2 onward—was explicitly designed to target rumination and maladaptive, judgmental thinking patterns.

For example, we incorporated strategies from rumination-focused cognitive behavioral therapy (RF-CBT; Watkins, 2018), adapted to an anger context (Denson, 2013). In RF-CBT, rumination is conceptualized as a maladaptive mental habit that can be disrupted by cultivating alternative, incompatible thinking habits and behavior.

Accordingly, eCREAT included strategies theorized to counteract rumination (Watkins, 2018), some of which are also used in anger treatment manuals, such as compassionate thinking (Kolts, 2022), flow-inducing activities (i.e., engagement in absorbing, goal-directed tasks; Watkins, 2018), and mindfulness practices, such as radical acceptance of thoughts and present-moment awareness, which were also part of the MEA + CR treatment (Bjureberg et al., 2023).

One possible explanation for the comparatively smaller effects, relative to anger-in, is that these strategies may have been effective in disrupting rumination once initiated, but less effective in preventing its onset. Accordingly, future treatments may benefit from incorporating components that more directly may influence the onset of rumination, such as positive metacognitive beliefs about the utility of ruminating (Wells, 2002), as discussed earlier.

An alternative, though not mutually exclusive, explanation is that individuals with maladaptive anger inhibition may require not only a reduction in rumination, but also the cultivation of more adaptive forms of emotional processing. Following anger-provoking events, such individuals may benefit from structured guidance in how to reflect constructively on interpersonal conflicts—integrating emotional experience, understanding their reactions, and identifying goal-consistent responses for future situations. This raises the broader question of how to distinguish adaptive emotional processing from maladaptive rumination (Kross & Ayduk, 2011; Watkins, 2004).

This inquiry, sometimes referred to as the *self-reflection paradox* (Kross & Ayduk, 2011), has long preoccupied researchers. On the one hand, a substantial body of work demonstrates that reflecting on and processing emotional experiences can be adaptive, promoting both psychological and physical health (e.g., Pennebaker, 1989; Rachman, 1980). Such benefits are thought to arise because structured reflection facilitates meaning-making, emotional integration, and the resolution of unresolved experiences (Rachman, 1980). On the other hand, an equally extensive literature identifies rumination as a robust transdiagnostic mechanism underlying a range of psychological disorders and psychological problems, including heightened anger and aggression (e.g., Aldao et al., 2010; Denson, 2013; Nolen-Hoeksema et al.,

2008). In this case, rumination is posited to lead to repetitive dwelling on negative experiences, with limited engagement in constructive problem solving or emotional resolution (for two comprehensive reviews on repetitive thoughts and rumination, see Watkins, 2008; Watkins & Roberts, 2020).

Within the field of anger rumination, two main lines of research have tried to dissect the question of adaptive versus maladaptive anger processing, with somewhat mixed findings. The first, based on Watkins' processing mode theory from the depression literature (e.g., Watkins & Roberts, 2020), distinguishes between abstract (analytical, "why-focused") and concrete (experiential, "what/how-focused") processing. Abstract processing involves evaluative, generalized thinking (e.g., "Why does this always happen to me?", "What does this say about me?"), whereas concrete processing is more specific and contextualized (e.g., "What happened?", "How did I respond?", "What can I do next?"). In depression research, concrete processing is generally associated with more adaptive outcomes, whereas abstract processing is associated with negative outcomes traditionally associated with rumination (Watkins & Roberts, 2020). However, with regards to anger rumination, evidence is more mixed. Some experimental studies show that concrete processing is related with lower levels of anger responding compared to abstract (Wimalaweera & Moulds, 2008), others suggest the reverse (Kross et al., 2005), and even others indicate that both modes may be associated with maladaptive outcomes (Heinzel et al., 2023).

