Anger Makes You Feel Stronger: The Positive Influence of Trait Anger in a Real-Life Experiment

Abstract Although anger as a negative emotion is associated with unpleasantness, recent research on anger highlights its motivational effect. The present study tested whether individuals experience both, an unpleasant and an activating affect, after real-life provocations. Results revealed that an anger situation evoked not only typical subjective and cardiovascular anger reactions but also a sense of strength, which is a positive affect. A comparison of participants with low versus high anger disposition according to the STAXI-2 at baseline, treatment, and recovery showed that participants with high trait anger consistently scored higher in subjective ratings of feeling strong than their counterparts did. Moreover, we found a larger and longer lasting effect of feeling strong than feeling angry after an anger treatment. Thus, differences in anger disposition influence the positive correlation between trait anger and positive affect.

Keywords: state anger, trait anger, affect, psychophysiology, recovery, STAXI-2

While adaptive aspects of anger have received little consideration (e.g. Frijda, 1986; Harmon-Jones, 2004; Litvak, Lerner, Tiedens, & Shonk, 2010; Tamir, Mitchell, & Gross, 2008), most studies of anger emphasize negative aspects of state and trait anger (for review, see Potegal, Stemmler, & Spielberger, 2010). Anger is strongly associated with negative terms like aggression and hostility, also known as the AHA! Syndrome (Spielberger, Johnson, Russell, Crane, Jacobs, & Worden, 1985). In this context, state anger is considered a well-known, although not sufficient or necessary, antecedent of aggressive behavior (Averill, 1982; Berkowitz, 1962; Tavris, 1989). Furthermore, anger disposition in particular is linked with cardiovascular heart diseases (Williams, Paton, Siegler, Eigenbrodt, Nieto, & Tyroler, 2000), stroke (Everson, Kaplan, Goldberg, Lakka, Sivenius, & Sallonen, 1999) and substance use (Awalt, Reilly, & Shopshire, 1997). Despite the common view of anger as a negative emotion, there is also empirical evidence showing a positive side of state and trait anger, such as instrumental (Frijda, 1986; Lerner & Keltner, 2001) and hedonical aspects (Harmon-Jones, 2004; Izard, 1991; Lazarus, 1991; Litvak et al., 2010; Smith & Ellsworth, 1985; Tamir et al., 2008) of anger. Accordingly, anger promotes the attainment of a goal, making subjects feel capable of altering the situation (Berkowitz & Harmon-Jones, 2004). Thus, the motivational component of anger can be emphasized as a positive and adaptive aspect.

The current study addresses adaptive aspects of state and trait anger. Recent research on anger focuses on the motivational direction of anger by investigating its association with positive activation (Carver & Harmon-Jones, 2009a; Harmon-Jones, Harmon-Jones, Abramson, & Peterson, 2009; Harmon-Jones, Vaughn-Scott, Mohr, Sigelman, & Harmon-Jones, 2004) as well as with approach-related affects (Carver, 2004; Carver & Harmon-Jones, 2009a; Harmon-Jones, 2003, 2004, 2007; Harmon-Jones & Allen, 1998; Harmon-Jones, Schmeichel, Mennitt, & Harmon-Jones, 2011; Harmon-Jones & Sigelman, 2001; Harmon-Jones, Sigelman, Bohlig, & Harmon-Jones, 2003; Watson, 2009). Likewise, this experiment is designed to investigate especially anger responses that are commonly experienced as positive affect. We argue that an anger-inducing situa-
tion evokes not only negative affect, but also positive affect (see also Harmon-Jones et al., 2009; Harmon-Jones et al., 2004). In addition to previous experimental studies in this context using neurophysiological correlates (Harmon-Jones, 2007; Harmon-Jones et al., 2004), we examine cardiovascular variables as physiological correlates of anger. Furthermore, this is the first experimental study in the research area of anger in association with positive activation, which comprises process analyses. Psychophysiological reactions are experimentally tested before, immediately after an anger evoking treatment, and at the end of a recovery phase. Finally, we investigate the role of anger disposition in anger responses related to positive affect. Sincetrait anger influences state anger (Spielberger, Jacobs, Russell, & Crane, 1983) and state anger after an anger treatment is associated with positive affect (Harmon-Jones et al., 2009; Harmon-Jones et al., 2004), it can be expected that trait anger is also linked with positive affect occurring after an anger-inducing situation.

