THE TIME COURSE OF ANGER:

AN EXPERIMENTAL INVESTIGATION

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ABSTRACT

Conceptualizations of anger have suffered from a lack of research investigating the temporal dynamics of anger episodes. Furthermore, though some studies have provided valuable insights into the time course of anger, no study to date has utilized a standardized laboratory paradigm designed to mimic an interpersonal provocation. The purpose of this study was to characterize the time course of the affective, physiological, and behavioral components of anger in response to a standardized provocation. Our second aim was to assess potential effects of trait anger, trait aggression, trait hostility, and emotion regulation deficits on the time course of the different components of anger. Participants (n = 82) engaged in the Modified Taylor Aggression Paradigm (MTAP), a laboratory measure of anger/aggression in which provocation is manipulated by varying electric shocks selected for the participant by an (unbeknownst to the participant) fictitious opponent. This study utilized a modified version of the classic TAP that simulated an acute interpersonal provocation that one might encounter in the "real world." Subjective anger, physiological arousal (as evidenced by heart rate [HR], galvanic skin response [GSR], and high-frequency heart rate variability [HF HRV]), and the behavioral expression of anger (aggression) were measured throughout the task before, during and after provocation. Consistent with previous research, results showed that the rise time to peak levels of most outcome variables was significantly faster than the return time from peak back to baseline. Additionally, results showed that the majority of the time course variables were not correlated with one another providing evidence for the idea that different components of anger have independent time courses. Contrary to

our hypotheses, trait variables were largely unrelated to time course variables. The current study provides further evidence for the relationship between the rise time and return time in the time course of subjective, physiological and behavioral manifestations of anger using a standardized and ecologically valid provocation task.

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CHAPTER 1

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The Time Course of Anger: An Experimental Investigation

Anger is a basic human emotion and near-universal experience. One reason we have sought to understand anger is its impact on the world around us. Anger has been consistently linked to negative outcomes including poor interpersonal relationships (Tescher, Conger, Edmondson, & Conger, 1999) and occupational stress and burnout (Sloan, 2004). Chronic anger is also associated with several physical and psychological health consequences including increased levels of depression and anxiety (Tescher et al., 1999), as well as hypertension and coronary heart disease (Goldstein & Niaura, 1992; Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003; Sirois & Burg, 2003). When anger leads to aggression, deleterious effects worsen and may lead to damaged relationships, increased rates of alcohol use, legal problems, and vocational difficulties (Deffenbacher, Oetting, Lynch, & Morris, 1996). Despite anger's ubiquity and its significant public health impact, aspects of this basic emotion remain understudied. While research to date has delineated the different facets of anger, our knowledge of the way in which these components unfold over the course of a provoking event remains limited.

While there is some debate (see Potegal & Stemmler, 2010 for more detail), contemporary conceptualizations typically partition anger into four components - subjective/experiential, behavioral, physiological, and cognitive (Averill, 1983; Deffenbacher, 2011; Novaco, 1977; Potegal & Stemmler, 2010), which has been

supported by factor analyses (Buss & Perry, 1992; Spielberger, 1999). The subjective, or "affective", component refers to an individual's qualitative experience of anger. The behavioral component is conceptualized as the "action urge" that accompanies angry emotions. While this urge can sometimes be to withdraw from a situation or ruminate on the emotion (Spielberger, Krasner, & Solomon, 1988), the primary action urge associated with anger is aggression (e.g., Berkowitz, 1989). The physiological component refers to the activation of the autonomic nervous system (ANS) implicated in regulating biological processes such as blood pressure and heart rate during an emotional event (Scarpa & Raine, 1997). An increase of angry emotions is linked to the activation of the sympathetic nervous system (Funkenstein, 1955) causing heightened blood pressure, increased heart rate (Funkenstein, King, & Drolette, 1954; Schachter, 1957), and an increased number of galvanic skin responses (Scarpa & Raine, 1997). Activation of the parasympathetic nervous system is implicated in down regulation of biological processes and regulation of anger (Thayer & Lane, 2000). In particular, researchers have been able to link high-frequency heart rate variability (HF HRV) to parasympathetic responding and have utilized this variable to look at regulation of negative emotion states (Vaschillo et al., 2008). Lastly, the cognitive component is comprised of the appraisals and attributions made during a provocation (Novaco, 1977). Appraisals about the triggering event often have an impact on how the angry emotion manifests (if at all). While research thus far has done well to characterize the different components of an anger response, less is known about how these processes change over time during a provocation.

"Affective chronometry," the study of the time course of affective reactions, consists of four key components–threshold, peak/amplitude, rise time to peak, and recovery time (Davidson, 1998). The threshold is the amount of a particular stimulus that an individual needs to trigger an affective response. The peak or amplitude is the highest level of a particular emotion that one experiences after a triggering event. The rise time to peak and recovery time correspond to the rise and fall of the emotion from baseline and back again. The sparse research on the time course of anger suggests that the peak of a "typical" anger response is about 50% of maximum possible intensity– a "five out of ten" (Beck & Fernandez, 1998; Fernandez & Beck, 2001; Jacob et al, 2008; Stets & Tsushima, 2001). Provocation is strongly associated with anger intensity, with more intense provocations resulting in more intense anger and aggression (Ferguson & Rule, 1983; Santor, Ingram, & Kusumakar, 2003). Contextual factors such as hot temperatures (Anderson & Anderson, 1996) and acute intoxication (Bushman, 1993; Giancola, 2013; McCloskey, Berman, Echevarria, & Coccaro, 2009) have also been shown to increase subjective anger intensity in response to provocation. The "typical" duration of an angry episode has proven to be extremely variable, ranging from "a few minutes" (Frijda, Mesquita, Sonnemans, & Van Goozen, 1991) to several hours (Fridhandler & Averill, 1982; Simon & Nath, 2004; Stets & Tsushima, 2001). Within anger episodes, time to peak anger is almost always more rapid than recovery time (return to baseline) (Potegal, Kosorok, & Davidson, 1996; Potegal, Kosorok, & Davidson, 2003; Potegal & Qiu, 2010; Qiu, Yang, & Potegal, 2009) with some studies showing an average rise to peak anger occurring within 30 seconds after provocation (Beck & Fernandez, 1998; Fernandez &

Beck, 2001). However, there is no standardized provocation task in the time course literature, limiting our ability to generalize findings and account for discrepancies found across studies. Furthermore, few studies have considered the effects of individual differences on the time course of anger.

Heterogeneity between individual time courses are potentially due to the influence of general predispositions toward anger, aggression, and hostility, as well as one's overall ability to regulate emotions. These trait variables have been used frequently to explore differences between individuals in anger related constructs (Deffenbacher, 1999) and there are clues from the research suggesting that these variables could potentially influence individual time courses. For example, individuals high in trait anger experience higher peaks of subjective anger during a provocation (Spielberger, Reheiser, & Sydeman, 1995). Those high in trait aggression also report more frequent and more intense anger episodes suggesting lower thresholds for anger and higher peaks once provoked (McCloskey, Berman, Noblett, & Coccaro, 2006). The dynamic elements of the time course also show susceptibility to influence by trait variables. For example, greater trait hostility predicts slower rates of physiological recovery from anger (Fredrickson, 2000; Jacob et al., 2008). Similarly, individuals with greater emotion dysregulation report experiencing a faster build to anger and a longer recovery period (Beckham et al., 2002; Linehan, 1993). Although these studies have provided us with valuable insights, directly investigating the relationship between trait variables and the time course of anger using a standardized laboratory provocation would allow us to predict an individual's experience

of anger with more reliability. This could have important implications both for violence risk assessment and treatment of problematic anger and aggression.

Our limited understanding about the time course of anger is based predominately on retrospective self-report, which, though useful, has significant inherent limitations (Henry, Moffitt, Caspi, Langley, & Silva, 1994). Furthermore, anger studied experimentally via exposure to pictures or movies (e.g. Tottenham, Borscheid, Ellertsen, Marcus, & Nelson, 2002; Gross & Levenson, 1995) is often removed from the interpersonal context in which anger typically takes place (Berkowitz & Donnerstein, 1982; Deffenbacher & McKay, 2000; Kassinove & Tafrate, 2002). Relatedly, time course studies of anger typically only assess the subjective component of anger while failing to consider how this aspect of anger does (or does not) covary with behavioral and physiological components of anger. As such, obtaining subjective, physiological and behavioral anger responses in a real time interpersonal context would provide a more accurate and nuanced understanding of the experience of anger. This would require the development of a standardized laboratory anger-provocation task to allow for interpretability across studies, as to date, no such task exists. Validated laboratory tasks of reactive aggression such as the Taylor Aggression Paradigm (TAP: Taylor, 1967) have been developed. Though these tasks include interpersonal provocation and increase anger (Giancola & Parrott, 2008), the ordering of the provocation does not allow for the establishment of baseline values nor does it have a period after high provocation for the participant to return to baseline levels. As such, the typical TAP would not allow for a full assessment of the time course of anger and aggression. However, if modified, this

task may serve as an effective standardized way to assess the time course of anger in response to an ecologically valid provocation.

The current study analyzed the time course of subjective (anger rating), physiological (autonomic arousal), and behavioral (aggression) components of provoked anger. Participants completed a series of interviews and questionnaires. Participants then completed a modified version of the TAP in which trials were meant to simulate a typical provocative interaction, escalating from pre (no shock) provocation to peak (high shock) provocation before de-escalating to post (no shock) provocation. While engaging in the TAP, subjective anger was assessed on a 0-10 scale after each trial. Overall autonomic responding was represented by heart rate while sympathetic and parasympathetic responding were assessed via number of galvanic skin responses and HF HRV respectively. TAP aggressive behavior was assessed as the level of opponent shock selected by the participant at each trial. Our aim was to (1) characterize the time course of the subjective, physiological, and behavioral components of anger in response to a "typical" (standardized) provocation, and (2) examine the influence of putatively related trait constructs such as trait anger, trait aggression, trait hostility, and emotion regulation on the intensity and time course of anger. We hypothesized that subjective, physiological, and behavioral components of anger will rise from a baseline level to peak levels more quickly than they fall from a peak level back to baseline (the "build" to anger will be faster than the "cool-down" period). We also hypothesized that individuals higher in trait anger, trait aggression, and trait hostility and lower in emotion regulation would exhibit

quicker increases to peak levels of each of the components assessed, higher peaks of those components, and slower returns to baseline levels.

Methods

Participants

Participants consisted of 82 undergraduate students (65.8% female) recruited from a large urban university. Participants were offered three research credits for their participation. Participants in the study ranged from 18-43 years old (M = 22.05, SD = 4.51), were primarily Caucasian (58.2%), African American (22.8%), and Asian/Other (19.0%), and were predominantly non-Latino (93.7%). Participants were first screened for the study over the phone by trained assessors in the lab. Participants were not invited to take part in the study if they reported (a) age under 18 or over 45, (b) current drug or alcohol dependence, (c) a history of a psychotic disorder, (d) current (past month) psychopharmacotherapy, or (e) current pregnancy. Informed consent was obtained for all participants.

Measures

Laboratory Task.

Modified Taylor Aggression Paradigm *(MTAP).* The MTAP is a slight modification of the Taylor Aggression Paradigm (TAP: *Taylor, 1967*), a classic laboratory measure of retaliatory aggressive behavior. In the TAP, the participant competes against a fictitious opponent in a reaction-time game during which electric shock is administered and received. Before each trial, the participant selects a shock level for the opponent to receive should the participant have a faster reaction-time on that trial.

Aggression is defined as the intensity of shock selected. Provocation is manipulated by having the ‘‘opponent’’ select increasingly intense shock levels for the participant over the course of the TAP.

Before each trial on this task, the participant (and ostensibly the opponent) presses one of 12 buttons on a panel to select a shock level (0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, or 20), and the person (participant or opponent) who had the slower reaction time on that trial receives the shock set by the other person before the trial. The faster person does not receive a shock, but sees what shock level the other person had set. The 10 shock is equivalent to the tolerance-threshold current. The 9 shock is set at 95% of this maximum, the 8 shock at 90%, and so forth. Participants are informed that the 20 setting will administer a ‘‘severe’’ shock ‘‘twice the intensity’’ of the tolerance threshold (however, this shock is never administered to the participant in this current study). For the ‘‘0’’ response, no shock is administered. This non-aggressive response option was included to increase the ecological validity of the task. A rich literature supports the validity of the inferences that can be drawn from the TAP and related laboratory measures of physical aggressive behavior (Anderson & Bushman, 1997; Giancola & Chermack, 1998; Giancola & Parrott, 2008).

The current study used a slightly modified version of the TAP in which, rather than the traditional continually escalating pattern of shocks, the levels of opponent shocks (provocation) were intended to follow the course of a “typical” altercation with a pre-provocation baseline, a sharp build to peak provocation and a sharp decrease after, followed by a “cooling off” period. To achieve this effect, the MTAP consisted of 36

reaction time trials broken into nine 4 trial "blocks." The first two blocks were made up of eight trials consisting of all "0" shocks. The first block was meant to allow participants to acclimate to the task and allow the experimenter to clarify directions if need be. The second block was utilized as the baseline period for the time course variables measured throughout the task. This was followed by a four-trial transition block meant to mimic the beginning and escalation of a provocation. Shocks during this trial ranged from four to seven in an increasing pattern (i.e. 4, 5, 5, 7). The next two blocks consisted of high level shocks between eight and ten, simulating a provocation (during this block, the opponent set a "20" shock for the participant on two trials but participants were never shocked at this level as the participants always won these trials). Following the provocation blocks, there was another four trial transition block signifying the de-escalation of the provocation. Shocks during this block ranged from seven to one in a decreasing pattern. The final three blocks consisted of twelve trials of "0" shocks and simulated a period of time where the stressor, the provocation, abated. The shock settings during the MTAP were designed to mimic a real-life provocation that one might find in an interpersonal context. This task served as our experimental provocation, and the shocks selected by the participant were our measures of aggressive responding.

Questionnaires.

Visual Analogue Scale (VAS: Miller & Ferris, 1993). A visual analogue scale was used before, during, and after the Taylor Aggression Paradigm as our measure of subjective anger. The scale used both before and after the MTAP contained fifteen (15) emotions and bodily sensations, for each of which the participant rated their current level

of that emotion or sensation on a 0-100 scale. During the MTAP, the VAS was modified to contain just three emotions (anger, happiness, and fear) rated on a 0-10 scale and was completed after each MTAP trial. These responses were spoken out loud during the task by the participants, audio recorded, and then entered into a database. This served as the primary self-report measure of current mood and specifically, levels of subjective anger throughout the task. The VAS is a commonly used, reliable, and valid measure of subjective affect (Miller & Ferris, 1993).

Buss Perry Aggression Questionnaire (BPAQ; Buss & Perry, 1992). The BPAQ is a self-report measure of trait aggression. The BPAQ consists of 29 items each scored using a 4-point Likert-type scale. The BPAQ consists of four scales: physical aggressiveness, verbal aggressiveness, anger, and hostility (i.e., suspiciousness and resentment). The BPAQ shows good internal consistency (scale alphas between .72 and .85) with a total score alpha of .89 and good test-retest reliability (r = .80) after 9 weeks. BPAQ scores also correlate with measures of aggressive behavior and personality traits associated with aggression (Buss & Perry, 1992). The physical aggression and verbal aggression scales were combined in the current study to create a measure of trait aggression. Scores on the scales of the BPAQ were used as our measures of trait anger, aggression, and hostility.

The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004). The DERS is a 36-item self-report questionnaire that was used to assess emotion (dys) regulation. The DERS assesses dysregulated emotion across six domains: non-acceptance of emotional responses, difficulties engaging in goal-directed behavior, impulse control

difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity. Each item is rated on a 5-point Likert scale ranging from almost never to almost always, with higher scores representing more emotion dysregulation. The DERS has demonstrated strong internal consistency ($\alpha = .93$) and test-retest reliability ($r = .88$ after 1-2 months) as well as adequate construct and predictive validity as evidenced by its correlation to behavioral outcomes such as intimate partner violence and deliberate self-harm (Gratz & Roemer, 2004). The overall score on the DERS was used as our measure of trait emotion regulation.