A second, more extensively studied line of research, led by Kross, Ayduk, and colleagues, focuses on self-distancing versus self-immersion as two vantage points for processing emotional experiences. Self-distancing (adopting a third-person, observer-like perspective on one's experience) versus self-immersion (reliving the experience through a first-person, emotionally immersed perspective) has been shown to differentially affect emotional responding. Specifically, adopting a self-distanced perspective when processing anger has been found to lead to lower levels of emotional reactivity compared to a self- immersion (Ayduk & Kross, 2010; Kross et al., 2005), even directly following provocation (Mischkowski et al., 2012).

According to Kross and Ayduk (2009), self-distancing facilitates a shift away from recounting emotionally arousing details in a self-immersed and possibly impulsive manner toward reconstruing experiences in ways that may promote insight and closure. Indeed, recent meta-analytic evidence indicate that self-distancing techniques, as induced in the lab, is associated with a small-to-moderate advantage relative to self-immersion in relation to various adaptive outcomes, particularly when self-distancing from emotionally salient stressors (g = 0.45; Murdoch et al., 2023). Techniques such as mindfulness-based decentering, cognitive defusion, and nonreactive awareness illustrate concrete ways of shifting from a self-immersed to a self-distanced perspective (see Bernstein et al., 2015 for a detailed review of decentering strategies).

In sum, while findings regarding mode of processing (abstract vs. concrete) remain somewhat mixed, there is more consistent support for the importance of vantage point and perspective when processing emotions, with a self-distanced vantage perspective potentially being a key facilitator of adaptive processing of anger.

Methodological Considerations

This section outlines key strengths and limitations related to study design, causal inference, and analytical approaches across the included studies. It also addresses measurement issues, particularly the heavy reliance on self-report, the primary reliance on between-person data, and limitations in generalizability related to the samples used in the present thesis.

Study Design and Causal Inference

Across studies in the present thesis, there is considerable variation in design, each with distinct strengths and limitations. Studies I and II employed cross-sectional designs, which inherently preclude conclusions about causality or temporal ordering. For example, in Study I, demographic and clinical characteristics were specified as predictors and emotion regulation strategies as distal outcomes in the latent profiles; however, in the absence of temporal information, this distinction is ultimately arbitrary, as only associations can be inferred. Similarly, edges in the network model in Study II reflect conditional associations and should not be interpreted as causal or directional.

Nevertheless, most studies using latent profile analysis (LPA), as in study I, relies on cross-sectional data to identify latent subgroups (Berlin et al., 2013; Spurk et al., 2020). Indeed, when the aim is not to examine the development of subgroups over time, cross-sectional designs may be particularly well-suited for identifying unobserved population heterogeneity, without the added complexity of modeling within-person dynamics. Moreover, when interpreted in light of prior theory and empirical findings, exploratory approaches such as cross-sectional network analysis (Study II) can serve as a basis for generating testable causal hypotheses, a commonly emphasized strength of the network framework (Borsboom & Cramer, 2013; Robinaugh et al., 2020).

At the same time, the cross-sectional and exploratory nature of studies I and II underscores the need for confirmatory and longitudinal replication. In particular, future research should examine whether the anger-in subgroup identified in study I, replicate across other

independent samples. Moreover, longitudinal and intensive repeated-measures designs (e.g., daily diary or ecological momentary assessment) may also be important for testing hypothesized dynamic processes suggested by the network results, such as whether suppression at one time point predicts subsequent increases in rumination or cognitive activation (e.g., hostility) at another, which in turn may lead to additional suppression attempts (i.e., a self-reinforcing feedback loop).

In contrast to Studies I and II, Studies III and IV employed repeated measurements over time, allowing for temporal separation between putative causes and effects—one fundamental requirement for causal inference[10] (Shadish et al., 2002). In Study III, this was further strengthened by an experimental design in which both anger induction and subsequent suppression were systematically manipulated.

However, the absence of a non-suppression control condition during the within-subject phases (1–4) in Study III limits causal inference, as alternative explanations cannot be fully ruled out. For example, observed reductions in anger may reflect natural emotional decay or habituation to the recalled anger memory, rather than the effects of suppression per se. Additionally, small elevations in anger following suppression (phase 3) and during the subsequent free-recall phase (phase 4) suggest that minor rebound effects may be present. Importantly, however, mean anger levels during these phases were very low ($M_{\text{State anger t3}} = 1.52$; $M_{\text{State anger t4}} = .73$; range: 0-10), indicating that any potential rebound were likely minimal and of limited practical significance.