According to approaches based on emotion specificity (Ekman, Friesen, & Ellsworth, 1982; Izard, 1977; Ortony & Turner, 1990; Plutchik, 1994), emotions are assumed to be elicited by distinctive antecedent events (Ekman, 1992, 1999). Appraisal models of emotions emphasize the cognitive appraisal of antecedents and assume that emotions are important to promote goals. Thus, anger is strongly associated with antecedents that are interpreted as externally caused obstructions to attain ones goal or violation of standards (e.g. Dollard, Doob, Miller, Mowrer, & Sears, 1939; Berkowitz, 1993; Frijda, 1986; Lazarus, 1991; Oatley & Johnson-Laird, 1987; Ortony, Clore, & Collins, 1988). Reviews on anger induction (Bongard & Wilke, 2008; Lobbestael, Arntz, & Wiers, 2008; Stemmler, 2010) recommend methods including personal contact that comprises one of the described antecedents of anger to ensure measurable anger responses. For this reason, we conducted an experiment in which anger was induced by real-life provocations representing violation of standards (see also Bongard, Pfeffer, al’Absi, Hodapp, & Linnenkemper, 1997; Everson, McKey, & Lovatto 1995; Suarez & Williams, 1989).

While cognitive models elaborated distinct patterns of cognitive appraisals for emotions (Lazarus, 1991; Scherer, 1997; Smith & Ellsworth, 1985), physiological approaches of differentiation were rather difficult. Especially the differentiation of anger and fear was of particular interest (e.g. Axt, 1952; Stemmler, Heldmann, Pauls, & Scherer, 2001). A meta-analysis conducted by Stemmler (2004) revealed significant differences in physiological correlates between anger and fear. Accordingly, a larger response in facial temperature, total peripheral resistance, muscle tension, and especially in diastolic blood pressure is characteristic of anger. Yet, there are also unspecific physiological reactions in line with the fight-or-flight hypothesis first postulated by Cannon (1929). Higher responses in systolic blood pressure and heart rate, for instance, are associated with both anger and fear (Stemmler, 2010). In order to assess anger not only on the subjective level, but also on the physiological level, we examined heart rate, systolic blood pressure, and especially diastolic blood pressure as cardiovascular correlates of anger responses.

Anger is commonly known as a negatively valenced emotion which is associated with unpleasantness (e.g. Berkowitz & Harmon-Jones, 2004). An important basic for distinguishing between positive and negative emotions are dimensional approaches (e.g., Russell, 1980; Watson & Tellegen, 1985; Larsen & Diener, 1992). Prominent circumplex models (e.g., Lang, 1995; Russell, 1980; Watson & Tellegen, 1985) organize emotions in two dimensions, valence (positive vs. negative) and arousal (low vs. high). Watson (2000) postulated a direct relationship between valence and motivational direction (approach vs. withdrawal), suggesting a link between positive affect and approach motivation as well as an association of negative affect with withdrawal motivation. Following investigations, however, demonstrated that negative affects such as anger are also considered an approach-related affect (Carver, 2004; Carver & Harmon-Jones, 2009a; Harmon-Jones, 2003; Harmon-Jones, 2004; Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001; Harmon-Jones et al., 2003; Watson, 2009). Though anger represents an unpleasant affect in circumplex models (Russell & Barrett, 1999; Tellegen, Watson, & Clark, 1999; Watson & Tellegen, 1985), it is associated, although moderately, with positive activation (Russell & Barrett, 1999) and engagement (Watson & Tellegen, 1985) unlike other negative affects such as sadness. Feeling strong, alert, determined, and active, in turn, are highly associated with positive activation (see e.g. Russel & Barrett, 1999; Watson & Tellegen, 1985). In comparison to highly pleasant emotions with a clear positive valence such as happiness, these feelings are quite neutral ones (Carver, 2004; Carver & Harmon-Jones, 2009b; Russel & Barrett, 1999). Therefore, the linking of anger with strength, determination, and alertness seems to be appropriate (Carver & Harmon-Jones, 2009a; Harmon-Jones et al., 2009; Harmon-Jones et al., 2004). In addition, there is empirical evidence based on qualitative analyses suggesting that angry people report feeling more energized (e.g. Frijda, Kuipers, & ter Schure, 1989; Shaver, Schwartz, Kirson, & O’Connor, 1987). Likewise, cognitive appraisal models postulate that anger arises in order to attain a blocked goal (Frijda, 1986). Thus, we argue that sense of strength, activeness, and determination also occur after an anger-inducing situation besides typical anger reactions comprising negative affect.