Post-TAP Questionnaire. Participants answered questions about their experience completing the TAP including questions about the levels of shock they set or received, whether they felt that their opponent had a strategy, and how their reaction time may have been affected by the competition. This questionnaire is given to increase the believability of the task as well as assess whether the participant believed the deception inherent in the task.

Psychophysiological Assessment.

The physiological component of the anger response was assessed using the Biopac Systems MP150 acquisition system and recorded using AcqKnowledge software version 3.8.1. The Biopac System is currently the gold standard in the acquisition of physiological data across a variety of research settings. The AcqKnowledge software provides an interface for collecting, storing, and analyzing the data. Heart rate data were transferred from AcqKnowledge into the Kubios-HRV program for HRV analyses (Tarvainen, Niskanen, Lipponen, Ranta-aho, & Karjalainen, 2014).

Heart Rate and Heart Rate Variability. Heart rate was our measure of overall autonomic functioning. Heart rate is modulated by both the sympathetic and parasympathetic branches of the ANS and is widely used as a measure of overall autonomic functioning (Cacioppo, Tassinary, & Berntson, 2007). Heart rate was chosen over other measures of ANS functioning such as respiratory rate and blood pressure due to ease of collection and interpretation, robustness against artifacts in the data, and ability to be calculated over short time intervals. Participants had their heart rate monitored throughout the task using the electrocardiogram amplifier module (ECG100C). The ECG100C is a single channel, high gain, differential input, biopotential amplifier designed to obtain measures of heart rate. The module uses a 3-lead system and EL504 1" square cloth solid gel disposable snap electrodes were used in conjunction with the LEAD110/LEAD110S pinch leads. Respiration rates were collected throughout the task utilizing the RSP100C respiration amplifier with the respiration belt attachment.

Because heart rate is a measure of overall autonomic functioning, the specific components of the autonomic system, the sympathetic and parasympathetic branches, were assessed utilizing galvanic skin response (described below) and high frequency heart rate variability (HF HRV), respectively. HF HRV measures the variability in heart rate that occurs over short periods of time as the circulatory system responds to internal and external stimuli and corresponds to parasympathetic regulation/activity while other indices of HRV such as low and very low frequency HRV represent a mix of both sympathetic and parasympathetic responding (Vaschillo et al., 2008). HF HRV was calculated utilizing the Kubios-HRV program. Because HRV requires extended periods

of time to be calculated reliably, measurements were taken over the course of four trials (one block).

Galvanic Skin Response (GSR). GSR served as our primary measure of sympathetic nervous system response during the anger provocation. GSR is a measure of the electrical conductance of the skin. When a person is under stress, there is an increase in the level of moisture on the skin (i.e., sweat), and GSR is sensitive to this change.. GSR was measured using the GSR100C electrodermal activity amplifier. The GSR100C is a single channel, high gain, differential amplifier designed to measure skin conductance via the constant voltage technique. GSR was collected using two TSD203 Ag-AgCl finger electrodes. Because individual skin responses take time to complete their cycle, GSR was also calculated over the course of four trials (one block). The number of GSRs per block was calculated utilizing the AcqKnowledge software using a conservative threshold value of .05 μS in accordance with parameters laid out by Dawson, Schell, & Filion (2007).

Procedure

Participants were recruited from Temple University's SONA System providing research opportunities to undergraduate students in return for research credits, often required for their psychology courses. Participants who expressed interest in the study were initially contacted via telephone by trained research staff for pre-screening of inclusion/exclusion criteria. Participants who meet inclusion criteria were invited to come into the laboratory.

Upon entering the lab, participants read an informed consent, demonstrated

understanding, and then signed the form. Participants provided a urine sample for drug and pregnancy testing. Positive tests excluded participants from participating. Participants then took part in a series of interviews designed to gain background demographic information. Following the interviews, participants filled out the self-report questionnaires consisting of the BPAQ, DERS, and VAS.

During the next portion of the visit, participants engaged in the MTAP. First, a research assistant attached the breathing belt around the chest of the participant. Then, two ECG leads were attached to the outside of each upper arm and one lead to the inside of the left wrist. Next, the GSR100C was attached which included two fingertip skin conductance sensors placed on the middle and index finger of the non-dominant hand. For the MTAP, fingertip electrodes were attached to the index and middle fingers of the dominant hand. The participant was told that he or she would be competing in a task against another person in the adjoining room. The participant was given ten minutes before the MTAP began to provide time to acclimate to having the psychophysiological equipment attached and to obtain baseline measurements of ANS functioning. During this time, participants watched a neutral stimulus video (an episode of “How It’s Made” about water bottles, socket sets, and leather shoes). Next, the participant’s shock-tolerance threshold was determined by administering increasingly intense shocks, at 100-mA intervals, until the participant reported that the shock was “beginning to hurt.” To enhance the credibility of the experimental situation, the participant overheard an audiotape ostensibly portraying the same procedure being conducted with the fictitious other participant. Instructions for the reaction time task were then provided via intercom

to the “two” competitors. During the MTAP, participants were asked to say out loud their current levels of anger, happiness, and fear on a 0-10 scale every trial. Immediately following the MTAP, subjects completed another 15-question VAS and the Post-TAP Questionnaire. At the end of the visit, participants were debriefed as to the true nature of the experiment and compensated with three research credits for their time.

Data Analysis

Of the 82 individuals who completed the study, three individuals reported the study was designed to study anger/aggression or reported feeling as though they were playing against a computer. These participants were excluded from all data analyses, consistent with previous research using the TAP (Giancola & Parrott, 2008) making a sample of 79 total participants available for the analyses below. For each time course variable, individuals who did not express any change from baseline levels throughout the task were excluded from analyses specific to that variable as no rise time or return time could be calculated (n = 1 to 35 depending on the variable). In this vein, 31 individuals rated their subjective anger at a level of 0 and 12 individuals selected a shock level (aggression) of 0 for every trial of the task. When these individuals were excluded from peak analyses, the mean subjective rating for peak anger (M = 4.70, SD = 2.63 vs. M = 2.88, SD = 3.08) became significantly higher (t (118) = 3.29, $p < .01$, $d = .61$) while peaks for all other variables remained unchanged (all p’s > .05). As such, mean statistics presented below represent the data including “non-responders” so as not to overestimate peak values. For more information about differences between responders and non-responders, please see the Additional Results section. Physiological recordings were

visually inspected and artifacts were removed utilizing the AcqKnowledge software. Participants for which 5% of physiological data or more contained artifacts such as noise from external sources, loose electrodes, or equipment malfunction (n = 4 for heart rate, HF HRV and n = 1 for GSR) were removed from the physiology analyses.

For all time course variables, peak level was defined by the first occurrence of the highest value of each outcome variable that occurred after the baseline period. MTAP 20-shocks were recoded as 11 to minimize their influence on aggression calculations (McCloskey & Berman, 2003). Physiological recordings were visually inspected and artifacts were removed utilizing the AcqKnowledge software. Participants for which 5% of physiological data or more contained artifacts (n = 4) were removed from the physiology analyses. HRV indices were calculated using the Kubios-HRV software and were collected for each block of four trials to ensure enough time for indices to be valid. The duration of the rise and decline to and from peak levels was determined by evaluating change from baseline when receiving no provocation ("0" shocks). During block 1, participants acclimated to the task, and as such, block 2 was used as the baseline for subjective ratings, aggressive response, and physiological response (HR, GSR, HF HRV).

The rise from baseline for subjective anger, aggression, and HR was defined as the number of trials between the first statistically significantly different trial from baseline and the peak trial (provided there were no trials between these two trials that fell back below the baseline). Baseline was determined by the mean of trials 5, 6, 7, and 8. Significant departures from baseline were signified by exceeding the upper bound of the

95% confidence interval created around the mean of trials 5, 6, 7, and 8. The return to baseline was defined by the number of trials between the peak trial and the trial after the peak that returned to baseline for at least two consecutive trials. Again, return to baseline was signified by returning to a value below the upper bound of the 95% confidence interval created around the mean of trials 5, 6, 7, and 8. Due to the extended time necessary to compute GSR and HF HRV (computed over each 4 trial block), baseline for these variables corresponded to the value of block 2. The rise from baseline was defined as the number of blocks between the first block that exceeded the value of block 2 and the peak block (provided there were no blocks between these two that fell below the baseline block value). The return to baseline was defined by the number of blocks between the peak block and the block after the peak that fell below the value of block 2 for at least two consecutive blocks. Individuals without any change from baseline levels during the task were excluded from analyses for that individual measure as no time course could be computed.

All variables were examined for outliers and significant skew. While some outliers greater than three standard deviations from the mean were identified, 5% trimmed means were not significantly different from the non-trimmed means for any outcome variables. As such, outliers remained in the analyses to increase statistical validity. An analysis of skew statistics and a visual inspection of histograms indicated that all time course variables were normal to minimally skewed (skew statistics = .68 to 1.92). Raw data for subjective anger by block were significantly skewed (5 blocks out of 9 with skewness > 2) and were log transformed. Means and standard deviations for

subjective anger below represent the non-transformed data for interpretability. Trait variables were also examined for collinearity. Using a VIF of 2.50 as a cutoff value (Allison, 1999), it was determined that the variables were not multicollinear (all VIF's between 1.31 and 2.00).

Results

Preliminary Analyses

To assess the validity of the MTAP we examined the effect of provocation on subjective anger. There was an overall effect of block on subjective anger [F (8, 71) = 5.98. $p < .001$, $\eta^2 = .40$]. Planned comparisons examined if anger increased from baseline (block 2) to peak provocation (mean of blocks 4 and 5) and then decreased post provocation (mean of blocks 7-9). As expected, even when including participants who showed no change in anger, there was an overall increase in subjective anger from baseline (M = 0.31, SD = 0.84) to peak provocation [M = 1.70, SD = 2.14; t (78) = 6.28, $p < .001$, $d = .72$] and a similar decrease in subjective anger from peak provocation to post-provocation [M = 0.41, SD = 0.73; t (78) = 6.21, $p < .001$, $d = .82$] (see also Figure 1). Thus, the MTAP was effective at eliciting an anger response.

Means (SD) for all of the study variables (i.e., time course and trait variables) are presented in Table 1. The relationship between time course variables and demographic variables (i.e. age, race, gender) were also calculated. Age was not correlated with any time course variable (r's between -.20 and .13, p's between .14 and .91), nor did any of the time course measures vary as a function of race (all F's between .10 and 2.49, all p's between .08 and .90). However, women reported slower returns to baseline levels for

both subjective anger (t (42) = 3.01, p < .01, d = .93) and aggression (t (58) = 2.43, p = .02, d = .64). Due to this result, gender was initially added as a covariate to regressions predicting time course variables from trait variables. As adding gender did not change the overall pattern of results, it was removed from the final analyses.

Correlational analyses revealed few significant relations among the time course variables (see Table 2). Peak GSR was positively associated with rise time of subjective anger, peak HR, and return time of GSR. Peak HR was positively correlated with rise time of aggression. Peak aggression was negatively correlated with return time of HR. Finally, the rise time of HF HRV was positively correlated with the return time of HF HRV. Overall, the results suggest that the different components of anger are relatively independent of each other, at least with regard to time course and intensity. In contrast, all trait variables were positively correlated with each other except for the relation between trait aggression and emotion regulation deficits (for full results, see Table 3).

Primary Analyses

The time course of each component was defined by the rise time to peak levels and the return to baseline levels. For means and standard deviations for each component of the time course, please see Table 1. Graphs of the time course for each component are presented in Figures 1-5. For each component, the rise time was compared to the return time. Paired t-tests revealed that the rise time was significantly faster than the return to baseline for subjective anger (t (45) = -3.06, p <.001, d = .47), aggression (t (60) = -4.22, p <.001, d = .59), GSR (t (75) = -11.89, p <.001, d = 1.37), and HF HRV (t (56) = -5.12,

p <.001, d = .66). The rise time for measurements of HR was significantly faster than the return time only at a trend level (t (73) = -1.77, p =.08).

A series of correlations were performed to determine the relationship between trait variables and the different aspects of anger's subjective, behavioral, and physiological time course (rise time to peak, return to baseline, and peak) (see Table 4 for full results). Trait anger was significantly associated with an increase in the length of the rise time of GSR. Trait aggression was significantly associated with an increase in peaks of subjective anger and a decrease in the return time of aggression. Lastly, trait hostility was associated with an increase in the peak of subjective anger, an increase in the peak of GSR, and a decrease in the return time of aggression. Deficits in emotion regulation were not associated with any time course variable. Overall, the results suggest a weak to non-existent relationship between trait measures of anger / emotion regulation and anger time course.

Discussion

The current study set out to describe the time course of the different components of anger utilizing a standardized provocation as well as examining the potential effects of putatively related trait variables on anger's time course. For most of the anger component indices, the rise time to peak levels was significantly faster than the return time to baseline. This indicates that individuals typically become angry and aggressive quickly, but take longer to calm themselves down. This extends past research investigating temper tantrums in children as well as anger in clinical populations (Jacob et al., 2008; Potegal, 2010). We also found that peak subjective anger reached about 30% of the maximum

threshold (50% when not including non-responders), which is generally consistent with previous findings on the topic utilizing retrospective self-reports (Beck & Fernandez, 1998; Fernandez & Beck, 2001; Stets & Tsushima, 2001). In contrast, average peak aggressive responding over the task (of individuals for whom the task exceeded their aggression threshold) was approximately 70% of maximum threshold, suggesting that at least within the context of this task, aggressive responding is disproportionate to reported anger. Investigations of the physiological components of an anger episode showed that the time course of general autonomic response as measured by heart rate showed no difference between the rise time to peak levels and the return to baseline from peak levels. However, when each component of the autonomic response was investigated separately, we found that the rise times to peak levels of both sympathetic response and parasympathetic response (as measured by GSR and HF HRV, respectively) were faster than the returns to baseline levels. Lastly, it was found that overall, trait variables of anger and associated constructs were minimally associated with time course variables.

In categorizing the time course of the subjective component of anger, we found a pattern consistent with the majority of research suggesting that in a typical anger episode, the rise time to peak levels of subjective anger occurs more quickly than the return from peak to baseline levels (Potegal, 2010; Potegal & Qiu, 2010; Qiu, Yang, & Potegal, 2009). As each trial lasted approximately 25 seconds, the average rise time of subjective anger was approximately 2 minutes and 14 seconds whereas the average return time was approximately 3 minutes and 33 seconds. This is similar to self-report studies suggesting that the average duration of an anger episode lasts “minutes” (Frijda, Mesquita,

Sonnemans, & Van Goozen, 1991). Rise time during the study was somewhat longer than previous self-report research (Beck & Fernandez, 1998; Fernandez & Beck, 2001); however, the average return time was in line with previous findings (Ekman, 1994; Potegal, 2010). Of course, average rise and return times are influenced by the duration and level of provocation (Potegal, 2010). As such, the rise time to peak anger appeared to follow the provocation as its escalation was consistent with previous research (Pruitt, Parker, & Mikolic, 1997). Because the actual duration of the time course variables is so dependent on the length of the provocation, the roughly 1 to 1.5 ratio between the rise time and return time found in the current study could be a more useful metric in comparing results across provocations in both experimental and naturalistic settings.