In the pilot treatment study (Study IV), we employed an interrupted time series design, a robust quasi-experimental approach commonly used when true experimental designs (e.g., randomized controlled

[10] Three commonly cited criteria for making causal statements are: (1) the variables of interest should covary, indicating a statistical association; (2) the putative cause must precede the effect in time (temporal precedence), and, ideally, the cause should be experimentally manipulated; and (3) plausible alternative explanations should be ruled out (Shadish et al., 2002).

trials) are not feasible (Shadish et al., 2002), as was the case in this pilot feasibility study. Specifically, we compared a prolonged baseline phase to a treatment phase, each with four weekly assessments. The stable baseline phase served as a counterfactual trajectory, approximating what would have occurred in the absence of treatment. By comparing growth trajectories across the baseline and treatment phases, we estimated phase-specific effects. Although the prolonged baseline helped control for many threats to internal validity, such as maturation and testing effects, the absence of a control group and randomization remains a limitation. We attempted to minimize the influence of some alternative explanations by, for example, excluding participants undergoing concurrent psychological treatment and those who had recently changed psychotropic medication within the past two months (see Study IV exclusion criteria). However, other unmeasured confounders, expectancy effects, and non-specific factors (e.g., attention) cannot be ruled out (Kazdin, 2023).

Analytical Considerations

A notable strength of the present thesis is the alignment of analytical methods with the specific study designs and research questions, using state-of-the-art analytical methods.

Study I

In study I, we followed best-practice recommendations for latent profile analysis (LPA) to empirically identify anger subgroups (Nylund-Gibson & Choi, 2018). Unlike traditional clustering methods (e.g., k-means), which rely on heuristic or arbitrary decisions, LPA provides formal evaluation of model fit and classification uncertainty (Nylund-Gibson & Choi, 2018). It also allows validation of subgroups against relevant variables, such as emotion regulation strategies, without influencing profile formation (Asparouhov & Muthén, 2014; Nylund-Gibson & Masyn, 2016).

With regards to sample size and power, sample size requirements in LPA are influenced by factors such as the rarity of a subgroup, the degree of class separation, and the number of indicators and classes (Nylund-Gibson & Choi, 2018; Tein et al., 2013). As we had no prior

information regarding several of these parameters, no a-priori power analysis was conducted. Nevertheless, the final sample size (N = 534) falls within commonly recommended ranges for robust estimation and reliable use of fit indices in LPA (N $\approx$ 300-1000; Nylund-Gibson & Choi, 2018).

Study II

In Study II, we followed state-of-the-art recommendations for estimating cross-sectional networks, including the evaluation of robustness and stability of the derived networks and clusters (Epskamp et al., 2018; Golino & Epskamp, 2017). Additional sensitivity analyses were also conducted to assess the risk of type II errors by comparing different degrees of regularization, i.e., the strictness with which small associations (edges) were removed.

As in Study I, no formal power analysis was conducted, reflecting the absence of consensus on power estimation in network models, which depends on multiple factors such as the number of nodes, edge strength, network density, and clustering structure (Constantin et al., 2023; Epskamp et al., 2018). This represents a potential limitation, as power constraints may affect stability and reliability. Indeed, two of the three centrality metrics (i.e., indices of node influence within the network)—betweenness and closeness—were unstable and therefore not interpreted. However, as noted by Bringmann et al. (2019) such instability is common, as these metrics are highly sensitive to sampling variability. In contrast, strength centrality—reflecting the overall magnitude of a node's direct connections with other nodes—tends to be more stable and interpretable, which was also the case in our networks. As such, we solely focused on the strength centrality metric.