Spielberger and colleagues (1983) presented a model integrating the conceptualization of state and trait anger. Accordingly, individuals with high trait anger frequently experience angry feelings. Moreover, they experience angry feelings more intensely and show higher state anger levels when they are angry. For this reason, we assert that anger disposition influences the intensity of subjective and
physiological responses following an anger provocation. Specifically, individuals with high trait anger are expected to show higher subjective ratings for negative anger related items as well as for items associated with positive affect.

To extend this assumption, we investigate the duration of the effect, which is supposed to be influenced by anger disposition. Therefore, we assessed anger responses not only immediately after the anger treatment, but also after a recovery period of eight minutes (Bongard et al., 1997; Engebretson, Matthews, & Scheier, 1989; Everson et al., 1995; Suarez & Williams, 1989; Suchday, Carter, Ewart, Larkin, & Desiderato, 2004). Studies on anger dealing with recovery measures (Bodemann, Bodemann, & Perez, 1993; Hodapp, Bongard, Heinrichs, & Oltmanns, 1993) demonstrate that anger disposition prolongs self-reported anger feelings and cardiovascular recovery. Thus, this effect might also apply to self-reported positive affects after an anger-inducing situation.

The Current Investigation

The present study aimed to examine the link of positive affect with anger responses in a psychophysiological experiment. First, we assumed that real-life provocations in the anger condition evoke subjective and physiological anger responses (Bongard et al., 1997). In line with recent experimental findings (Harmon-Jones et al., 2009; Harmon-Jones et al., 2004), we further hypothesized on the subjective level that the anger-inducing situation will evoke not only negative anger-related affect (upset, hostile, irritable) but also positive affect (active, alert, determined, strong). Moreover, the recovery period after the real-life provocation was analyzed for typical anger responses, as well as for emotional responses with positive connotations, which has been a neglected aspect in research on anger up to now. For this purpose, we contrasted subjects with low versus high trait anger with regard to their emotional and physiological reaction during the entire experimental phase (baseline, treatment, and recovery). We argue that for both, treatment and recovery period, participants with high trait anger in comparison to participants with low trait anger show higher subjective ratings for anger related (upset, hostile, irritable) and positive affect items (active, alert, determined, strong). Thus, in the anger condition, trait anger should correlate positively with ratings of anger-related and positive-affect items not only in the treatment period but also in the recovery period.

Method

Participants

75 female undergraduates of the Goethe University Frankfurt am Main between the ages of 18 and 49 (M = 24.0; SD = 5.8) participated in this study. They were recruited by personal contact or by advertisement at the University. All data were collected by two female investigators. Male students were excluded because of confounding effects of gender between participants and investigators. Since cardiac responses to anger induction were assessed, further exclusion criteria concerned hypertension as well as use of cardioactive medication. Participants received sweets and took part in a lottery with shopping vouchers as compensation for participation.

Measures

Anamnesis questionnaire. Participants were given an anamnesis form in order to assess demographic, medical (e.g. coronary heart disease), and life-style variables(e.g. alcohol, smoking, caffeine consumption, sport activity). These data were used as control variables.

PANAS. The state scale of the German version (Krohne, Egloff, Kohlman, & Tausch, 1996) of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) was administered to assess the current affective state. It comprises 20 items assessing activated positive affect (PA: active, alert, attentive, determined, enthusiastic, excited, inspired, interested, proud, and strong) and negative affect (NA: afraid, ashamed, distressed, guilty, hostile, irritable, jittery, nervous, scared, and upset). Participants rated all items on a 5-point Likert-type scale ranging from 1 = not at all to 5 = extremely. Similar to a previous study conducted by Bongard and colleagues (1997), we considered three items of the NA Scale of the PANAS as anger-related items (upset, hostile, irritable). Internal consistency of PA reached a Cronbach’s alpha a = .85, for NA a = .73.