Our finding that the rise time to peak aggression occurs faster than the return to baseline levels confirms our suspicions that the temporal dynamics of aggression follow a similar pattern to that of subjective anger. However, the fact that these measures were not correlated with one another suggests that anger and aggression are distinct components of the anger episode. In support of this idea, we found that peak levels of aggression were significantly higher than peak levels of subjective anger. Although one could argue that it is unclear that the scales used to measure anger and aggression are equivalent and comparable, it remains interesting that participants shocked their opponent at 70% of the maximum level on average while only endorsing 30% anger. Furthermore, of the 31 individuals who did not report any anger, only three of those individuals did not set any shocks above "0." One argument for this finding is that participants did not believe they were behaving aggressively but simply participating in the sanctioned "game" of the

MTAP and responding to competition (Tedeschi & Quigley, 1996). However, studies investigating the external validity of the TAP show that responding on the TAP does not correlate with measures of helping or competition (Bernstein, Richardson, & Hammock, 1987) nor does it seem influenced by conformity to demand characteristics (Berkowitz & Donnerstein, 1982; Giancola & Chermack, 1998). A more plausible explanation is offered by models of aggression escalation that suggest reactive aggression increases in a step-wise fashion with individuals matching levels of aggression as the provocation unfolds (Gouldner, 1960; Kane et al., 1973). As such, escalation of aggression could be anchored to the provocation and not necessarily to levels of anger (Blair, 2008; Glenn & Raine, 2009; Felson, 1982). Additionally, aggression is motivated by a number of variables aside from anger (Giancola, 1995) including fear, social goals, and conflict resolution strategies (Tedeschi & Felson, 1994) all of which are likely at play during TAP (and MTAP) responding (Giancola & Chermack, 1998).

Individuals tended to experience physiological arousal during the task consistent with our predictions with the rise time of sympathetic (GSR) and parasympathetic (HF HRV) measures being quicker than the return time. Overall autonomic response, as measured by heart rate, did show a characteristic rise and fall, but the rise time was only significantly different from the return time at a trend level. One reason for this finding may be that heart rate is a measure of both sympathetic and parasympathetic responding which are complimentary systems with one rising as the other falls (Scarpa & Raine, 1997). As such, it is possible that our measure of heart rate captures a “cancelling out” effect triggered by the activation and deactivation of these systems at different times

during the provocation. When examining the time course by block (see Additional Results chapter for further information), GSR began rising during the ascending block (block 3) while the first difference in HF HRV did not occur until after the provocation began decreasing (block 6). Taken together, we have a picture of the sympathetic system rising quickly to deal with the onset of provocation only to subside when the parasympathetic system begins to dominate after the provocation ceases as found in other studies (see Stemmler, 2010 for overview). Thus our results seem to suggest that parasympathetic responding occurs at the end of a provocation. Similar results have been noted in anxiety research showing lower HF HRV power during experimentally induced worry (Thayer, Freidman, & Borkovec, 1996); however, to our knowledge, this is the first study to measure autonomic responding over the course of an extended provocation (as opposed to after being exposed to a negatively valenced stimulus (e.g., Vaschillo et al., 2008)). If parasympathetic responding typically does not occur until after the "danger" has subsided, treatments focusing on utilizing coping skills and de-escalation strategies during a provoking event (e.g., SAMHSA Anger Management: Riley & Shopshire, 2002) might be fighting an uphill battle against our physiology and could perhaps be reconsidered to accommodate this information.

Similar to findings for aggression, physiological responding did not correlate with subjective anger. Additionally, a number of individuals who reported anger levels of '0' throughout the task still exhibited the characteristic rise and fall of physiological response. While it is plausible that physiological changes were due to the effects of general competition (Veldhuijzen Van Zanten et al., 2002; Harrison et al., 2001), we

would expect them to remain somewhat constant throughout the task instead of rising and falling around the provocation. Another possible explanation is that concordance rates tend to be low between physiological responses and subjective ratings suggesting that people are typically bad at reading their own physiology (Stemmler, 1992; Weinstein, Averill, Opton, & Lazarus, 1968). It is also possible that a number of individuals interpreted the provocation as more of a fearful than angering event, which did occur in our study (see Additional Results chapter for further information on fear responding during the task). Because the physiology of fear and anger (both being two sides of the same "fight or flight response") are very similar (Stemmler, 2010; Cacioppo, Berntson, Larson, Poehlmann, & Ito, 2000), we would expect the same physiological response for fearful and angry individuals. Though this initial pilot study was underpowered to compare individuals with predominately a fear vs. anger response, this is an area for future research.

While many of our hypotheses were supported, we did not find that increased levels of trait anger, aggression, hostility, and emotion dysregulation were particularly associated with anger time course. This was surprising in light of previous research (Beckham et al., 2002; Jacob et al., 2008; Potegal, 2010). Of the positive results, we did find that higher trait anger was significantly associated with slower rise times of GSR, higher trait aggression was associated with higher peaks of subjective anger and aggression as well as faster return times of aggression, and higher trait hostility was associated with an increase in the peak of subjective anger, an increase in the peak of GSR, and faster return times of aggression. The pattern of these positive results suggests

that if trait variables have any effect on elements of the time course, it is likely to increase peaks as opposed to altering the rise and return times. This notion is supported by some research suggesting that individuals who exhibit pathological levels of anger and aggression report higher intensity of anger (fury and rage), but do not necessarily report that they get angry faster or stay angry longer (Kulper, Kleiman, McCloskey, Berman, & Coccaro, 2014). However, considering the large number of comparisons performed, these findings may not generalize to other samples. Additionally, it is possible that the study's utilization of an undergraduate population resulted in relatively lower trait scores, which had less of an effect on the experience of anger during this task. The majority of studies on the effect of these particular trait measures on real world anger and aggression have included clinical or offender samples with higher trait means (e.g. Beckham et al., 2002; Diamond & Magaletta, 2006; Jacob et al., 2008; McCloskey et al., 2006). As such, future research would do well to directly compare clinically angry and more normative samples.

The results of the current study can serve to inform both treatment and future research. Patients with anger problems often report being unable to utilize anger coping skills because their anger is experienced as too instantaneous and out of control (Kulper et al., 2014). This is problematic as many current treatments for anger will often ask clients to employ coping skills when they recognize lower levels of anger that are starting to escalate (e.g. Riley & Shopshire, 2002; Linehan, 1993). The current study suggests that individuals often do not realize when they are physiologically showing signs of anger and can often behave aggressively without subjectively labeling themselves as "angry". Therapies can take a number of approaches to incorporate this information into their

protocols. For one, coping skills that affect pre-provocation states and cognitive appraisals of provocation events could serve to decrease feelings of anger and aggressive behavior. In addition to the findings in this study, other research has found that individuals with pathological affective aggression who show decreased HF HRV at baseline behave more aggressively during the TAP (Puhalla et al., 2015). As such, techniques designed to increase baseline parasympathetic activity (or alternatively decrease sympathetic functioning) such as progressive muscle relaxation or imaginal relaxation (Bernstein, Carlson, & Schmidt, 2007; Kerr, 2000) could serve to decrease anger and aggressive behavior. This sentiment is backed by research investigating a CBT-based therapy for anger and aggression–Cognitive Restructuring, Relaxation, and Coping Skills Training (CRCST; Deffenbacher & McKay, 2000). Studies have found that this protocol, utilizing relaxation techniques and attempting to change pre-provocation cognitive appraisals, is effective for treating individuals with problematic aggression (McCloskey, Noblett, Deffenbacher, Gollan & Coccaro, 2008).

This study has several strengths including the development of a standardized provocation with clear pre-provocation, provocation, and post provocation periods as well as a multidimensional assessment of anger. There were also aspects of the study that limit the conclusions that can be drawn from our findings including the use of an undergraduate population, lack of assessment of cognitive appraisals during the provocation, and a large number of individuals whose time course measures did not move from baseline levels. As such, future studies utilizing a clinical sample of individuals with problematic anger and aggression (and presumably higher levels of anger related

traits) would help assess if the current results are generalizable and would better inform treatment efforts. Clinical samples are also likely to have lower thresholds for anger and aggression (Novaco, 2010) which should result in fewer non-responders to the provocation task.

Furthermore, future research should seek to determine the role of the cognitive component of anger in regulating emotional response during a provocation. It seems likely that a number of non-responders were using cognitive strategies to refrain from getting angry or aggressive. When speaking to these individuals after the task, their opponent was often categorized as non-threatening or dismissed as "crazy." The cognitive component was left out of the current study due to the difficulties of measuring the construct in real time while avoiding demand characteristics. Future research should seek to find a creative solution to measuring appraisals accurately and in real time to better understand the role of cognition in regulating emotional response. Despite these limitations, the study succeeds in validating previous findings that the rise time to peak levels of the components of anger is faster than the return to baseline levels using a standardized and ecologically valid laboratory provocation. Additionally, the lack of relationships between the time course variables provides evidence for the idea that different components of anger have independent time courses. Furthermore, by developing a clearer picture of the temporal dynamics of anger, we may be able to develop more effective treatments for problematic anger that take into account the time-sensitive nature of the anger experience.

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Table 1.

Means (SD) of study variables

Variable (n)	Mean (SD)
Subjective Anger	
Rise Time (44)	5.39 (4.29)
Return Time (44)	8.52 (5.15)
Peak (76)	2.88 (3.08)
Aggression	
Rise Time (60)	4.56 (3.74)
Return Time (60)	7.85 (5.52)
Peak (79)	7.44 (3.90)
Heart Rate	
Rise Time (71)	4.85 (5.30)
Return Time (71)	6.59 (6.41)
Peak (76)	87.26 (13.50)
Galvanic Skin Response	
Rise Time (73)	0.64 (0.81)
Return Time (73)	2.97 (1.58)
Peak (73)	6.33 (3.07)
High Frequency Heart Rate Variability	
Rise Time (55)	0.95 (1.44)
Return Time (55)	2.00 (1.17)
Peak (75)	2177.19 (2411.46)
Trait Measure Scores	
BPAQ Anger (79)	12.92 (4.86)
BPAQ Aggression (79)	29.00 (9.47)
BPAQ Hostility (79)	16.68 (5.91)
DERS Total (79)	71.03 (18.12)

Note. BPAQ = Buss Perry Aggression Questionnaire, DERS = Deficits in Emotion Regulation Scale; units for rise time and return time variables for subjective, aggression, and heart rate are number of trials; units for rise time and return time for GSR and HF HRV are number of blocks; units for peak heart rate are beats/minute; units for peak GSR are number of galvanic skin responses; units for peak HF HRV are meters/second^2

Table 2.

Correlations (r) among time course variables

	Subjective Anger			Aggression			Heart Rate			GSR			HF HRV	
	Rise	*Return*	*Peak*	*Rise*	*Return*	*Peak*	*Rise*	*Return*	*Peak*	*Rise*	*Return*	*Peak*	*Rise*	*Return*
Anger														
Return	-0.09													
Peak	0.29	0.25												
Aggression														
Rise	0.14	0.15	0.04											
Return	0.14	0.12	-0.21	0.19										
Peak	0.12	0.17	0.09	0.17	0.16									
Heart Rate														
Rise	0.07	0.19	-0.03	0.02	0.19	-0.03								
Return	-0.17	-0.17	-0.01	-0.19	-0.20	-0.37**	-0.03							
Peak	0.15	-0.05	-0.07	0.32*	0.12	-0.22	0.03	0.06						
GSR														
Rise	-0.06	-0.17	-0.16	0.01	-0.08	0.01	-0.21	-0.22	-0.14					
Return	0.27	0.18	0.2	0.04	-0.24	-0.12	0.01	0.04	-0.01	0.09				
Peak	0.38*	0.00	0.08	0.2	-0.18	0.15	-0.18	-0.04	0.39**	0.05	0.32**			
HF HRV														
Rise	0.04	0.00	-0.01	0.23	0.41**	-0.11	-0.07	-0.14	0.05	0.13	-0.25	-0.07		
Return	0.2	0.14	0.1	0.02	0.04	-0.2	0.00	-0.12	-0.11	0.02	-0.08	-0.13	0.29*	
Peak	0.04	0.22	0.05	0.02	0.07	-0.02	-0.03	0.04	-0.17	-0.05	0.13	-0.01	0.00	0.11

Note. GSR = galvanic skin response, HF HRV= high-frequency heart rate variability;
* = $p < .05$, ** = $p < .01$

Table 3.

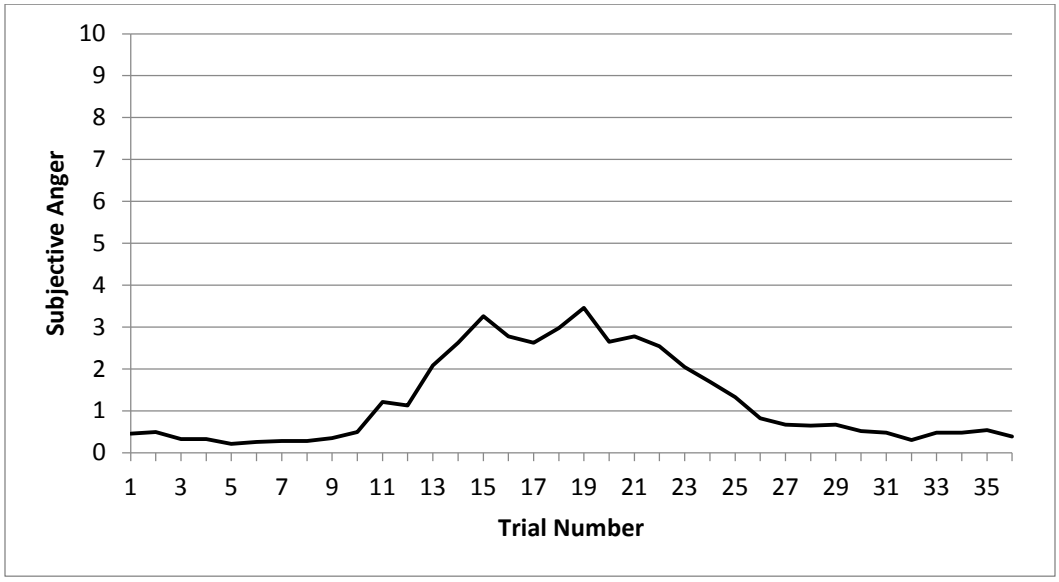
Correlations (r) among trait variables (N = 79)

	BPAQ Anger	BPAQ Aggression	BPAQ Hostility
BPAQ Aggression	0.61***		
BPAQ Hostility	0.54***	0.44***	
DERS Total	0.35**	0.01	0.46***

Note. BPAQ = Buss Perry Aggression Questionnaire, DERS = Deficits in Emotion Regulation; ** = $p < .01$, *** = $p < .001$

Figure 1.

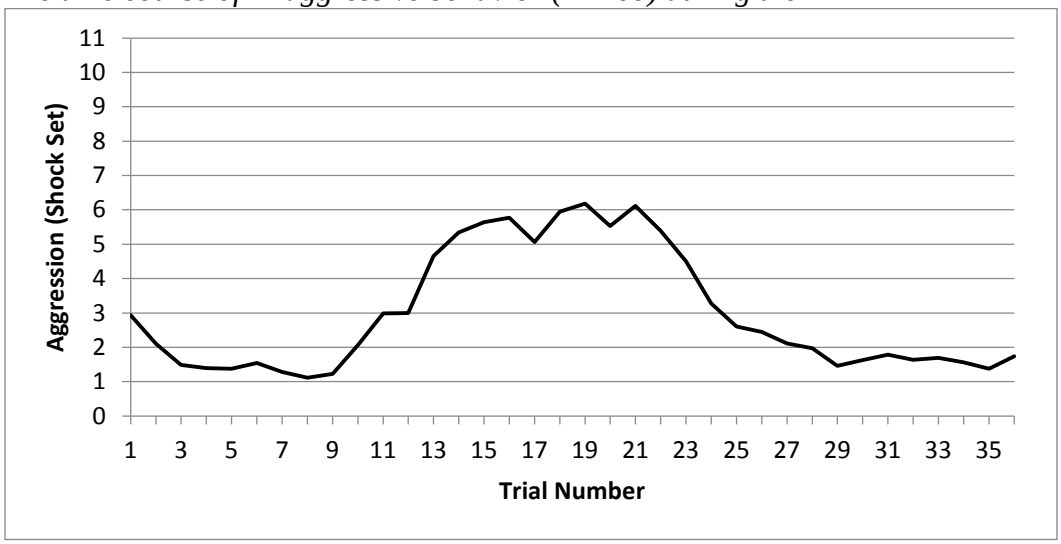
The time course of M subjective anger rating (n = 44) during the MTAP



Note. MTAP = Modified Taylor Aggression Paradigm

Figure 2.