Study III

In Study III, linear mixed models were used to examine within-subject effects (i.e., suppression-related effects), and ANCOVAs were used to analyze between-subject effects (i.e., post-suppression regulation effects). A key analytical challenge concerned the HRV data, which was positively skewed—a common characteristic of such measures

(Laborde et al., 2017). To assess the robustness of the HRV findings, sensitivity analyses were conducted by excluding extreme outliers, log-transforming rMSSD scores (i.e., HRV scores), and rerunning the analyses using generalized linear mixed models (GLMM) with a Gamma distribution and log-link function to account for the skewed data. These analyses largely confirmed the main ANCOVA results (see supplemental Study IV), supporting the robustness of the findings.

The sample for study IV was relatively large with 203 participants in total, which exceeded our a-priori power calculations. Indeed, it was estimated that 192 participants would be sufficient to detect a medium-sized (d = 0.45) between-condition effect with 80% power.

Study IV

A key strength of study IV was that we followed specific recommendations for estimating piecewise latent growth models (LGM) adapted for interrupted time series data (Duncan & Duncan, 2004). This allowed for the estimation of separate growth trajectories for the baseline and treatment phases, enabling comparisons of both level and slope changes in these phases as well as estimation of phase-specific effects.

An analytical limitation of this study, however, was the relatively small sample size, which restricts the precision of the effect estimates and suggests that findings should be considered preliminary. Nevertheless, the a-priori Monte Carlo simulation indicated that at least 30 participants were required to detect a standardized mean difference of d = 0.74 between the baseline and treatment phases for anger-in (AX-I), with adequate power (> .80, α = .05, two-tailed), while controlling for baseline level and trend. The final sample (N = 38) exceeded that target.

Measurement Considerations

As is common in psychological research, all four studies in this thesis relied primarily on self-report data, which introduces the risk of shared method variance—that is, correlations in measurement error across measures due to using the same measurement method (e.g.,

self-report; Kazdin, 2024). Shared-method variance may lead to inflated associations and biased estimates of results (Kazdin, 2024). Self-report data is also susceptible to social desirability bias. However, several features of the present studies likely mitigated some of these concerns. For example, all participants were informed that their responses to the questionnaires would be treated anonymously, reducing evaluation apprehension, Moreover, responses showed substantial variability across anger and emotion regulation measures, often covering the full range of the scale, potentially suggesting reduced influence of socially desirable responding.

With regards to shared method variance, latent variable modeling in Studies I and IV reduced the amount of shared measurement error by isolating variance common to multiple observed measurements (e.g., profile indicators as in study I) into a latent variable, thus potentially reducing the risk of inflating associations. Similarly, in study II we reduced the risk of estimating spurious associations (i.e., type I errors) by using lasso-regularization, which shrinks small associations to zero. Finally, and perhaps the strongest "remedy" to shared method variance, Study III incorporated a multi-method approach, including self-report, behavioral, and physiological measures of anger. This triangulation allowed us to examine whether patterns of results converged across different types of measurements, strengthening confidence that observed effects reflected true phenomena rather than method artifacts—which, in this case, they largely did.

Moreover, although Spielberger's (1999) Anger Expression Inventory, which was used across all four studies, is one of the most widely used and validated measures of anger expression (anger-out and anger-in), several limitations should be noted. First, the anger-in/anger-out dichotomy has been criticized as overly narrow and may fail to capture other relevant expression styles, such as assertive anger expression (Linden et al., 2003). Second, as noted in the introduction, the anger-in scale does not clearly distinguish between the act of suppression and the potential consequence of rumination (Burns et al., 2008a). Lastly, Spielberger's framework implicitly assumes that higher levels of anger-in or anger-out are maladaptive per se, whereas contemporary accounts emphasize that adaptiveness is better understood in

terms of functional consequences (e.g., interpersonal outcomes, goal attainment, and social functioning) rather than expression direction alone (Gardner & Moore).