STAXI-2. The State-Trait Anger Expression Inventory-2 (STAXI-2; Spielberger, 1999) is a self-report measure of state and trait anger. Additionally, it provides four scales for assessing anger expression (Anger-Expression-In and Anger-Expression-Out) and anger control (Anger-Control-In and Anger-Control-Out). Dispositional anger was assessed with the Trait Anger Scale of the German version of the STAXI-2 (Rohrmann, Hodapp, Schnell, Tububos, Schwenkmezger, & Spielberger, in preparation). All items consist of 4-point Likert-type scales. Internal consistency of the Trait Anger Scale used in this study reached a Cronbach’s alpha = .91.

Physiological data. Heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) were recorded with an oscillometric blood pressure monitor (Elmed ASM 2000).

Procedure

Participants were randomly assigned to an anger versus control condition before entering the laboratory. The first investigator welcomed the participants providing them with a description of the study including a cover story (Participants were told that the study investigated the correlation of task performance and psychophysiological measurements. Therefore, they would have to perform a task dur-
ing psychophysiological variables.), which was necessary for successful anger provocation. Afterwards, they were asked to fill in the anamnesis questionnaire and to sign a consent form. Height and weight were also measured in order to calculate body mass index (BMI). Variables of the anamnesis form as well as the BMI were used as control variables. Finally, participants were asked to complete the STAXI-2. They were then seated in a comfortable recliner chair in the experimental room, which was a sound-proof cabin with a table in front and a window at the back. With the cabin-door closed, communication between investigator and participant was possible by using an intercom.

**Baseline period.** Participants were asked to relax while the investigator was testing the physiological data transfer. During the seven minute baseline period HR, SBP, and DBP were measured every two minutes. Afterwards, the investigator entered the sound-proof cabin and participants were asked to complete the PANAS to assess their affective state at the end of the baseline.

**Treatment period.** In line with the cover story, participants were administered to read aloud different numbers they were going to see on the monitor for six minutes. In order to increase the credibility of the task, all participants were told that they had been randomly assigned to the condition with an easy task. Depending on the experimental condition (control vs. anger) the following procedure was different: The anger provocation method was based on experiments conducted by Suarez and Williams (1989), Everson et al. (1995), and Bongard et al. (1997). Special care was taken not to induce fear in the treatment condition by criticizing participants’ lack of cooperation. In the anger condition, the investigator entered the cabin after a minute telling the participant that she had forgotten a very important appointment. Therefore, she would have to ask a colleague to proceed with the experiment. Two minutes later, a second investigator continued the experiment in an unfriendly manner. She asked the participant to summarize the instruction. Subsequently, the second investigator faked a phone call by activating the ring tone of her cell phone and interrupted the participant. A simulated 3-minute conversation followed in which she pretended to chat with a friend, predominantly consisting of small-talk. At the end of the call, the investigator irritably told her friend that she had to get off the phone because she would have to continue an experimental session for her colleague. Afterwards, she slammed the cabin-door and ordered the participant in a harsh and offensive manner. For the second and third provocation, the wording ran as follows: “You have to sit calmly, otherwise the recorded physiological data will be invalid!” and “What are you doing? You’re still moving! I would have thought that you would behave better!”.

Physiological data were measured three occasions (before and after the first provocation as well as after the last provocation). Subjective measures were assessed after six minutes. In the control condition, participants were able to perform the task without being disturbed by the investigator who behaved friendly in this condition. Time of measurement for self-report and physiological data was equivalent in both conditions.

**Recovery Period.** In this part physiological data was recorded four times in eight minutes at 90-seconds intervals. Subsequently, participants were asked to complete the PANAS. As expected, a comparison of the recovery period between the anger and control condition revealed different response patterns. No significant changes in self-reported anger as well as in physiological reactions were observed in the control condition corroborating the validity of the treatment responses in the anger condition.

After completion of the PANAS, participants were administered to fill in a questionnaire including items for manipulation check. Analyses of these items revealed that participants were not aware of the real intention of the experiment. In line with our expectations, participants in the anger induction condition felt badly treated by the second investigator. All participants were fully debriefed and thanked for their participation.

**Results**

**Comparability of groups.** Both groups differed neither in measured control variables nor in emotional state at baseline. There was only a significant difference in HR values. Participants in the anger condition showed lower HR levels than their counterparts in the control condition, $F(1, 60) = 7.15, p < .05, \eta^2_p = .11$. Therefore, we controlled for the following analyses the baseline values by using them as covariates.