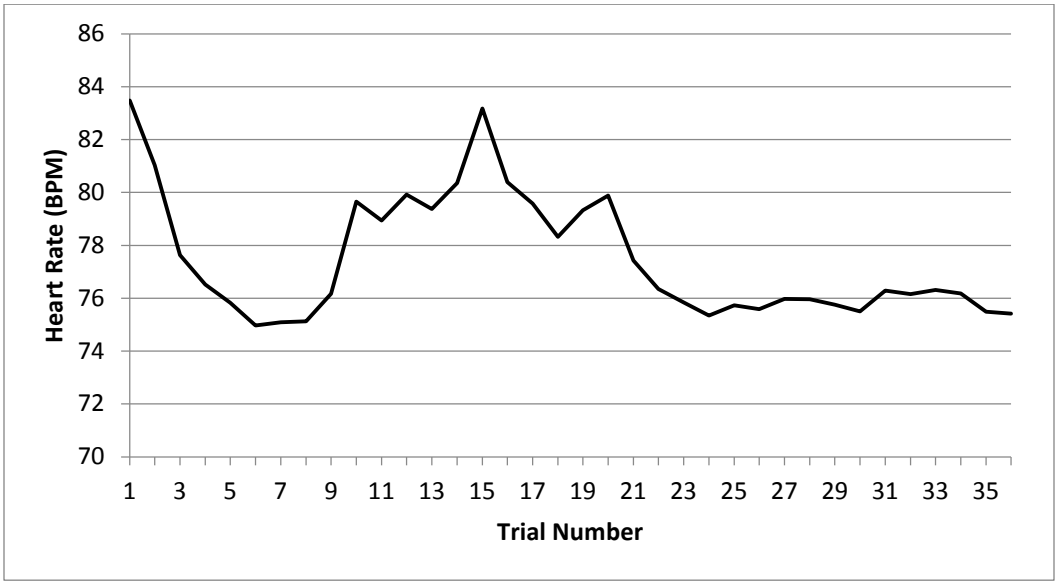
The time course of M aggressive behavior (n = 60) during the MTAP



Note. MTAP = Modified Taylor Aggression Paradigm

Figure 3.

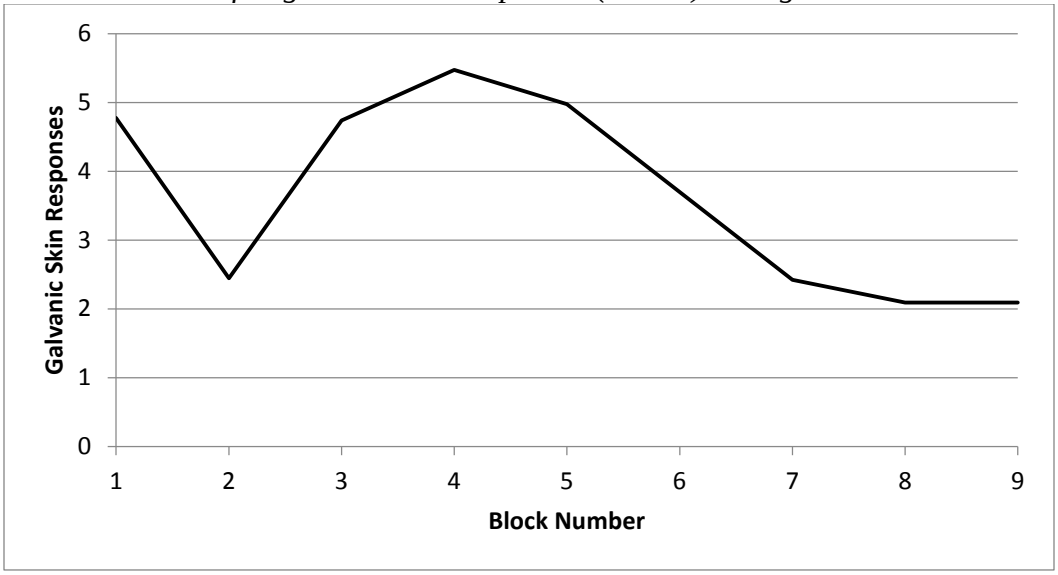
The time course of M heart rate (n = 71) during the MTAP



Note. MTAP = Modified Taylor Aggression Paradigm

Figure 4.

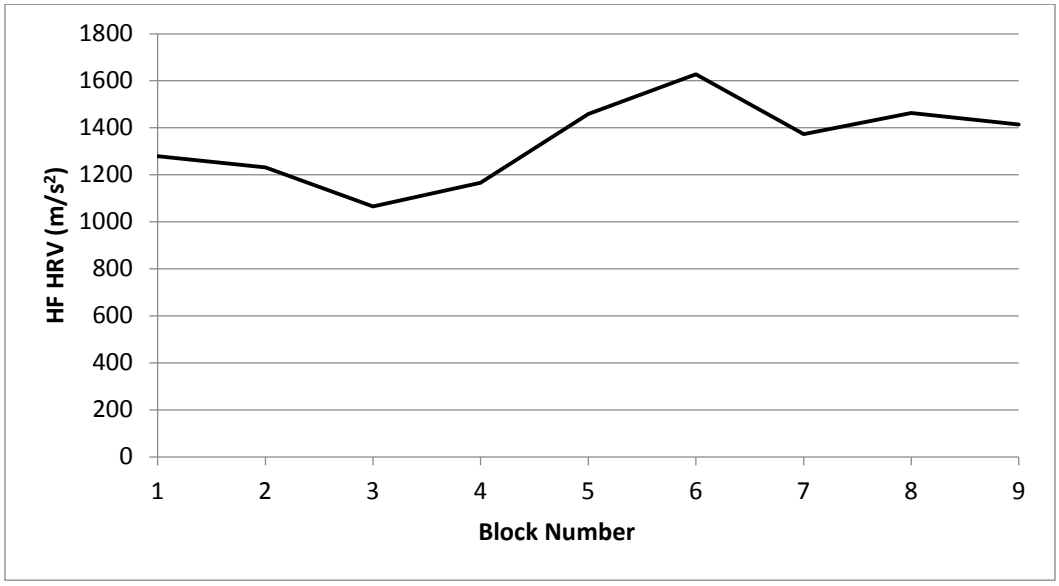
The time course of M galvanic skin responses (n = 73) during the MTAP



Note. MTAP = Modified Taylor Aggression Paradigm

Figure 5.

The time course of M HF HRV (n = 55) during the MTAP



Note. HF HRV = high frequency heart rate variability, MTAP = Modified Taylor Aggression Paradigm

Table 4.

Correlations (r) between time course variables and trait variables

	BPAQ Anger	BPAQ Aggression	BPAQ Hostility	DERS Total
Subjective Anger				
Rise Time (n=44)	0.06	0.23	-0.02	0.10
Return Time (n=44)	-0.09	-0.22	-0.06	0.12
Peak (n=76)	0.20	0.26*	0.24*	0.18
Aggression				
Rise Time (n=60)	0.03	-0.03	-0.07	-0.03
Return Time (n=60)	-0.10	-0.36**	-0.28*	-0.16
Peak (n=79)	0.16	0.28*	0.10	-0.07
Heart Rate				
Rise Time (n=71)	-0.08	-0.21	0.11	0.03
Return Time (n=71)	-0.16	-0.23	-0.12	0.00
Peak (n=76)	-0.20	-0.15	-0.04	-0.10
Galvanic Skin Response				
Rise Time (n=73)	0.25*	0.17	0.04	-0.01
Return Time (n=73)	-0.02	0.08	0.06	0.11
Peak (n=73)	0.06	0.20	0.27*	0.04
High Frequency Heart Rate Variability				
Rise Time (n=55)	-0.21	-0.16	-0.20	-0.14
Return Time (n=55)	-0.16	0.01	-0.13	0.10
Peak (n=75)	-0.08	-0.11	-0.17	0.09

Note. BPAQ = Buss Perry Aggression Questionnaire, DERS = Deficits in Emotion Regulation Scale; * $p < .05$, ** $p < .01$,

CHAPTER 2

LITERATURE REVIEW

The Time Course of Anger: A Review of Theory and Research

Anger and Its Impacts

Anger has been identified as a basic human emotion and near-universal experience in both philosophical conceptualizations of emotion as well as empirical studies of the subject for thousands of years. Early philosophers differed in their opinions of the utility of anger with philosophers such as Seneca arguing that anger was a useless emotion that was "worthless even for war," (Kemp & Strongman, 1995). Aristotle, on the other hand, thought anger was a useful tool for interacting with others and seeking justice, as long as the emotion was practiced in moderation (Aristotle, trans. 1999, 1125b). However, all early philosophers saw the need for the control of angry emotions as, in excess, anger often leads to a number of negative consequences. In the modern era, anger has been consistently linked to a number of negative outcomes such as negatively impacted relationships with friends and family (Tescher, Conger, Edmondson, & Conger, 1999), occupational stress and burnout (Sloan, 2004), as well as a number of physical and psychological health consequences. Chronic anger has been associated with increased levels of depression and anxiety (Tescher et al., 1999) as well as negative physical health outcomes such as high blood pressure and coronary heart disease (Diamond, 1982; Goldstein & Niaura, 1992; Matthews & Haynes, 1986; Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003; Sirois and Burg, 2003).

Despite anger's ubiquity and its significant public health impact, there are still many aspects of this basic emotion that have been understudied and are thus poorly defined. Specifically, the time sensitive mechanisms at play in an individual's progression from a baseline state to elevated levels of anger and aggression and back to a baseline state are not well understood. While the scientific community have developed some quite extensive conceptualizations of anger and its correlates, understanding the temporal components of anger is essential for placing our knowledge in a time-sensitive context and understanding the totality of an angry experience.

This paper reviews the theories and extant research on the time course of anger. First, the review provides background theories of emotion and anger to provide a context for understanding the different processes involved in the time course. Second, the study of the emotional time course is introduced and a review of the relevant literature with respect to anger is evaluated. Gaps in the current literature, methodological issues, and future directions are explored.

Theories of Emotion

In the broadest sense, anger theories are inexorably linked to basic theories of emotion, and most of what we understand about the time course of anger extends from our knowledge of the necessary components of an emotional response. The study of emotion can trace its roots back all the way to Ancient Greece when Hippocrates developed his theory of the four bodily humors which corresponded to affective temperaments and Ancient China where theorists felt that certain emotions caused damage to internal organs (Suchy, 2011). However, modern scientific research into the

dynamics of emotional response is a relatively recent development. The earliest scientific theories of emotional response include the James-Lange theory (James, 1894), the Cannon-Bard theory (Cannon, 1927), and the work of Schachter and Singer (1962) (See Figure 1). The three theories represent the beginnings of the basic framework for understanding emotional reactivity wherein stimuli from the environment cause changes in the body which result in emotional response, the basis for developing a time course of emotional response. For James and Lange, emotion is the result of the interpretation of physiological changes which come about from environmental cues (Ellsworth, 1994). In this theory, the physiological changes that occur following an environmental cue are the emotion. These physiological changes take place in what was called "the viscera" but is now known as the autonomic nervous system. The popularization of this theory led to important changes in the way emotion was investigated. For one, the James-Lange theory was the first to popularize the idea that human responding is very much reactive and dependent on environmental cues, providing a mechanistic account of how emotions come to be (Cannon, 1927; James, 1894) in which the body essentially functions as an input-output machine, processing environmental cues and producing an emotional response. Some theorists see this as an early reductionist theory of emotion suggesting that emotions are not "mental" events, but are equal to physical states (e.g. Zajonc, 1984). This eventually led theorists and researchers to look for unique physical states that corresponded to the different emotions observed referred to as physiological specificity (Davidson, Jackson, & Kalin, 2000). Secondly, the theory posited the idea that there was no dedicated brain center for experiencing emotions, an idea that was questioned by other

theorists (Canon, 1927; 1929) and led to discoveries about the neural mechanisms of emotional responding (Davidson, Jackson, et al., 2000).

Among other criticisms, Cannon (1927) challenged the James-Lange theory's idea that emotional responding occurred solely in what James referred to as "the viscera," the internal organs and sensory/motor systems that comprise the physiological response following an emotion provoking stimulus. According to his theory, now dubbed the Cannon-Bard theory, an emotional response occurs in conjunction with physiological arousal, but the two are distinct entities. This theory is based in animal research that suggests that emotional responding remains unaltered when the "visceral system" is removed (Bard, 1928; 1929). Additional criticisms to the James-Lange theory of the time included the fact that similar physiological responses can result in different emotional states, that physiological changes are often too slow to account for emotional responding, and that artificial induction of the physiological changes typical in strong emotions do not always reproduce these emotions (Cannon, 1927). As such, the physiological response occurred separately from the experience of an emotion and is not a necessary component of the emotional response.

Cannon drew on previous studies related to emotional expression (e.g. Bechterev, 1887) to posit the idea that certain subcortical structures were more responsible for emotional response than the visceral system. His most influential claims stemmed from research on decorticized animals (e.g. Woodworth & Sherrington, 1904; Cannon & Britton, 1925) wherein emotional responses could be induced by stimulating areas of the thalamus in the absence of the cortex. As such, Cannon and Bard hypothesized that

certain neuronal structures such as the thalamus and hypothalamus are likely responsible for aspects of the expression and experience of emotion. This idea was instrumental in the investigation of different brain regions that, in conjunction with physiological changes in the autonomic nervous system, may be implicated in emotional responding. Further research into the neurobiological bases of emotion has revealed a great number of sub-cortical structures. In response to Cannon-Bard's thalamic theory of emotion, Papez (1937) and later MacLean (1949) investigated the diencephallic structures and posited their own theory of the neurobiological bases of emotion. Papez (1937) described a neural circuit that involved the hypothalamus, anterior thalamic nucleus, hippocampus, and cingulate cortex (Davidson, Jackson, et al., 2000). Other structures were later added to the circuit such as the amygdala and led to the development of the first conceptualization of the limbic system, presumed to be prominently involved in emotional expression and experience (MacLean, 1952).

Insights in neurobiology have created a resurgence in James's purely somatic theories of emotion, suggesting that the emotional response is a function of both autonomic responding (the physiological changes James cited) and physiological changes to include the brain. By extending the realm of physiological change into the brain, modern somatic theorists such as Antonio Damasio (2008) and Robert Zajonc (1997) are able to avoid many of Cannon's initial criticisms and support the idea that environmental cues cause physiological changes in an individual's system (including the brain) and that this results in an emotional response. As such, the physiological changes that comprise the first step in the James-Lange theory still cause an emotional experience and thus are

not a separate entity from the emotion. While this notion has been further supported by research showing physiological specificity for some negative emotions such as fear and anger (Ax, 1953; Schachter, 1957), many researchers have suggested that adding a cognitive factor to the physiological circuits better explains the differentiation between emotions, especially ones with similar physiological signatures (Barrett, 2006; Fehr & Stern, 1970; Lazarus, 1984).

Schachter and Singer (1962) argued that a physiological response was necessary, but not sufficient for the experience of emotion. Their two-factor theory argued that emotion is the result of an interpretation of that physiological response, an interpretation that typically takes into account acute changes in one's environment. In a famous study, Schachter and Singer (1962) investigated the effects of environmental cues on emotional responding following an epinephrine injection. They found that participant's emotional responses depended on environmental cues (the emotions of confederates in the study). This two factor theory suggests that physiological responses are interpreted based on environmental cues, resulting in the emotion. This cognitive theory of emotion has been further substantiated by research into misattribution of arousal wherein individuals placed in a physiologically arousing state (the classic study has them walk over a dangerous looking bridge) are more likely than those in a non-aroused state to judge things around them as more arousing (in the classic study, an attractive experimenter) (Dutton & Aron, 1974).