Relatedly, in Study III the anger-in scale was used as the primary outcome, with anger rumination and anger assertion included as secondary outcomes. While this ensured a focused examination of central constructs, it may also have limited the assessment of broader functional consequences, such as interpersonal functioning and social well-being. At the same time, including a larger number of outcomes would have introduced important methodological trade-offs. Specifically, multiple testing increases the family-wise Type I error rate (i.e., false positives), which in turn typically requires more conservative inferential adjustments (e.g., correction for multiple comparisons). Such corrections in turn may reduce statistical power for individual tests and may thereby, instead, increase the risk of Type II errors (i.e., false negatives). As a result, effects of particular relevance (e.g., anger-in) may be missed, particularly in small-sample contexts such as in study III (N = 38).

Individual Differences and Heterogeneity

As indicated by Study I, where distinct anger response profiles were identified, maladaptive anger likely manifests differently across individuals. However, with the exception of Study I, the present thesis primarily relied on aggregated between-person data, which may obscure meaningful heterogeneity in anger responding and anger regulation.

This limitation relates to a broader methodological development in psychological science highlighting the distinction between between-person and within-person processes. In particular, the so called *ergodicity problem* suggests that associations observed at the group level cannot be assumed to generalize to individuals unless highly restrictive assumptions are met, such as stationarity (i.e., the observed processes do not change over time) and homogeneity of responding (Fisher et al., 2018; Molenaar, 2004). Instead, it has been suggested that many psychological processes are better captured by modeling the individual person-data over time (Hamaker, 2025), or at least by,

in some way, taking into account individual heterogeneity within the data.

For example, one possible approach in Study II would have been to estimate separate network models for the anger-in profile and the high anger profile identified in Study I. Such an approach may have allowed for a more nuanced investigation of subgroup-specific associations among anger and emotion regulation-related processes. However, given the relatively modest subgroup sample sizes (Anger-in Profile n = 91; High Anger Profile n = 101) in combination with the large number of network nodes included (16 nodes), such analyses would likely have resulted in insufficient statistical power and unstable network estimation (Constantin et al., 2023).

Similarly, although Study III included repeated measurements across 10 time points, the analyses primarily focused on aggregated changes in levels and slopes across the baseline and treatment phases. An alternative approach could have involved more explicitly idiographic or single-case experimental analyses examining individual trajectories of change over time. For instance, participants could have been randomized to baselines of varying lengths (e.g., 2, 3, or 4 weeks), a design feature commonly used in multiple-baseline single-case experiments to strengthen causal inference by controlling for time-related confounds (Kazdin, 2024). Moreover, as briefly noted in Study III (see article), the data collection also included two 7-day bursts of daily assessments of anger regulation dynamics, one pre- and one post-treatment. These data could in future work be modelled using idiographic or multilevel time-series approaches, such as dynamic structural equation modelling (DSEM; Hamaker et al., 2023), which allows for the separation of within-person dynamics from between-person differences and their change over time

Overall, these considerations highlight the importance of complementing group-level inference with methods capable of capturing within-person dynamics and heterogeneity in anger responding and regulation.

Constraints on Generality

A notable strength across the studies is the inclusion of relatively large and heterogeneous samples, particularly in Studies I–III, comprising both men and women across a broad age range and with varying manifestations of anger problems. At the same time, important constraints on generalizability remain. The samples were predominantly drawn from Western, educated, industrialized, rich, and democratic (WEIRD) contexts, and findings may not readily extend to non-WEIRD populations, nor to children and adolescents. Moreover, Study IV relied on a smaller and more demographically restricted sample (primarily middle-aged, well-educated women), further limiting generalizability and the strength of conclusions regarding treatment efficacy.

Another limitation regarding generalizability concerns the experimental manipulations in Study III. While suppressing a recalled angry memory in the lab may approximate the suppression of intrusive angry thoughts, it likely differs from suppressing anger in response to real-life interpersonal provocations. Future research could use daily diary or ecological momentary assessment designs to better capture the naturalistic dynamics of anger, anger suppression and other relevant anger-regulation strategies.

Main Conclusions

The overarching aims of this thesis were to advance understanding of maladaptive anger inhibition in adults (aim 1) and to contribute to the development of more effective internet-delivered psychological interventions for adults who self-report maladaptive anger inhibition tendencies (aim 2).