**Emotion Induction.** For this purpose, multivariate GLMs including covariance analyses were performed using Hotelling’s Trace as criterion. Treatment values were entered as dependent variables while baseline scores of the independent variables were used as covariates. Two multivariate GLMs including covariance analyses were conducted for emotional state and one for physiological responses. A 2 (experimental condition: control vs. anger) x 2 (emotional state on scale level: Positive vs. Negative Affect) multivariate GLM revealed a main effect of the experimental condition, $F(2, 59) = 9.39, p < .001, \eta^2_p = .24$. With emotional state on item level as dependent variables, an approached significant main effect was obtained, $F(20, 38) = 1.78, p = .06, \eta^2_p = .48$. For physiological data, there was a significant main effect in the 2 (experimental condition: control vs. anger) x 3 (HR vs. SBP vs. DBP) multivariate GLM, $F(3, 58) = 13.58, p < .001, \eta^2_p = .41$.

Anger provocation was successful. As expected, there were significant changes for anger related items in the an-
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Process Analysis. Process analyses were conducted for subjective and physiological data in the anger provocation condition. The sample was divided into tertiles for trait anger in order to contrast the lowest (n = 17) with the highest tertile (n = 15). Univariate GLM repeated measures were used to test main effects of between-subjects factors and within-subjects factors, as well as interaction effects between factors. Self-report data were entered into separate 2 (trait anger: low, high) x 3 (time: baseline, treatment, recovery) univariate GLM repeated measures, with trait anger as between-subjects factor and time as within-subject fac-

Table 1
Means, Standard Deviations, and results of Simple Effect Tests for control vs. anger condition comparing subjective anger ratings and cardiovascular responses at treatment

<table>
<thead>
<tr>
<th>Treatment</th>
<th>control (n = 17)</th>
<th>anger (n = 45)</th>
<th>F</th>
<th>η²p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Emotional</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative Affect</td>
<td>1.19 (0.18)</td>
<td>1.65 (0.42)</td>
<td>19.48***</td>
<td>.25</td>
</tr>
<tr>
<td>Positive Affect</td>
<td>2.26 (0.72)</td>
<td>2.33 (0.62)</td>
<td>0.40</td>
<td>.00</td>
</tr>
<tr>
<td>Upset</td>
<td>1.41 (0.80)</td>
<td>2.02 (1.06)</td>
<td>3.78*</td>
<td>.06</td>
</tr>
<tr>
<td>Hostile</td>
<td>1.00 (0.00)</td>
<td>1.56 (0.87)</td>
<td>6.89*</td>
<td>.11</td>
</tr>
<tr>
<td>Irritable</td>
<td>1.00 (0.00)</td>
<td>2.29 (1.02)</td>
<td>13.32**</td>
<td>.19</td>
</tr>
<tr>
<td>Fear</td>
<td>1.00 (0.00)</td>
<td>1.02 (0.15)</td>
<td>0.38</td>
<td>.00</td>
</tr>
<tr>
<td>Active</td>
<td>2.29 (1.10)</td>
<td>2.36 (1.01)</td>
<td>0.02</td>
<td>.00</td>
</tr>
<tr>
<td>Alert</td>
<td>2.71 (0.92)</td>
<td>3.02 (0.81)</td>
<td>1.87</td>
<td>.03</td>
</tr>
<tr>
<td>Determined</td>
<td>2.18 (0.95)</td>
<td>2.45 (0.99)</td>
<td>1.20</td>
<td>.02</td>
</tr>
<tr>
<td>Strong</td>
<td>1.41 (0.71)</td>
<td>2.07 (1.03)</td>
<td>7.15*</td>
<td>.11</td>
</tr>
<tr>
<td><strong>Physiological</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>110.94 (11.71)</td>
<td>127.36 (9.14)</td>
<td>74.86***</td>
<td>.57</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>67.12 (11.34)</td>
<td>76.74 (8.03)</td>
<td>75.52***</td>
<td>.57</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>77.47 (8.13)</td>
<td>90.18 (11.08)</td>
<td>27.09***</td>
<td>.32</td>
</tr>
</tbody>
</table>

Note. Anger represents a scale comprising anger related PANAS items upset, hostile, and irritable. For physiological data, only the last measurement occasion at treatment was used.