Building upon Schachter and Singer's initial conceptualization, Richard Lazarus (1982), proposed that cognitive processing played an integral role in determining which

emotion was experienced at a physiological level. In Lazarus's theory, the environmental cue is first understood cognitively, appraised, and labeled (Lazarus, 1984). This appraisal triggers physiological changes in the body which in turn causes the experience of an emotion. An important final step in Lazarus's theory is the action stage wherein the individual feels the emotion and decides how to react. This is distinct from the Schachter and Singer model where the physiological response precedes the cognitive appraisal, however both theories claim that an appraisal is a necessary component. Lazarus's theory has been somewhat supported by neurobiological studies investigating the pathways of cognitive activity in the brain that state that the amygdala (proposed to do much of the emotional processing in the brain) receives input from both the thalamus (integrating sensory information) and the cortex (appraisals and judgments of the situation) (LeDoux, 1989). However, neurobiological evidence also suggests that the pathway from the cortex reaches the amygdala after the amygdala processes thalamic information (LeDoux, 1992). Additionally, the pathway between the amygdala and the cortex travels in both directions suggesting that emotional processing has an impact on cognition as well (LeDoux, 1995). These findings have had a significant impact on the debate about the relative importance of cognitive and physiological processes in determining an emotion.

While the debates of the past focused on whether emotions could occur without cognitive or physiological input and which type of input was primary in the causal chain (e.g. Lazarus, 1984), current theories assert that this distinction creates a false dichotomy given that as the brain structures previously labeled "cognitive" and "affective" have dynamic and reciprocal relationships, making the two systems integrative as opposed to

separate (Pessoa, 2008). Evidence shows that while the amygdala is linked to affective responding, it also plays a significant role in cognitive processes such as attention and associative learning (Holland & Gallagher, 1999). Similarly, areas of the prefrontal cortex such as the orbito-frontal cortex and anterior cingulate cortex have been linked specifically to emotional responding (Pessoa, 2008). As such, it appears that emotional responding occurs on multiple levels through a number of different pathways. Additionally, the equation between physiological/affective components and cognitive components changes throughout the course of the emotional event (Davidson, 1999). In light of these findings integrating the components of emotional responding, theories have shifted toward a systems-oriented approach in an attempt to determine the relations between these elements. Emotion is not a single, static event. The expression and experience of emotion change in response to both internal and external cues in a dynamic way (Ellsworth & Scherer, 2003). Additionally, these dynamics likely differ between different emotions. One of the reasons researchers have largely failed to find physiological specificity for a number of emotions is that the extant literature has largely focused on emotion in a static way, analyzing the consequents of an initial environmental cue, but focusing less on how these consequences might change temporally (Sander, Grandjean, & Scherer, 2005). While basic theories of emotion have done well to describe the general mechanisms of emotional responding in humans, it is clear that these mechanisms are different and distinct in key ways in order to produce the wide range of individual emotions that humans experience.

Anger Conceptualizations

The understanding gained by early emotion researchers has allowed us to become more nuanced in our investigations and to study individual emotion processes. The study of anger in its earliest forms occurred in parallel with the study of basic emotions, and our contemporary understanding of anger continues to borrow from this tradition. In particular, current conceptualizations of anger typically break the anger experience down into different components (Deffenbacher, 2011; Novaco, 1977; Potegal & Stemmler, 2010). This breakdown of components has been supported by factor analyses in the development of common anger assessment tools (Buss & Perry, 1992; Spielberger, 1999). While occasionally employing different names, contemporary researchers focus on four main components of the angering experience: physiological, cognitive, behavioral, and subjective/experiential (Novaco, 1977; Potegal & Stemmler, 2010). The physiological component refers to the physiological changes that take place in the body as a result of an anger-provoking stimulus. Within this component, there is sometimes a distinction between physical sensations (responses of the autonomic nervous system) and brain activity (Potegal & Stemmler, 2010), although these two aspects are both understood within a physiological context and will be talked about in conjunction throughout this paper. The cognitive component is comprised of the "appraisals, attributions, expectations, and self-statements that occur in the context of provocation," (Novaco, 1977). The behavioral component is conceptualized as the "action urge" which accompanies angry emotions. While this urge can sometimes be to withdraw from a situation or ruminate on the emotion (Spielberger, 1999), many theorists claim that the

primary action urge tied to anger is aggression (e.g. Berkowitz, 1989). In this vein, aggression is studied both as a component of the anger experience as well as a consequence/corollary of anger and thus a separate phenomenon. This distinction is discussed further below. Lastly, the subjective component refers to an individual's qualitative experience of anger.

Criticism of this componential conceptualization comes primarily from those who subscribe to a somatic theory of emotion, proposing that emotions, including anger, have distinct physiological signatures that result in a conceptualization of anger similar to James's early emotion theory (ex. Damasio, 2008). Variations of this criticism include the view that physiological specification is enough to account for emotional responding as well as the notion that the componential view creates a false separation between the components. These theories are largely reductionist in nature, purporting that the molar concepts that we often talk about (cognition, behavior, emotion) can all be functionally reduced to molecular processes that occur within the body.

While these theorists often place a premium on the physiological component of anger, these reductionist theories can be compatible with the cognitive-behavioral conceptualizations with both sides agreeing that the different components influence the emotional experience in some way. Although it may be true that the components involved in an emotional response are essentially physical processes that occur within the body and brain, Lazarus (1982) argues that this conceptualization limits our understanding of these concepts to only what we know about the brain. While this was likely a greater concern thirty years ago, we are still continuing to learn about how the brain processes

emotional information. Lazarus suggests that molar concepts such as “cognition” give us a more versatile lexicon to talk informatively about the different processes involved in emotional responding, specifically those that have not been fully reduced to physiological terms (which could arguably be none of them). As such, the componential view of anger as laid out earlier allows us to talk significantly about different concepts without giving up the possibility of understanding these processes in a reductionist framework. At this point, the main disagreement between theorists lies more in the relative importance assigned to the components rather than which components are at play. Interestingly, this question has gone largely unanswered, mostly due to the difficulty of evaluating the different ways these components change and interact over time (Davidson, 1999). Taking these theories as a whole, the basic conceptualization of anger states that environmental cues function as an input, which is then processed within the internal context of the individual (comprised of the body and the brain), which in turn then leads to an angry emotion comprised of physiological, cognitive, behavioral, and subjective components.

Physiological Component

In regard to anger, researchers have sought to explicate the individual factors associated with the emotion (physiological, cognitive, behavioral, and subjective). Early research on anger was tied to the physiological research on general emotion, often extrapolated from emotional responding in animals (ex. Cannon, 1915; Hall, 1899). These early studies described the basic physiological responses associated with anger such as “vaso-motor disturbances, glandular secretions, swallowing, nausea, spitting, disturbances in respiration” (Hall, 1899), more concisely described as upregulation of the

autonomic nervous system associated with the “fight or flight response” (Cannon, 1929). The autonomic nervous system upregulates various aspects of the body (e.g. increases heart rate, pumps blood to the extremities) in order to either flee a threatening situation or fight back against it. Anger has specifically been linked to the activation of the sympathetic nervous system (Funkenstein, 1955), and is accompanied by distinctive patterns of activity in the central and peripheral nervous systems including activation of the hypothalamus, increased blood flow to the musculature, heightened blood pressure and pulse rate, pupillary dilation, and decreased flow of blood to the viscera (Funkenstein, King, & Drolette, 1954; Johansson & Oest, 1982; Schachter, 1957).

These physiological reactions produce sensations in the body that contribute to our experience of the emotion. Anger produces the subjective experiences indicative of a fight or flight response including racing heart, trembling, fear or panic, fury or rage, and feeling out of control (Böddeker & Stemmler, 2000). Anger is also associated with sensations of heat caused by increased blood pressure, increased sweating, and facial warming (Potegal, Stemmler, & Spielberger, 2010). Because this type of fight or flight response is indicative of fear and anxiety as well as anger, researchers sought to extend Cannon’s initial conceptualization of the response and establish more unique physiological signatures for the emotions experienced. As such, anger differentiates itself from fear in terms of greater increases in diastolic blood pressure, increased number of galvanic skin responses, increases in muscle tension, increased facial temperature, lower cardiac output, and lower respiration rate (Ax, 1953; Schwartz, Weinberger, & Singer, 1981; Stemmler, 2004). Additionally, differences in baseline autonomic response

(specifically lower baseline levels of autonomic arousal, but higher autonomic reactivity to stressors) have been predictive of increased angry emotions and aggressive behavior in children (Lorber, 2004) and adults (Suls & Wan, 1993).

In addition to the physiological responses in the peripheral nervous system, there is strong evidence implicating the central nervous system as integral in the physiological component of the anger response. The neurobiological theories of anger grew from the earlier research on the neurobiology of basic emotional responding. As mentioned earlier, subcortical structures such as the thalamus and hypothalamus were the first to be implicated in emotional responding (Cannon & Britten, 1925). This led to later conceptualizations of an emotion system in the brain, dubbed the limbic system (MacLean, 1952). Since these early conceptualizations, researchers have been able to discern pathways in the brain that differentially correspond to different emotions.

Research into the neural components of anger has implicated regions of the frontal and temporal lobes of the cerebral cortex, the anterior cingulate cortex, and subcortical structures such as the thalamus, hypothalamus, and amygdala in playing primary roles in the generation and perpetuation of anger (Adams, 2006; Davidson, Putnam, & Larson, 2000; Potegal & Stemmler, 2010). The study of the neural components of anger relies in part on dysfunction or damage in certain brain regions in order to elucidate some of the functions of particular structures. The implication of regions of the temporal lobe is due in part to studies of temporal lobe dysfunction and temporal lobe epilepsy. Researchers have found positive relationships between temporal lobe abnormalities and violent/rageful behavior during psychosis (Tonkonogy, 1991),

aggression in TBI patients (Greve et al., 2001), and aggression and violence in a non-clinical sample (see Bufkin & Luttrell, 2005 for a review). Similarly, temporal lobe epilepsy has been associated with temporary increases in irritability as well as incidents of "episodic dyscontrol" characterized by rage and verbally aggressive behavior (Blumer, 2000). Furthermore, temporal lobectomies in patients with temporal lobe epilepsy not only result in fewer seizures, but also reduce the frequency of rage episodes (Fenwick, 1989). In a non-clinical sample, increased cerebral activation in temporal lobe EEG was correlated with increasing subjective intensity of angry memories. Additionally, in a study of children's tantrums, left temporal EEG activation was significantly associated with children's anger (Potegal, Goldsmith, Chapman, Senulis, & Davidson, 1999).

Also implicated in anger are subcortical (limbic) structures such as the amygdala and the hypothalamus. The amygdala has long been associated with angry and aggressive behavior, so much so that bilateral amygdalotomy (surgical removal of the medial amygdala) was a treatment (reportedly successful in 75% of patients) for intractable aggression (Mpakopoulou, Gatos, Brotis, Paterakis, & Fountas, 2008). Additionally, the amygdala has been linked specifically to aggressive behavior in clinical populations (Coccaro, McCloskey, Fitzgerald, & Phan, 2007). The connection between the amygdala and processing emotionally relevant stimuli is well documented and in particular, the amygdala seems integral in the processing of threat-related stimuli (for a review, see Davis & Whalen, 2001). The amygdala also has pathways that project into areas of the hypothalamus (Adams, 2006). Dysfunction of the hypothalamus (particularly in the form of tumors) has been associated with affective aggression (Tonkonogy & Geller, 1992).

Additionally, 75% of children with sizable hypothalamic hamartomas (masses of tissue that interfere with functioning) present with clinical levels of aggressivity, oppositionality, and rage (Weissenberger, Dell, & Liow, 2001). The association of the amygdala and hypothalamus with anger and aggression is also supported by animal studies wherein these subcortical structures are important in determining behaviorally aggressive responses (Siegel, Roeling, Gregg, & Kruk, 1999). While it has been argued that that the aggressive responses of the animals studied (rats and cats) do not perfectly map onto our conceptualizations of human aggression (Potegal & Stemmler, 2010), it is evident that these subcortical structures play a role in generating at least some types of human aggression (Blanchard & Blanchard, 2003). The notion that the amygdala and hypothalamus serve somewhat different functions in animals and humans is supported by research suggesting that although the amygdala is associated with aggressive responding, it is connected more strongly to fear response (Murphy, Nimmo-Smith, & Lawrence, 2003; Whalen et al., 2001). Additionally, damage and dysfunction in the amygdala has been associated primarily with increases in aggression, but not necessarily anger, leading some to theorize that processing of anger specifically in humans is performed mainly by other structures such as areas of the frontal cortex and areas of the temporal lobe.

Perhaps the most famous example of the role of the frontal lobe in anger processes is the large frontal lobe lesion incurred by Phineas Gage which subsequently made him a more irritable and aggressive individual (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994; Harlow, 1868). Since then, numerous studies have linked frontal lobe lesions to emotional lability as well as increases in anger and aggression in

individuals with premorbid tendencies toward anger and aggression (Greve et al., 2001; Silver & Yudofsky, 1987; Tateno, Jorge, Robert, & Robinson, 2003). In a study of Vietnam War veterans, those with damage to the anterior temporal lobe exhibited more anger and hostility while those with damage to the medio-frontal or orbito-frontal cortex exhibited more aggression and violence (Grafman et al., 1996). This suggests that angry emotionality as well as cognitive conceptualizations of anger (i.e. hostility) may be processed mainly by the anterior temporal lobe while the behavioral component may be more influenced by regions of the frontal cortex associated with decision making and social comportment. Supporting this notion, factor analyses reveal that the orbito-frontal cortex is involved in processing action related to both reward and punishment. EEG evidence supports frontal asymmetry in this type of anger response showing that greater left than right frontal activity is associated with anger-motivated approach while greater right than left frontal activity is associated with anger-motivated withdrawal (Carver & Harmon-Jones, 2009). However, other studies indicate that damage to areas of the frontal cortex such as the ventromedial frontal cortex and dorsofrontal regions have also been linked to increases in anger and hostility (Grafman, Vance, Weingartner, Salazar, & Amin, 1986). These mixed results on which regions are responsible for anger and which are responsible for aggression neglect the integrative nature of the structures involved. The pathways between frontal regions and temporal regions are reciprocal, creating a feedback loop which serves to alter the anger episode over its time course (Potegal & Stemmler, 2010). It has been hypothesized that areas processing angry emotions serve to mediate the effects of anger on calculating the pros and cons of an aggressive response,

linking both anger and aggression (Potegal & Stemmler, 2010). Despite these findings, we still lack a coherent understanding of how the different components of anger interact during an anger response.

Cognitive Component

The idea of a cognitive component to anger can be traced back to the frustration-aggression hypothesis (Dollard, Miller, Doob, Mowrer, & Sears, 1939). This theory, later revised, (Berkowitz, 1989) created a link between the state of frustration (an interpretation of negative stimuli in the environment) and feelings of anger as well as aggressive behavior. Since this theory, the cognitive component of anger has been conceptualized as being comprised of a pre-anger state as well as cognitions and appraisals that occur during a provocation (Deffenbacher, 2011). The pre-anger state involves an individual's mood (Berkowitz, 1990), the cultural and familial conceptualizations the individual has about anger and anger expression (Thomas, 2006), as well as the individual's personal world view (Deffenbacher & McKay, 2000; Kassinove & Tafrate, 2002). As stated earlier, the working theory of emotional responding involves a triggering event which is processed within the context of a system. The pre-anger state represents the cognitive make-up of the system through which the event is processed. When these pre-anger states are consistent over time and over diverse situations, they are conceptualized as traits. For instance, Spielberger (1999) identified that an individual's propensity for feeling angry (trait anger) and for acting aggressively (trait aggression) positively correlates with their intensity of anger and aggression during a provocation. One of the more researched traits as it relates to anger is hostility. At the

high end of the spectrum it is often evidenced by a “hostile attribution bias” which refers to the individual’s tendency to appraise other’s ambiguous behavior as hostile or threatening (Milich & Dodge, 1984; Crick & Dodge, 1996). Individuals high in trait hostility and/or who display a hostile attribution bias have been shown to be more likely to become angry during a neutral or provoking event and are more likely to act aggressively (Camodeca & Goossens, 2005; Epps & Kendall, 1995; Steinberg & Dodge, 1983).