Using cross-sectional, experimental, and within-subject treatment evaluation designs, the findings suggest that maladaptive anger inhibition indeed constitutes a clinically relevant problem potentially in need of psychological treatment. Specifically, results indicate that a subset of anger-treatment individuals is characterized by elevated anger suppression and rumination, alongside relatively low levels of aggressive and anger-out tendencies (i.e., maladaptive anger inhibition). Moreover, in examining how components of anger interact, anger suppression and anger rumination emerged as central and potentially interrelated processes. Extending this, the experimental study on anger suppression suggested that a sequence of suppression followed by rumination was particularly maladaptive, being associated with increased anger and less adaptive physiological responding. However, there was limited evidence that this regulatory sequence was driven by a rebound effect of suppression per se. In fact, suppression alone was associated with relatively large immediate reductions in self-reported anger and generally adaptive physiological changes, and similar patterns were observed when suppression was followed by either continued suppression or reappraisal. This might be suggestive of that rumination, rather than suppression, may represent a key maintaining process in maladaptive anger inhibition.

Perhaps most importantly, findings in the present thesis indicate that maladaptive anger inhibition is amenable to psychological treatment. Our brief internet-delivered treatment targeting emotion regulation processes (eCREAT) showed good feasibility and acceptability, and was associated with substantial improvements in anger-in, anger rumination, and constructive anger expression (i.e., anger assertion).

Future Directions

Despite these promising results more work is clearly needed. First, future research should further develop and refine psychological treatments for maladaptive anger inhibition, ideally beginning by, for example, comparing whether our newly developed emotion regulation treatment (eCREAT) is superior to more generic emotion-regulation treatments not specifically tailored to maladaptive anger inhibition (e.g., the MEA + CR treatment by Bjureberg et al., 2023). Moreover, more basic research would benefit from exploring more constructive ways of expressing and processing anger that does not fall prey to the counterproductive effects observed in the anger venting and anger rumination literature (e.g., Bushman, 2022; Denson, 2013; Lohr et al., 2007).

Second, to better understand the potential negative consequences of excessive suppression, future research should examine the relationship between maladaptive anger inhibition and aggression. As reflected in lay expressions such as "the straw that broke the camel's back," chronic suppression of anger is often assumed to culminate in sudden, disproportionate outbursts, sometimes directed at uninvolved others. Although this phenomenon has been extensively studied within the triggered displaced aggression literature (Lagios et al., 2025)—where the inhibition of an initial angry impulse is followed by an intensified response to a subsequent minor provocation—little research has examined these processes among individuals with habitual tendencies to suppress anger (i.e., maladaptive anger inhibition). Future studies would therefore benefit from investigating triggered displaced aggression in this population, exploring for instance the moderating effect of trait anger-in versus anger-out.

Third, advancing the assessment of maladaptive anger inhibition will require the development of improved measurement approaches, including both self-report and experimental paradigms that more precisely capture its underlying processes (e.g., anger suppression and anger rumination). In particular, this calls for theory-driven measures that can more effectively assess and distinguish between key processes involved in maladaptive anger inhibition (e.g., anger suppression and anger rumination), as well as further experimental and naturalistic

research examining the intra- and interpersonal consequences of persistently suppressing anger while maintaining high levels of internal anger activation.

Finally, I call for the development of an empirically informed typology of maladaptive anger inhibition. This could, for example involve empirically verifying my two proposed subtypes of maladaptive anger inhibition: the suppressed anger directed inward subtype and the suppressed anger directed outward subtype. Importantly, future research should clarify whether these subtypes can be reliably distinguished and, in particular, whether the inward-directed subtype represents a distinct anger-related phenomenon or is better conceptualized within existing frameworks for internalizing problems (e.g., depression and anxiety disorders). It is also important to determine whether the outward-directed subtype primarily reflects withdrawal tendencies or, as proposed here, also involve strong approach and aggressive action tendencies that, although mostly inhibited, may nonetheless manifest covertly in the form of aggressive fantasies, revenge-related cognitions, and, at times, episodic outbursts of aggression.

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