SBP = systolic blood pressure.
DBP = diastolic blood pressure.
HR = heart rate.
mmHg = millimetre of mercury.
bpm = beats per minute.

p < .10. * p < .05. ** p < .01. *** p < .001.
tor. Similarly, SBP, DBP, and HR scores were analyzed in
three separate 2 (trait anger: low, high) x 8 (time: baseline,
treatment 1 to 3, recovery 1 to 4) univariate GLM repeated
measures.

Regarding physiological data, DBP reached signifi-
cance for the within-subject factor time, \( F(2.87, 86.30) = 73.81, p < .001, \eta_p^2 = .71 \), and for the interaction term time x trait anger, \( F(2.87, 86.30) = 3.03, p < .05, \eta_p^2 = .09 \). Only
a main effect of time was revealed for HR, \( F(2.60, 78.08) = 47.63, p < .001, \eta_p^2 = .61 \), as well as for SBP, \( F(2.36, 70.85) = 61.49, p < .001, \eta_p^2 = .67 \). There were no inter-
action effects for HR and SBP. None of the physiological
variables revealed a main effect of trait anger.

As displayed in Table 2, process analyses of self-report
data revealed significant main effects for time and trait
anger, as well as significant interaction effects for time x
trait anger. These results support the assumption that trait
anger influences subjective affect state after an anger treat-
ment. Mean results of anger related items (upset, hostile,
irritable) varied across the three measurement occasions,
as well as mean ratings of the PA item strong. Additionally,
participants with low and high trait anger differed in their
subjective ratings for these items, except for the item upset.

Figure 1 plots the processes for the anger related items up-
set, hostile, and irritable, as well as for the PA item strong
in the anger provocation condition. No process analyses
were conducted for the other PA items (active, alert, de-
determined), since there were no significant increases for the
respective items after anger provocation.

Subsequently, one-way ANOVAs were conducted to
analyze group differences for each measurement occasion.

<table>
<thead>
<tr>
<th></th>
<th>df /effect</th>
<th>df/ error</th>
<th>( F )</th>
<th>( \eta_p^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>upset( _{NA} )</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>time</td>
<td>1.45</td>
<td>43.37</td>
<td>5.47*</td>
<td>.15</td>
</tr>
<tr>
<td>trait anger</td>
<td>1</td>
<td>30</td>
<td>1.28</td>
<td>.04</td>
</tr>
<tr>
<td>time x trait anger</td>
<td>1.45</td>
<td>43.37</td>
<td>2.07</td>
<td>.07</td>
</tr>
<tr>
<td><strong>hostile( _{NA} )</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>time</td>
<td>1.47</td>
<td>43.96</td>
<td>18.17***</td>
<td>.38</td>
</tr>
<tr>
<td>trait anger</td>
<td>1</td>
<td>30</td>
<td>6.18*</td>
<td>.17</td>
</tr>
<tr>
<td>time x trait anger</td>
<td>1.47</td>
<td>43.96</td>
<td>5.64</td>
<td>.16</td>
</tr>
<tr>
<td><strong>irritable( _{NA} )</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>time</td>
<td>2</td>
<td>60</td>
<td>29.07***</td>
<td>.49</td>
</tr>
<tr>
<td>trait anger</td>
<td>1</td>
<td>30</td>
<td>10.24**</td>
<td>.25</td>
</tr>
<tr>
<td>time x trait anger</td>
<td>2</td>
<td>60</td>
<td>7.42**</td>
<td>.20</td>
</tr>
<tr>
<td><strong>strong( _{PA} )</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>time</td>
<td>2</td>
<td>60</td>
<td>4.21*</td>
<td>.12</td>
</tr>
<tr>
<td>trait anger</td>
<td>1</td>
<td>30</td>
<td>8.27**</td>
<td>.22</td>
</tr>
<tr>
<td>time x trait anger</td>
<td>2</td>
<td>60</td>
<td>5.52**</td>
<td>.16</td>
</tr>
</tbody>
</table>

Notes. NA = Negative Affect. PA = Positive Affect. * \( p < .05 \). ** \( p < .01 \). *** \( p < .001 \).
recovery period. However, low and high anger participants differed significantly only in their ratings in the treatment period. Yet for the PA item strong, high trait anger participants scored not only at treatment but also at recovery significantly higher than those with low trait anger levels (see Table 3).