During a provocation, appraisals about the triggering event often have an impact on how the angry emotion manifests (if at all). The idea that appraisals are important to emotional responding received traction during the cognitive revolution with the work of investigators such as Schachter and Singer (1962) and Lazarus (1967). Lazarus (1991) has discussed the idea of primary and secondary appraisals during a triggering event. Primary appraisals are thought to be directed at toward the trigger and its characteristics while secondary appraisals are directed toward personal coping resources (Deffenbacher, 2011). Primary appraisals such as perceiving the event as being intentional, preventable, unwarranted, and/or blameworthy have been shown to increase the probability and intensity of anger (Deffenbacher & McKay, 2000; Kassinove & Tafrate, 2002). Secondary appraisals such as feeling overwhelmed or unable to cope, feeling as though one should not have to deal with or experience negative emotions, and feeling as though anger and/or aggression is an appropriate response to the event, have all been associated with a high probability of becoming angry as well as higher levels of anger (Deffenbacher & McKay, 2000). In summation, the cognitive component of anger has

been shown to substantively impact not only the intensity of an anger response but also whether or not an angry response occurs. Unfortunately, cognitive appraisals are difficult to measure in real time, and as such, the way in which primary and secondary appraisals change over the course of a provocation is not well understood.

Behavioral Component

The third conceptual component of anger is the behavioral component which is often understood as the choice to engage in various forms of aggressive behavior. Although aggressive behavior can also be conceptualized as a consequent or correlate of anger as opposed to a direct component, its impact on the overall experience of an angry episode makes it an important aspect to include in our conceptualization (Potegal & Stemmler, 2010). Research suggests that individuals who are more likely to respond with aggression experience their outbursts differently than non-aggressive individuals (Kulper, Kleiman, McCloskey, Berman, & Coccaro, 2014). And, as different forms of aggression can occur throughout the length of a provocation, aggression is likely to impact the other components throughout the time course. In the same vein, when anger is supplemented by aggression, the deleterious correlates observed are often more pronounced. Individuals who are physically aggressive report damaged friendships, increased rates of alcohol use, legal and vocational difficulties (Deffenbacher, Oetting, Lynch, & Morris, 1996), and negative health outcomes (McCloskey, Kleabir, Berman, Chen, & Coccaro, 2010). Aggression also has a high societal cost in terms of violent crime, child and elder abuse, intimate partner violence, and billions of dollars per annum in lost productivity and

violence related expenditures (Max, Rice, Finkelstein, Bardwell, & Leadbetter, 2004; World Health Organization, 2002).

Aggression can vary in both form and function. With respect to form, aggression can be expressed along a continuum of severity from verbal to lethal physical aggression (McCloskey, Berman, Noblett, & Coccaro, 2006; Salari & Baldwin, 2002). Verbal aggression (e.g., yelling, screaming, heated arguments) is the most common form of aggression with almost everyone engaging in some form of verbal aggression at some point in their life (Averill, 1983). Physical aggression is a less common and typically more severe form of aggression that can be broadly divided into aggression against property (e.g., hitting walls, throwing a cell phone) and aggression against others (e.g., slapping, punching, or kicking another person). A recent study showed that approximately half of all adults have engaged in some form of physical aggression during their adult lifetime (Kessler, Coccaro, Fava, & McLaughlin, 2012). The function of aggression can also vary. Aggressive acts are often conceptualized as either "affective" or "instrumental" (Anderson & Bushman, 2002a), with the former describing aggression used to express anger and/or retaliate and the latter referring to the use of aggression as a tool to achieve a goal not directly connected to anger (e.g., knocking someone down to rob them). Though the same aggressive act can serve both of these functions (Bushman & Anderson, 2001), it is believed the large majority of aggression is primarily affective (Averill, 1983; Crick & Dodge, 1994) suggesting that subjective anger is the primary motivator for aggressive behavior.

Subjective Component

The subjective component of anger represents our experience of anger as a whole. This component is generally characterized by the qualitative aspects of anger and is our individual representation of the experience of an angry emotion. This component of the anger experience is the most commonly assessed, usually by the question "How angry are/were you?" In this sense, the subjective component is viewed as one's level of state anger and is comprised of the individual's own perception of their anger. This component is thought of as distinct, albeit overlapping with other components of anger due to the generally moderate correlations between physiological indices (e.g. autonomic arousal), subjective self-report of angry affect, self-reported cognitive appraisals of the situation, and aggressive response (Deffenbacher, 2011; Potegal & Stemmler, 2010). For example, concordance between an individual's self-report of an emotion and the physiological and/or behavioral response record is often poor, suggesting that the subjective experience of an emotion is distinct from the other components (Stemmler, 1992; Weinstein, Averill, Opton, & Lazarus, 1968). Furthermore, studies have shown that high levels of anger can be present without the existence of aggression (Averill, 1983), while aggressive behavior is not always accompanied by increased anger (Blair, 2008; Glenn & Raine, 2009) again indicating that there is separation between the two components.

While the subjective experience of anger is largely viewed as a distinct, yet overlapping component of anger, some theories of emotion conceptualize this component as more of an overarching concept (Davidson, 1992). In this conceptualization, anger

represents a gestalt, the (not necessarily linear) summation of the physiological, cognitive, and behavioral components of anger. As mentioned earlier, the physiological components often contribute heavily to the subjective experience of the episode (Böddeker & Stemmler, 2000). The subjective experience of anger can also be colored and influenced by cognitive appraisals of the event, leading to feelings of shame and guilt, embarrassment, or even pleasure (Deffenbacher et al., 1996; Kulper et al., 2014). These theories are sometimes understood under a reductionist model where all components are understood as physiological and neuronal responses (Davidson, 1992). When each component, as well as the interactions between them, are integrated by our brain, the subjective experience is the resultant of these factors as opposed to a separate component. Studies on emotional response coherence (the agreement between the different components of an emotion) have found some evidence for correlations between components during an emotional event, suggesting that the components work together, integratively to produce a resultant experience (Mauss, Levenson, McCarter, Wilhelm, & Gross, 2005). This idea is supported by the reciprocal neurological pathways discussed earlier suggesting that the different aspects of an emotional response can change each other throughout the course of an event. This provides us with two working models of anger, one where the subjective component is combined with the other components to produce the resulting emotion of anger and another where the subjective component *is* the resulting emotion of anger (See Figure 2). It is currently unclear which theory more accurately represents the reality of how anger is manifested and understood. This uncertainty is in part due to our limited understanding of how the different components of

anger change over the course of a provocation. Additionally, it has been posited that the interactions between the components of anger and the environment as well as our perception of how an anger episode plays out over time are often difficult to assess experimentally (Davidson, 1998).

The Time Course of Emotion

Although researchers have done well to explore the different components of anger, and in some cases the interactions between these components (Mauss et al., 2005), our understanding of how these components play out over the course of an angry response is relatively poor. In response, researchers in the field have since extended the theory of anger toward a systems-oriented approach which embeds the angry emotion in the context of an environmental system (Robins & Novaco, 1999). The main point of this extension is the recognition that emotions are not static events. Anger is dynamic, changing as the angry event plays out. In their formulation of this approach, Robins and Novaco (1999) criticize researchers for focusing too much on the immediate circumstances and current thoughts. As such there is a call to extend our current understanding of the dynamics of anger from a flat one-dimensional approach to an evaluation of the event as it plays out over time.

Davidson (1998) uses the concept of "affective chronometry" or the study of the temporal dynamics of emotional reactions. In his description, Davidson points to four key components of affective chronometry, namely threshold, peak/amplitude, rise time to peak, and recovery time. The threshold is the amount of a particular stimulus that an individual needs to trigger an affective response. The peak or amplitude is the highest

level of a particular emotion that one experiences after a triggering event. The rise time to peak and recovery time are simply the rise and fall of the emotion from baseline and back again. Davidson proposes that there are important individual differences among these variables and that more research on what these individual differences may be, why these differences exist, and what these differences can tell us about the way particular people react in different situations is essential to our understanding of emotional responding.

Research in affective chronometry is intended to build upon the notion of separate components of an emotional response by investigating how these components change over time and how they might interact with each other. This framework changes the basic mechanistic conceptualization (event + internal composition = emotional response) into a more complex equation wherein the event and the emotional components are continuously in flux, affecting the emotional response in different ways across time. By investigating the temporal dynamics at play in emotional responding, we can establish a more nuanced account of the way an individual and his/her environment "co-act" to create an emotional response. Due to the relatively young nature of this particular endeavor, our understanding of the normative aspects of these processes is sparse (Davidson, 2004). Additionally, as opposed to running more descriptive studies evaluating the normative processes, researchers have instead investigated a number of individual differences that help explain some of the variance in emotional responding.

Threshold

The threshold of an emotional response refers to the amount of a stimulus needed to produce an emotional response. Our knowledge of normative thresholds for emotional

responses is largely based upon the development of emotion-elicitation paradigms. In these types of paradigms, threshold is reached when participants move significantly from a "baseline" state after viewing an emotion-inducing stimulus (e.g. Gross & Levenson, 1995; Lang, Bradley, & Cuthbert, 1997). Thus, any stimulus that creates an emotional response exceeds that response's threshold. For example, in research using the International Affective Picture System (IAPS), a picture of a gun exceeds the physiological threshold for fear as evidenced by increased heart rate when an individual is exposed to the stimulus (Lang, Bradley, & Cuthbert, 2001). This description points to an inherent problem in categorizing normative thresholds, namely, creating an objective measure of the emotional arousal potential of a stimulus. To this end, researchers have developed sets of stimuli with categorizations based on participants' ratings of the valence (positive or negative) and arousal (high vs. low) (Lang et al., 1997). While these sets of stimuli, organized this way, provide us with an objective way to perform a dose-response test to see what types of stimuli exceed thresholds for different emotions as well as different components of emotions, no such study to date has been carried out. One potential reason for this is that threshold is conceptualized as an all-or-nothing concept where a stimulus either exceeds the threshold or not. Because the threshold for emotional response is thought to be very low (Davidson, 1992), this type of research is less informative. Instead, researchers have sought to explicate some of the individual differences that effect whether or not a person is emotionally responsive to a stimulus.

To this end, studies have used a diverse set of concepts to investigate the issue. Threshold has been studied under monikers such as emotion sensitivity, lability,

arousability, startle response, and neuroticism (Guarino, Roger, & Olason, 2007). Problematically, these concepts are functionally different, and while they can assess forms of threshold, it is often difficult to compare studies using different concepts. Consequently, it is often hard to establish conceptualizations of normative thresholds for individuals. Instead, researchers have focused on highlighting individual differences in both baseline arousability and sensitivity to emotional stimuli. For example lower thresholds for emotional responding has been exhibited in women (Gard & Kring, 2007; Sharp, Van Goozen, & Goodyer, 2006), younger individuals (Lundqvist, Svard, & Fischer, 2013; Scheibe & Carstensen, 2010) and those with a low socioeconomic status (Kishiyama, Boyce, Jimenez, Perry, & Knight, 2009). Similarly, lower thresholds in response to negatively valenced stimuli have been shown for individuals with various forms of psychopathology including post-traumatic stress disorder (PTSD) (Wolf, Miller, & McKinney, 2009), depression (Hamilton & Gotlib, 2008) and anxiety (Stenberg, 1992). Conversely, individuals with high levels of psychopathy exhibit higher thresholds in response to negatively valenced stimuli (Blair, Jones, Clark, & Smith, 1997). Although these individual differences in emotional threshold allow us to more easily predict whether or not an individual will have an emotional reaction, negative consequences associated with emotional outbursts are usually more related to the magnitude of the emotional response, described as the peak/amplitude.

Peak/amplitude

The peak, or amplitude, of an emotional response is characterized by the highest level of experienced emotion (or component of an emotion) throughout the emotion

episode (Davidson, 1998). During most emotional responses, there is one peak which occurs shortly after a triggering event (Frijda, 2007). The actual amplitude of the peak is heavily dependent on the intensity of the triggering event. For images, the amplitude of an emotional response is positively correlated with the judged arousability of an image as well as the strength of the valence (positive or negative) (Lang, Bradley, & Cuthbert, 1999). Physiologically, the intensity of negative emotional responses to visual stimuli has been associated with a greater magnitude of activation in the occipital lobe and longer duration of activation in the prefrontal cortex and the cingulate cortex (Waugh, Hamilton, & Gotlib, 2010) underscoring the role of biological processes in the experience of emotion. Similarly, increases in peak emotion have also been linked to increases in measurements of the autonomic nervous system thought to be associated with that emotion. For example, magnitude of subjective fear has been correlated with increases in heart rate (e.g. Bradley & Lang, 2000). As with the other components of the time course, the majority of research on peak amplitude of emotional responding has been devoted to understanding the influence of individual differences. Specifically, higher affect magnitude (sometimes referred to as “affect intensity”) has been linked to higher scores on temperament measures of sociability, arousability/reactivity and emotionality in addition to measures of somatic and neurotic symptoms (Bachorowski & Braaten, 1994; Larsen & Diener, 1987; Larson, Diener, & Emmons, 1986). Additionally, individuals with high levels of emotion dysregulation, such as those with borderline personality disorder, consistently report higher levels of peak affect, particularly for negative emotions (Levine, Marziali, & Hood, 1997). This suggests that trait-like mechanisms can

influence the relationship between stimuli and emotional response. Along with threshold, peak is conceptualized as a static component of the time course, occurring at one time point. The dynamic quality of an emotional response is characterized by the rise and recovery periods.

Duration: Rise and Recovery

In addition to the peak amplitude of emotional response, researchers have sought to investigate how this intensity changes over time, creating the characteristic rise and fall of emotion (Davidson, 1998). In characterizing the duration of emotional reactions, findings have been mixed (Potegal, Kosorok, & Davidson, 1996). The duration of an emotional response was initially believed to be quick, sometimes conceptualized as "activity bursts" (Ekman, Friesen, & Ellsworth, 1972; Frijda, Mesquita, Sonnemans, & Van Goozen, 1991; Levenson, 1988). However, more recent studies have supported the idea that emotional responses can last for minutes, hours, or in some cases, even longer (Scherer, Walbott, & Summerfield, 1986; Sonnemans & Frijda, 1994; Verduyn, Delvaux, Van Coillie, Tuerlinckx, & Van Mechelen, 2009). The trajectory of an emotional response is typically quick with a steep rise to a single peak followed by a more gradual return back to a baseline state (Frijda, 2007; Verduyn, Van Mechelen, Tuerlinckx, Meers, & Van Collie, 2009).

As with the other aspects of the time course, the rise time and recovery period have been shown to be subject to a number of individual differences. Previous research has found a positive association between neuroticism and recovery time for negative affect (Suls, Green, & Hillis, 1998). Other studies show distinct "affect change profiles"

in which participants who are extraverted, emotionally stable, and have high negative mood regulation expectancies (the participants expected to be able to regulate their negative moods) experience slow rates of positive affect decay and faster rates of negative affect decay relative to participants who are introverted, neurotic, and had low negative mood regulation expectancies (Hemenover, 2003). These studies suggest a link between enduring personality traits and aspects of the time course of an emotion. However, in most of these studies, negative affect is more closely related to sadness or anxiety as opposed to anger, which has been shown to be functionally different from sadness or fear on a number of different emotional outcome variables (Ekman, 1999). As theorists have posited that different emotions may have unique time courses (Davidson, 1998) it seems plausible that personality traits may vary to the extent which they impact the time course based on the specific emotion (e.g. sadness vs. anger). This is still an open question in the field and requires more concentrated research on particular emotions.

The Time Course of Anger

Although the emotion of anger has received considerable attention from researchers over the past century, relatively little attention has been paid to discerning its temporal dynamics. While some studies have looked at the phenomenon directly, the majority of studies approach the topic peripherally while in pursuit of other aims. In some research, the time course of anger has referred to the frequency at which angry episodes occur over different spans of time. The earliest studies of these temporal dynamics of anger utilized anger journals or “logs” and had participants record their angry emotions

over an extended period of time (e.g. Anastasi, Cohen, & Spatz, 1948; Deffenbacher, Demm, & Brandon, 1986; Stratton, 1927). While these studies provided information about the frequency of angry emotions, the logs were not nuanced enough to ascertain the time course of individual provocations. For this review, the time course of anger will refer to the temporal dynamics at play in an individual anger episode. Consistent with other emotions, the time course of anger can be broken down into the threshold, peak/amplitude, the rise time to peak, and the recovery time.

Threshold

The normative threshold for an anger response has not been studied directly. This is largely due to the methodological considerations wherein stimuli presented to the participants are specifically designed to surpass the individual's threshold and produce an anger response. Otherwise, the nature and effects of the anger response could not be investigated. Consequently, individual differences in threshold levels for anger have also not been studied directly. Studies assessing the overall frequency of anger episodes and aggressive behavior may provide some insight into how certain individual differences could make an individual more sensitive to provoking stimuli. For example, greater frequency of anger has been shown in individuals with high trait anger (Tafrate, Kassinove, & Dundin, 2002), younger individuals (Mirowsky & Ross, 1999), and individuals with lower socio-economic statuses (Schieman, 2003). Similarly, greater frequency of aggressive behavior has been shown in individuals high in impulsivity (Coccaro, 2013), high in trait hostility (McCloskey et al., 2006), and individuals with various forms of psychopathology (Novaco, 2010). While it is plausible that these

individuals have lower thresholds resulting in more frequent anger and aggression, it is unclear as to whether or not these individuals experience more frequent provocations which could also account for these findings. As such, before we can say anything definitive about the normative threshold for anger, more research needs to be completed. The peak/amplitude of anger has been studied more directly.

Peak/Amplitude

The peak, (a.k.a. "amplitude" or "intensity"), of an anger response is typically measured as a subjective rating of anger during an angry episode, usually on a 0-10 scale. While the peak is subject to variation (Ferguson & Rule, 1983), studies show that the typical peak of an anger response is about 50% of maximum possible intensity–a five out of ten (Beck & Fernandez, 1998; Fernandez & Beck, 2001; Jacob et al, 2008; Stets & Tsushima, 2001). Similar to the study of time course in other emotions, most studies have investigated the effect of situational and intrapersonal factors on the different time course components. Provocation is strongly associated with anger intensity, with more intense provocations resulting in more intense anger and aggression (Ferguson & Rule, 1983; Santor, Ingram, & Kusumakar, 2003). The level of provocation itself is impacted by several factors including whether the event occurs in public (Vasquez, Osman, & Wood, 2012) as well as how intentional, preventable, and unwarranted the provocation is believed to be (Deffenbacher & McKay, 2000; Kassinove & Tafrate, 2002). All of these variables increase the perceived intensity of the provocation and resultant anger and aggression (Geen, 2001). Contextual factors such as hot temperatures (Anderson & Anderson, 1996) and acute intoxication (Bushman, 1993; Giancola, 2013; McCloskey,

Berman, Echevarria, & Coccaro, 2009) also tend to increase subjective anger intensity in response to provocation both inside and outside of the laboratory.

Some enduring intrapersonal characteristics such as trait anger, trait aggression, trait hostility, and emotion regulation are associated with higher peaks of subjective anger during an anger episode (Spielberger, Krasner, & Solomon, 1988; Spielberger, Reheiser, & Sydeman, 1995). Additionally, individuals high in these traits also report higher peaks of aggressive behavior and display high peaks of aggression during laboratory tasks (McCloskey et al., 2006; Spielberger et al., 1995). Deficits in emotion regulation processes have also been associated with higher peaks of both anger and aggression (Gross, 2002). This link has been supported by research investigating clinical populations exhibiting deficits in emotion regulation such as individuals with depression (Fava & Rosenbaum, 1998), PTSD (Novaco & Chemtob, 1998), and Intermittent Explosive Disorder (IED) (McCloskey et al., 2006). These individuals report higher peak levels of anger than individuals without psychopathology (Novaco, 2010). However, it should be noted that individuals from these clinical populations are also often higher in trait anger, aggression, and hostility, so it is unclear to what extent these characteristics independently impact peak anger. As research into the peak of anger typically only assesses subjective anger, more multimodal assessment would better define the peak of the other components of anger. Trait hostility has been linked to higher peaks of physiological measures of anger such as blood pressure and heart rate (Suarez, Kuhn, Schanberg, Williams, & Zimmermann, 1998; Suls & Wan, 2007.) However, comparable research on peak anger and other traits is lacking. Furthermore, while research into peak

levels of anger has provided us with good methods by which to predict the overall amplitude of the anger response, it says little about how the anger experience changes over time. For this, researchers have investigated the duration of an angry episode, and more specifically, the rise time and recovery period of anger.

Duration: Rise Time and Recovery

Much like estimates of the duration of emotional reactions in general, the duration of angry episodes has ranged from estimates of "a few minutes" (Frijda et al., 1991) to hours (Simon & Nath, 2004; Stets & Tsushima, 2001). The earliest survey of the duration of angry episodes found that reported durations of college women's angry episodes followed a bimodal distribution with a near equal amount of episodes lasting ten to twenty minutes and five minutes or less (Gates, 1926). Subsequent studies have also found a bimodal distribution of the duration of anger with participants endorsing quick anger episodes lasting on the scale of minutes as well as a longer anger episodes lasting from hours to a day or more (Fridhandler & Averill, 1982; Kassinove, Sukhodolsky, Tsytsarev, & Solovyova, 1997; Meltzer, 1933; Simon & Nath, 2004; Stets & Tsushima, 2001; Ueda, 1962). These studies all utilized retrospective self-report wherein the definition of an "angry episode" was largely left to the individual. It is likely that those endorsing longer durations are not capturing the trajectory of the initial angry response, but rather the reactivation of the angry response by environmental cues (Potegal, 2010). Consequently, our understanding of the duration of a "typical" anger event is not well defined. Furthermore, studies utilizing a standardized provocation have not continually measured subjective anger, making comparisons across studies difficult. While these

studies have investigated the duration of anger, some studies provide a more nuanced account of the time course looking at the rise time to peak and the recovery period of anger.

For anger, the rise time to peak and the recovery time have been investigated primarily in the temper tantrums of children because children are believed to have less capacity for emotion regulation and consequently are less able to repress or mitigate their emotional response, resulting in a "purer" measure of the typical time course of anger (Potegal, Kosorok, & Davidson, 1996). The time course of anger has been characterized in a series of experiments evaluating temper tantrums in young children (ages 1 ½ to 5) (Potegal, Kosorok, & Davidson, 1996; 2003; Qiu, Yang, & Potegal, 2009). Results showed that the recovery from a peak level of anger intensity occurred at a much slower rate than the build to that peak level, and that 75% of tantrums lasted five minutes or less from first observed behavior back to a baseline state (Potegal et al., 2003). Modally, tantrums lasted 30 seconds to a minute. There is also some evidence that tantrum duration increases with age from a mean of two minutes at eighteen months to between four and five minutes in three- and four-year-olds (Qiu et al., 2009).

While evaluating the time course in children allows us to investigate the relationship between the rise and fall of anger, we are also aware that this response changes in key ways as humans develop. As noted earlier, adults are more likely to use emotion regulation strategies. Additionally, these studies of children focus solely on behavioral indicators that the researchers equate with an angry emotion. This could be problematic because the behavioral component of anger is only one facet of the anger

experience and is likely not synonymous with subjective anger as has been shown in other emotions (Hollenstein & Lanteigne, 2014; Mauss, Wilhelm, & Gross, 2004). While concordance rates are likely higher in young children (Potegal & Qiu, 2010), this still creates a problem for the generalization of these findings to adults and even adolescents. In order to understand the different components of the time course of anger in adults, additional investigation is needed.

In a sample of healthy adults, the time course of anger has been examined in a series of studies using participant generated anger charts (Beck & Fernandez, 1998; Fernandez & Beck, 2001). Across both studies results showed that the average length of an angry episode was an hour and a half with a standard deviation of about an hour. This is consistent with previous studies that demonstrated the variability of anger duration (e.g. Stets & Tsushima, 2001) and may be indicative of the bimodal distribution found in other studies (Fridhandler & Averill, 1982). Furthermore, in 94% of angry episodes, the time from onset of anger to the peak was significantly quicker (average of 30 seconds) than the recovery period back to baseline (average of an hour and a half) (Beck & Fernandez, 1998; Fernandez & Beck, 2001). This pattern has also been shown in a study of the time course using women with borderline personality disorder (BPD) and healthy controls wherein participants reached a peak level quickly after mood induction, but had not returned to baseline levels at the time of the last assessment (three minutes after provocation) (Jacob et al., 2008). While both groups reported a similar trajectory of quickly ascending anger followed by an extended recovery period, the women in the BPD group reported higher levels of anger during the last assessment, suggesting a longer

recovery period than healthy controls. As such, the characteristic negatively decelerating slope of anger across time has been a robust finding. However, the duration as well as the rise and fall of anger can be influenced by a number of different factors.

In terms of factors affecting the time course, the nature of the provocation has again been shown to be influential. Specifically, researchers have identified that typical social interactions follow a "tit-for-tat" rule (Gouldner, 1960) indicating that as the intensity and duration of the provocation increases, so does the intensity and duration of exhibited aggression, feelings of anger, and reported hostility (Averill, 1983; Dodge & Coie, 1987; Pruitt, Parker, & Mikolic, 1997). This suggests that the time course of both the rise and fall of anger can be heavily influenced by the rise and fall of the provocation. Duration is also positively correlated with subjective peak anger in both children (Potegal et al., 2003) and adults (Gates, 1926; Fernandez & Beck, 2001; Fridhandler & Averill, 1982; Mikula, Scherer, & Athenstaedt, 1998) suggesting that the more intense the experienced anger, the longer it takes to rise to peak anger and recover back to a baseline level.

Intrapersonal differences also influence the overall time course. Higher levels of trait hostility have been associated with faster processing of negative information about others suggesting a quicker rise time to peak anger (Allred & Smith, 1991) as well as slower rates of physiological recovery and longer overall recovery times (Fredrickson, 2000). Theories of emotion dysregulation posit that highly dysregulated individuals experience a faster build to anger and a longer recovery period when compared to non-dysregulated individuals (Linehan, 1993) as has been shown in both women with BPD

(Jacob et al., 2008) as well as veterans suffering from PTSD (Beckham et al., 2002). While the studies mentioned thus far give a general account of the normative time course of anger and provide us with a number of factors potentially impacting the elements of an individual's time course, there remain a great number of questions about the time course of anger.

Conclusion and Future Directions

Research to date has provided us with a rough account of the temporal dynamics of anger. The most robust finding has been that the rise time to peak anger is shorter than the recovery period back to a baseline state. This has been shown in students, adults, children, and individuals with some level of psychopathology. Studies suggest that a typical angry episode lasts between a few minutes and a few hours, however more rigorous study of the topic is needed due to previous studies' reliance on retrospective self-report. Limited data suggests that subjective peak levels of anger during a provocation are typically moderate (e.g. five out of ten) however, the findings from these studies are hampered by the subjective nature of these ratings. Additionally, what these subjective ratings mean in terms of physiology, behavior, and cognitive processes remains an open question in the field. Finally, the normative threshold for an anger response has not been directly tested to date. Potential factors affecting the time course of anger include factors related to the provocation such as location and intensity of the provocation, situational factors such as alcohol influence and the weather, and enduring characteristics such as trait anger, aggression, hostility, and emotion regulation. In the

end, our conceptualization of the time course is beginning to take shape, but will require much more research to become well understood.

Through our review of the extant literature on the time course of anger, we have been able to identify a number of important questions that remain to be answered, including methodological issues to be addressed. First, the extreme variability in self-reported duration and intensity of anger episodes has caused problems in determining a normative account of these processes (Potegal, 2010). Although much of this variability can be attributed to differences in provocation and lack of experimental control of factors known to affect anger response, another potential source of inconsistency is that individuals are usually asked to report on their own angry episodes either from memory or shortly after the provocation. This creates problems of retrospective recall validity which has been shown to be lower when reporting negative mood states (Sato & Kawahara, 2011). Additionally, due to vague and/or non-existent definitions for when an angry episode "ends," it is likely that some individuals are endorsing a ruminative form of anger where angry emotions are reactivated by memories, but are distinct from the initial reaction to the provocation (Potegal, 2010).

Secondly, studies on the time course of anger have almost exclusively examined the subjective experience of anger and have not measured the differing time courses of other components of the emotion. As such, there is a dearth of research on the time course for the physiological, cognitive, and behavioral components of anger. If we accept the model that the subjective experience of anger is the resultant of the other components, we are still unsure which components are accounting for the majority of variance for the

peak, the rise, and fall of subjective anger and conditions under which these phenomena occur. This line of inquiry is likely to prove fruitful as subjective experiences of emotions often do not correlate well with physiological and behavioral measures (Levenson, 2014) indicating that this experience must be different in some way from the time course of other components.

These current gaps in the literature are important to address for several reasons. First, it is clear that the components of anger have the capacity to influence each other as evidenced by studies showing how changing one component influences the resulting emotion (e.g. introducing a cognitive restructuring component as in Zaunmüller, Lutz, & Strauman, 2013). This is also supported physiologically by neurobiological evidence showing reciprocal pathways among structures thought to primarily handle different components of the experience (Potegal & Stemmler, 2010). Second, we understand that an angry experience is not produced in a vacuum. Each anger experience has an impact on our physiological, cognitive, and behavioral outcomes for the next provoking event. This is evident in the cognitive concept of pre-anger states that can dictate the course of an angry response (Berkowitz, 1990; Deffenbacher, 2011). Finally, the variability in the time course of anger both within individuals and between them is likely significantly influenced by a number of factors associated with both the individual (e.g. trait-like characteristics) and the situation (e.g. provocation and other contextual factors). As such, the resulting time course is a complex equation that we are only beginning to flesh out, and until these gaps are filled, our understanding of anger will remain incomplete.

The relative lack of research on this subject is due in part to the methodological difficulties in assessing the time course of anger. For one, anger needs to be induced in participants either naturalistically or experimentally. Naturalistic inductions have the benefit of being ecologically valid, but lead to the variability and retrospective self-report difficulties stated above. Experimental provocations allow for more situational control, but there is often concern about generalizing anger in a laboratory setting to anger outside of the lab. Additionally, to evaluate the dynamic aspects of the time course, one needs to measure the different components of an anger experience on a near continuous level. This is often difficult in both naturalistic and laboratory settings alike (Davidson, 1998).

Important for this type of research design is developing a strong and ecologically valid standardized provocation to compare between as well as within subjects. Anger induction (as opposed to inducing other emotions) is contingent upon appraisals such as perceiving the provoking event as being intentional, preventable, unwarranted, and/or blameworthy (Deffenbacher & McKay, 2000; Kassinove & Tafrate, 2002). These conditions are difficult to achieve with common methods of emotion induction such as the passive viewing of pictures (e.g., IAPS; Lang et al., 1997), emotional human faces (e.g., Tottenham, Borscheid, Ellertsen, Marcus, & Nelson, 2002), and films (e.g., Gross & Levenson, 1995). One study investigating the time course of anger utilized stories to induce angry emotions, asking the participant to picture themselves as the protagonist of the story (Jacob et al., 2008). While this strategy is superior to retrospective reports as well as the passive viewing of stimuli, more ecologically valid provocation measures exist. Studies have shown that laboratory provocations such as the Taylor Aggression

Paradigm (Taylor, 1967) and tasks that have a confederate express unpopular opinions in front of the participantt (Wheeler & Caggiula, 1966) or negatively evaluate their work (Rohsenow & Bachorowski, 1984) demonstrate strong external validity in terms of levels of anger and aggression as well as the ability to reproduce known individual difference effects (Anderson & Bushman, 1997). These types of laboratory provocations additionally allow one to collect behavioral data on the level of aggression displayed as well as physiological data throughout the provocation. This type of design not only allows us to categorize the time course of those components, but also to begin evaluating how the different components work together to produce the resultant experience.