Correlation analyses between trait anger and subjective affect measures (upset, hostile, irritable, strong) for participants in the anger provocation condition (n=45) showed moderate positive correlations for the items irritable and strong after the anger provocation. The PANAS item strong featured higher correlation coefficients than anger related items. Results are given in Table 4 for baseline, treatment, and recovery. They partially support the hypothesis that trait anger correlates positively with anger related NA items and PA items at treatment and recovery.

Table 3
Means, Standard Deviations, and Results of One-way ANOVAs comparing low vs. high trait anger group for feeling upset, hostile, irritable, and strong at three measurement occasions

<table>
<thead>
<tr>
<th>trait anger</th>
<th>baseline</th>
<th>treatment</th>
<th>recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M (SD)</td>
<td>F</td>
<td>M (SD)</td>
</tr>
<tr>
<td>Upset NA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>low</td>
<td>1.53</td>
<td>0.87</td>
<td>1.65</td>
</tr>
<tr>
<td>high</td>
<td>1.73</td>
<td>0.96</td>
<td>2.27</td>
</tr>
<tr>
<td>Hostile NA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>low</td>
<td>1.00</td>
<td>0.00</td>
<td>1.24</td>
</tr>
<tr>
<td>high</td>
<td>1.07</td>
<td>0.26</td>
<td>1.87</td>
</tr>
<tr>
<td>Irritable NA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>low</td>
<td>1.00</td>
<td>0.00</td>
<td>1.47</td>
</tr>
<tr>
<td>high</td>
<td>1.13</td>
<td>0.52</td>
<td>2.53</td>
</tr>
<tr>
<td>Strong PA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>low</td>
<td>1.53</td>
<td>0.80</td>
<td>1.47</td>
</tr>
<tr>
<td>high</td>
<td>1.53</td>
<td>0.99</td>
<td>2.60</td>
</tr>
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</table>

Note. NA = Negative Affect. PA = Positive Affect. + p < .10. * p < .05. ** p < .01. *** p < .001.
The current study supports recent findings suggesting a relationship between anger and positive affect (Carver & Harmon-Jones, 2009a; Harmon-Jones et al., 2009; Harmon-Jones et al., 2004). First, we demonstrated in a psychophysiological experiment that a typical anger evoking situation (see Suarez & Williams, 1989; Everson et al., 1995; Bongard et al., 1997) simultaneously leads to negative valenced, anger-related emotions (upset, hostile, and irritable) and to positive activation (strong). As expected, our findings are consistent with the view on anger as a negative approach-related emotion (Carver, 2004; Carver & Harmon-Jones, 2009a; Harmon-Jones, 2003, 2004; Harmon-Jones et al., 2009; Harmon-Jones & Allen, 1998; Harmon-Jones et al., 2011; Harmon-Jones & Sigelman, 2001; Harmon-Jones et al., 2003; Harmon-Jones et al., 2004; Watson, 2009). Accordingly, anger is associated with positive activation and both are related with approach motivation. Notably, instead of neurophysiological correlates (Harmon-Jones, 2007; Harmon-Jones et al., 2004), we used cardiovascular variables on the physiological level to ensure the assessment of state anger. Moreover, we can exclude that fear, which is sometimes confounded with anger reaction (for overview, see Stemmiller, 2010), was responsible for the increase in the PA item strong (cf. Harmon-Jones et al., 2009, p.192) as we took great care not to induce fear by the anger-induction method.

Second, this study is to our knowledge the first that tested positive activation on the subjective level not only immediately after an anger treatment, but also following a recovery period. In line with our hypothesis, we found a main effect of trait anger for self-report measures: Except for the NA item upset at recovery, participants with high trait anger showed higher self-report ratings for anger related NA items and the PA item strong at treatment and recovery. Surprisingly, results revealed a longer lasting effect of feeling strong in comparison to negative anger related feelings. As expected, correlation analyses between trait anger and subjective anger responses revealed a positive relationship. However, only correlations for the PA item “strong” and the NA item “irritable” reached significance.