Additionally, investigation into how certain individual differences affect the time courses of different components of anger would greatly add to our understanding of how the interplay between environmental stimuli and the make-up of an individual's internal system produce the resultant experience. By determining what traits cause variations in the time course, we can hope to better predict aggression and violence in an individual. This type of research could also have treatment implications for individuals who are experiencing forms of psychopathology related to higher levels of anger and aggression such as BPD, PTSD, and IED. Our current understanding of the time course shows that peak anger is reached quickly which means that interventions must act within that small window of time. Additionally, barriers to recovery from anger such as rumination and lack of emotion regulation strategies, while already addressed in some cognitive behavioral therapies for anger (Deffenbacher, 2011), could be more of major focus for treatment. As such, it seems important to investigate both the different components of the

anger response as well as which individual differences predict more deleterious time courses.

Despite the ubiquity of anger and the negative consequences of dysregulated anger, research into this basic human emotion has lagged behind that of other emotions. The limited research to date has provided some insights into the emotion of anger (e.g. anger rises more rapidly than it diminishes) but much about the time course of anger remains unanswered. Novel approaches may assist in this search. However, until a more systematic, holistic, and dynamic assessment of the time course of anger is performed, it is unlikely that significant progress will be made.

CHAPTER 3

ADDITIONAL RESULTS

Non-Responders

A number of individuals were excluded from time course analyses due to low variability over the course of the provocation (non-responders) for subjective anger (n = 35), aggression (n = 19), HR (n = 8), GSR (n = 6), and HF HRV (n = 24). Due to the higher number of participants excluded from the subjective anger, aggression, and HF HRV analyses, these individuals were compared to individuals included in those analyses (responders) on demographic and trait variables (See Table 5). Chi-square tests on gender between the two groups revealed that aggression non-responders were more likely to be male than aggression responders ($\chi^2 = 6.26$, $p = .01$; $\varphi = .28$). T-tests between trait variable means show that subjective anger non-responders had lower trait hostility when compared to responders (t (77) = 2.84, $p < .01$, $d = .65$). Aggression non-responders had lower emotion dysregulation scores than responders (t (77) = 2.10, $p = .04$, $d = .48$). Finally, HF HRV non-responders exhibited higher trait anger than responders (t (77) = 2.10, $p = .04$, $d = .48$). Overall the results suggest trait variables may have had a modest influence on responder status.

The proportion of non-responders was also compared across subjective anger, aggression and HF HRV. Subjective anger and aggression shared 9 non-responders with the proportion of anger non responders evenly distributed across aggression responders and non-responders ($\chi^2 = 0.10$, $p = .76$). Likewise, subjective anger and HF HRV shared seven non-responders, with the proportion of anger non responders evenly distributed

across HF HRV responders and non-responders ($\chi^2 = 3.20$, $p = .07$). Finally, aggression and HF HRV shared five non-responders with the proportion of aggression non responders evenly distributed across HF HRV responders and non-responders ($\chi^2 = 0.20$, $p = .66$). Overall, results show that non-responders were evenly distributed across the three variables.

Effects of MTAP on Fear and Happiness

There was an overall effect of block on subjective fear (F (8, 600) = 20.24. $p < .001$, $\eta^2 = .21$). Post-hoc analyses examined if fear increased from baseline (block 2) to peak provocation (mean of blocks 4 and 5) and then decreased post provocation (mean of blocks 7, 8, and 9). There was an overall increase in subjective fear from baseline (M = 1.26, SD = 1.85) to peak provocation (M = 2.63, SD = 2.72; t (75) = 5.39, $p < .001$, d = 1.24) and a similar decrease in subjective fear from peak provocation to post-provocation (M = 1.17, SD = 1.90; t (75) = 5.95, $p < .001$, d = 1.37) (see also Figure 6). Thus, the MTAP was effective at eliciting a fear response. Similar to anger, rise to time fear (M = 2.64 trials, SD = 2.75) was quicker than return to baseline (M = 9.55 trials (SD = 7.00); t (54) = 6.96, $p < .001$, d = 1.89). Furthermore, peak fear (M = 4.16, SD = 3.26) was greater than peak anger, (t (75) = 3.12, $p < .01$, d = .72), suggesting that the MTAP elicited even more subjective fear than anger. Fear return time was negatively correlated with trait aggression ($r = -.28$, $p = .04$); however, no fear time course variable (rise time, peak, return time) was associated with any other trait variable (anger, aggression, hostility, emotion regulation deficits, all r's between -.14 and .15, all p's between .19 and .86). An analysis of fear non-responders vs anger non-responders revealed that there were

a higher proportion of anger non-responders than fear non-responders (χ^2 (df = 1) = 6.99, p < .01, φ = .30). These findings suggest that the MTAP was an effective fear provocation task and that the relationship between the rise time and return time of subjective fear is similar to that of subjective anger.

There was an overall effect of block on subjective happiness (F (8, 600) = 4.62. p < .001, η^2 = .06). Post-hoc analyses examined if happiness decreased from baseline (block 2) to peak provocation (mean of blocks 4 and 5) and then increased post provocation (mean of blocks 7, 8, and 9). There was an overall decrease in subjective happiness from baseline (M = 5.89, SD = 2.37) to peak provocation (M = 5.03, SD = 2.68; t (75) = 3.69, p < .001, d = .85). Subjective happiness did not change from peak provocation to post-provocation (M = 5.42, SD = 2.64; t (75) = 1.84, p = .07, d = .42) (see also Figure 7). These findings indicate that happiness decreased during the MTAP as anger and fear increased, however happiness generally did not return to baseline levels after provocation.

Secondary Analyses of Anger Time Course

In addition to the computed time course variables for rise time and return time, raw outcome data for each component were combined into blocks that assessed the five time periods of interest; baseline (block 2), ascending provocation (block 3), peak provocation (block 4/5), descending provocation (block 6), post-provocation (block 7-9). An analysis of outliers, skewness, and kurtosis revealed significant skew (skewness > 2) for subjective anger in 2 out of 5 blocks, GSR in 3 out of 5 blocks, and HF HRV in 4 out of 5 blocks. As such, all blocks for these three components were log-transformed. This

transformation normalized data for both GSR and HF HRV, however subjective anger data remained significantly skewed (1 block with skew statistics above 2). Non-parametric tests (Wilcoxon Signed Ranks Tests) were performed using the log transformed subjective anger data and did not differ in the overall pattern of results from parametric tests. As such, parametric tests were used in the final analyses.

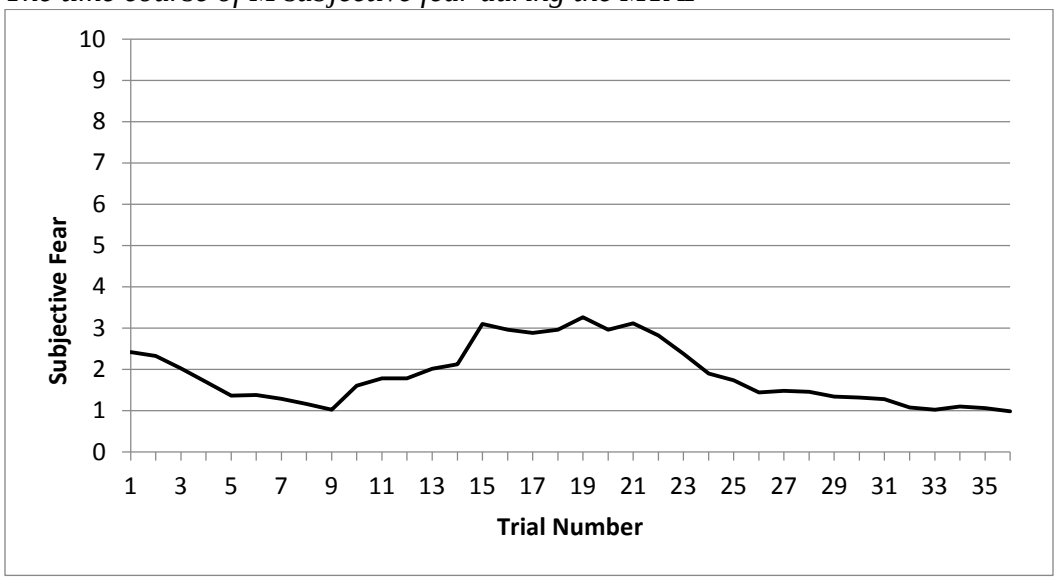
Overall time course was determined by performing repeated measures ANOVAs across the five time periods for each component variable (subjective anger, aggression, heart rate, GSR, and HF HRV) (See Table 6 for block means and SDs). When the sphericity assumption was violated as indicated by a significant Mauchly's Test of Sphericity, the Greenhouse-Geisser correction statistics were used. All omnibus tests indicated significant main effects of provocation period for each anger component (all *F*s between 6.21 and 72.94, all *p*'s < .001). Post-hoc comparisons looking at sequential provocation periods (i.e., between baseline and ascending provocation, ascending and peak provocation, peak and descending provocation and descending and post-provocation were performed using a Bonferroni correction (thus p-values below are Bonferroni corrected). Analyses showed that for subjective anger, there were significant differences between baseline and ascending (t (75) = 3.00, p = .03, d = .69), ascending and peak provocation (t (75) = 6.56, p <.001, d = 1.51) and descending and post provocation (t (75) = 5.62, p < .001, d = 1.30). These findings indicate that the MTAP was effective in changing subjective anger across the task in a rising and falling pattern.

For aggression, there were significant differences between baseline and ascending (t (78) = 4.81, p < .001, d = 1.01), ascending and peak provocation (t (78) = 7.89, p <

.001, d = 1.78), peak provocation and descending (t (78) = 3.00, p = .03, d = .68) and descending and post provocation (t (78) = 8.86, p < .001, d = 2.00). For heart rate, there were significant differences between baseline and ascending (t (74) = 6.32, p < .001, d = 1.45), and peak provocation and descending (t (74) = 8.18, p < .001, d = 1.90). For GSR, there were significant differences between baseline and ascending (t (77) = 8.86, p < .001, d = 2.02), peak provocation and descending (t (77) = 8.47, p < .001, d = 1.93), and descending and post-provocation (t (77) = 8.19, p < .001, d = 1.87). For HF HRV there were significant differences between peak provocation and descending (t (77) = 3.81, p < .01, d = .87) and descending and post provocation (t (77) = 4.10, p < .01, d = .93). Overall, the findings indicate that the MTAP was effective in changing levels of aggression and physiological responding across the task. The pattern followed a characteristic rise and fall around the provocation.

Figure 6.

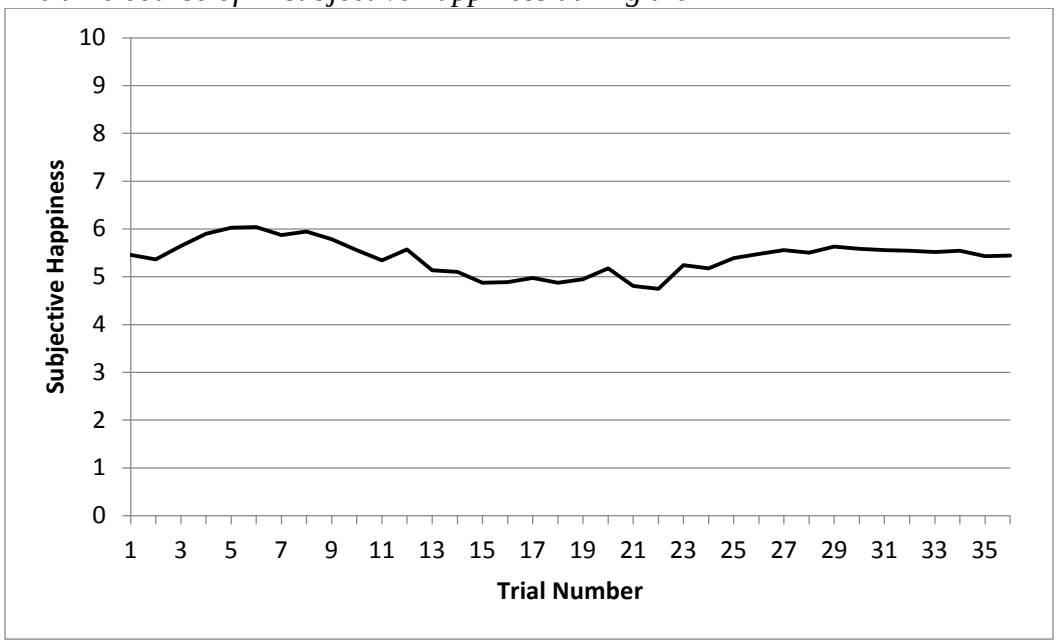
The time course of M subjective fear during the MTAP



Note. MTAP = Modified Taylor Aggression Paradigm

Figure 7.

The time course of M subjective happiness during the MTAP



Note. MTAP = Modified Taylor Aggression Paradigm

Table 5.

Means (SD) demographic and trait variables as a function of responder status (N = 79)

Variable	Responders	Non-responders
Subjective Anger (non-responders =35)		
Age	21.82 (3.69)	22.34 (5.41)
Female (%)	27 (61.4)	25 (71.4)
BPAQ Anger	13.00 (4.82)	12.83 (4.99)
BPAQ Aggression	29.98 (9.43)	27.77 (9.51
BPAQ Hostility	18.30 (5.76)	14.66 (5.54)**
DERS Total	72.39 (19.62)	69.31 (16.15)
Aggression (non-responders =19)		
Age	22.25 (4.68)	21.42 (3.96)
Female (%)	44 (73.3)	8 (42.1)*
BPAQ Anger	13.48 (5.12)	11.16 (3.47)
BPAQ Aggression	28.35 (9.17)	31.05 (10.33)
BPAQ Hostility	16.87 (6.38)	16.11 (4.18)
DERS Total	73.38 (18.47)	63.58 (15.05)*
HF HRV (non-responders = 24)		
Age	21.98 (4.25)	22.21 (5.15)
Female (%)	37 (67.3)	15 (62.5)
BPAQ Anger	12.18 (4.31)	14.63 (5.69)*
BPAQ Aggression	28.07 (8.74)	31.13 (10.88)
BPAQ Hostility	16.16 (5.94)	17.88 (5.83)
DERS Total	70.31 (18.30)	72.67 (17.95)

Note. BPAQ = Buss Perry Aggression Questionnaire, DERS = Deficits in Emotion Regulation Scale**,** HF HRV **=** High Frequency Heart Rate Variability
* = $p < .05$**, ** =** $p < .01$

Table 6.

Component variable means (SD) by provocation blocks

Component (n)	Baseline (Block 2)	Ascending (Block 3)	Peak (Blocks 4-5)	Descending (Block 6)	Post (Blocks 7-9)
Anger (76)	0.32 (0.86)	0.55 (1.01)	1.68 (2.14)	1.39 (1.88)	0.43 (0.74)
Aggression (79)	1.99 (3.06)	2.72 (2.92)	5.11 (3.43)	4.47 (3.13)	2.08 (2.82)
HR (75)	75.51 (11.53)	78.61 (11.76)	79.87 (12.03)	76.13 (10.89)	75.81 (9.92)
GSR (78)	2.63(2.64)	4.72(2.81)	5.20(2.91)	3.69(2.84)	2.17 (2.48)
HF HRV (75)	1495.01 (1502.98)	1131.99 (1205.32)	1304.54 (1700.65)	1554.70 (1771.33) 5)	1376. 24 (1728.52)

Note. Anger = Subjective anger, HR = heart rate, GSR = galvanic skin response, HF HRV= high-frequency heart rate variability. Means above represent untransformed data for ease of interpretation.

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