In summary, these findings indicate that the motivating effect of anger is not only due to negative feelings (e.g. upset, hostile, irritable), but probably also or maybe only because of positive activation occurring after an anger-inducing situation. For the latter interpretation, support can be provided by a theoretical framework introduced by Carver (2004, p.16). He postulated a model of affects in which anger is always associated with activation and engagement. Accordingly, anger arises, if effort can improve progress. In case of lacking perspectives or no improvement despite continued effort, other negative affects such as sadness emerge. These feelings on the other hand are associated with deactivation.

Interestingly, research on emotion regulation (Tamir & Ford, 2011; Tamir et al., 2008) demonstrated that people prefer to feel angry in specific contexts. Especially confrontational situations, like games or negotiations, were of particular interest. In these studies, people were motivated to increase angry feelings the more they expected anger to be useful. We argue that these findings, which seem to be counter-intuitive at first sight, can be explained by the positive activation accompanying negative angry feelings.

A crucial point of our study, mostly neglected in previous research in this context, is the role of individual differences. It is remarkable that sense of strength applies exclusively for participants with high anger disposition, which indicates differential effects. Whereas negative angry feelings decreased to the same level for both groups, participants with high trait anger still reported an elevated sense of strength over eight minutes after the treatment episode. In contrast, descriptive statistics indicated that subjective ratings of feeling strong slightly decreased for participants with low trait anger. Therefore it can be concluded that individuals with high trait anger benefit from their anger disposition. Individual differences in anger disposition seem to be an important factor for the explanation of the relationship between anger and positive affect.

<table>
<thead>
<tr>
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<th>strong&lt;sub&gt;PA&lt;/sub&gt;</th>
<th>irritable&lt;sub&gt;NA&lt;/sub&gt;</th>
<th>hostile&lt;sub&gt;NA&lt;/sub&gt;</th>
<th>upset&lt;sub&gt;NA&lt;/sub&gt;</th>
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<td>.40**</td>
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<td>recovery</td>
<td>.30*</td>
<td>.14</td>
<td>.11</td>
<td>.03</td>
</tr>
</tbody>
</table>

Note: NA = Negative Affect. PA = Positive Affect. All correlations were tested two-tailed. *p < .05. **p < .01.
Why do only subjects with high trait anger feel stronger when they get angry? It is conceivable that underlying cognitive mechanisms are responsible for this effect. There is evidence that aggressive cognitions are positively correlated with trait anger (e.g., Bond, Ruaro, & Wingrove, 2006). Aggression is considered as a drive and as an energy source respectively (e.g., Baron & Richardson, 2004; Berkowitz, 1989; Tedeschi, Smith, & Brown, 1974). Thus, the positive relationship between trait anger and sense of strength might be mediated by aggressive thoughts. Another potential mediator might be the optimistic cognitions of subjects with high trait anger. For instance, Lerner and Keltner (2001) demonstrated that subjects with high dispositional anger perceived higher controllability and certainty in contrast to fearful individuals. Similarly, trait assertiveness seems to be associated with trait anger (Buss & Perry, 1992; Doyle & Biaggio, 1981). It could be assumed that individuals who are dispositionally high in anger have gained reinforcing experiences by experiencing anger (e.g., Sinaceur & Diedens, 2006; Tamir & Ford, 2011; Van Kleef, De Dreu, & Manstead, 2004) due to their proneness to have angry feelings more frequently and more intensely. Therefore, they might associate angry feelings with the sense of strength. Future research should address cognitive mechanisms to gain a better understanding of the observed effect.

Given that the sample comprised only female undergraduates, it would be interesting to test whether similar patterns will occur in a male or a non-student-centered sample. Further, we believe that an anger treatment with concrete relevance and more far-reaching consequences for the target person than in our experiment will evoke even higher positive activation for those with high anger disposition.

Our findings bear the implication for future research to include dispositional variables when analyzing the relationship of anger with positive affect. Experiencing anger as pleasant (Litvak et al., 2010) and energizing (Frijda et al., 1989; Shaver et al., 1987) might be largely influenced by trait anger (Harmon-Jones, 2004).

References


Tamar, M., & Ford, B. Q. (2011). When feeling bad is expected to be good: Emotion regulation and outcome expectancies in social conflicts. *Emotion Advance online publication. doi: 10.1037/a0024443*


