Hormones and Behavior 57 (2010) 276–283

Contents lists available at ScienceDirect

Hormones and Behavior

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

What happens when we get angry? Hormonal, cardiovascular and asymmetrical brain responses

Neus Herrero [a,*], Marien Gadea [b], Gabriel Rodríguez-Alarcón [b], Raúl Espert [b], Alicia Salvador [b]

[a] Unitat de Psiquiatria i Psicologia Mèdica, Facultat de Medicina, Universitat de València, CIBERSAM, Avda. Blasco Ibáñez 15, E. 46010 València, Spain
[b] Departament de Psicobiologia, Facultat de Psicologia, Universitat de València, Avda. Blasco Ibáñez 21, E. 46010 València, Spain

ARTICLE INFO

Article history:
Received 30 July 2009
Revised 15 December 2009
Accepted 15 December 2009
Available online 4 January 2010

Keywords:
Anger mood
Salivary testosterone
Salivary cortisol
Heart rate
Blood pressure
Dichotic listening
Asymmetrical brain activity

ABSTRACT

This study aimed to evaluate neuroendocrine and cardiovascular responses together with changes in brain asymmetry following an anger mood induction laboratory task. Previous research has shown an increase in heart rate and blood pressure when anger is experienced. Increased testosterone and decreased cortisol in response to anger and aggressive behavior have also been reported. With regard to asymmetrical frontal brain activity and emotion, the valence model links negative affect (as anger) to the right hemisphere while the motivational direction model links approach-related emotions (as anger) to the left hemisphere. From the subjective perception and from the neuroendocrine and cardiovascular response of the subjects, we can conclude that the self-referent statement anger induction method by Engebretson et al. (1999) was able to generate an experience of an anger affect in 30 healthy men. Another question was to analyze the consequences of that experience upon perceptual asymmetry when measured with a non-emotional laterality task. Regarding dichotic listening, an enhanced REA (right ear advantage) was observed after anger which indicates greater left hemisphere activity, supporting the motivational direction model.

© 2009 Elsevier Inc. All rights reserved.

Introduction

Research has consistently indicated that the induction of several emotions generates profound changes in the autonomic nervous system (ANS) activity – which controls cardiovascular reactivity (for a review, see Bradley and Lang, 2000) – and in the endocrine system activity (for a review, see De Kloet, 2004). In addition, it has also been found that the expression and the experience of emotions produce significant changes in brain activity, particularly within the frontal and temporal lobes (Robinson, 1995). Indeed, despite that central nervous system (CNS) activation may mediate associations between emotion and peripheral physiological responses, such as heart rate (HR), blood pressure (BP) and/or hormonal release (Foster and Harrison, 2004; Ohira et al., 2006), it is unusual for each of these parameters to be examined in conjunction in a single investigation.

Focusing on ANS, the notion of an emotion-specific autonomic response has been the source of much controversy. Two major theoretical positions have emerged regarding this issue. The first position embraces Cannon's view of undifferentiated autonomic arousal during emotion states (e.g. Cannon, 1929; Cacioppo et al., 2000). The second position, consistent with the theoretical writings of Darwin (1872/1965) and James (1884), argues that different emotions are accompanied by unique patterns of physiological activity, and gives evidence supporting discrete emotion-specific autonomic activity (Ax, 1953; Christie and Friedman, 2004; Ekman et al., 1983; Rainville et al., 2006; Schwartz et al., 1981; Sinha et al., 1992).

One of the most consistent findings in research on emotion-specific ANS activity has been the differential increases in cardiovascular reactivity during fear and anger. Fear response is similar to the general arousal pattern seen during isotonic exercise. HR, systolic blood pressure (SBP), and muscular blood flow increase, while net peripheral vascular resistance decreases keeping diastolic blood pressure (DBP) relatively low. Anger, on the other hand, produces increases on HR and SBP as well as peripheral vascular resistance, thus differentially elevating DBP (Ax, 1953; Christie and Friedman, 2004; Ekman et al., 1983; Schwartz et al., 1981; Sinha et al., 1992).

It is well known that the steroid hormone testosterone (T) is involved in aggressive behavior and dominance in numerous species. However, the relationship between T and aggression in humans has remained controversial (for a review see Archer, 2006). Research in human social behavior indicates that greater T levels are associated with anger (Hohlagschwandtner et al., 2001; Persky et al., 1971; Thompson and George, 2003; Van Honk et al., 1999, 2000; Wirth and Schultheiss, 2007), aggression (Archer, 2006); and especially with interpersonal dominance, including competition (Mazur and Booth, 1998; Salvador, 2005; Salvador and Costa, 2009; Salvador et al., 2003), aggressive dominance (Gerra et al., 1996; Gray et al., 1991; Van der Meij et al., 2008), and criminal violence (Dabbs et al., 1995; Van Bokhoven et al., 2006). All of these behaviors are related to approach

* Corresponding author. Unitat de Psiquiatria i Psicologia Mèdica, Departament Medicina, Facultat de Medicina (Universitat de València), Avda. Blasco Ibañez 15, E. 46010, València, Spain. Fax: +34 96 3864767.
E-mail address: neus.herrero@uv.es (N. Herrero).

0018-506X/$ – see front matter © 2009 Elsevier Inc. All rights reserved.
doi:10.1016/j.yhbeh.2009.12.008

motivation. Accordingly, hormonal treatments that increase T levels have been shown to increase anger, hostility, irritability, aggression and/or euphoria (e.g., O'Connor et al., 2004; Pope et al., 2000; Su et al., 1993; Van Honk and Schutter, 2007).

On the other hand, cortisol (C), a marker of activation of the hypothalamic–pituitary–adrenal (HPA) axis in stressful situations, is positively related to many of the negative emotions: e.g. fear, sadness and stress (Gadea et al., 2005; Lerner et al., 2007; Lewis and Ramsay, 2005; Van Eck et al., 1996; Van Honk et al., 2003), and with avoidance behavior (e.g. Buss et al., 2003; Roelofs et al., 2005). However, the relationship of anger or aggressive behavior with C is not as clear as with T. While many authors have found an association between lower C levels and anger, aggressive and approach behavior (Lerner et al., 2007; Roelofs et al., 2005; Roy, 2004; Van Goozen et al., 1998; Van Honk et al., 2003), others have found the opposite, high C levels related to anger (Putman et al., 2007; Van Honk et al., 2000) and competitive aggression (Salvador, 2005; Salvador et al., 2003); and some studies have found no relation between C and anger (Lewis and Ramsay, 2005; Van Eck et al., 1996). Furthermore, using primate models to understand human aggression, Kalin (1999) distinguishes 2 chief categories of aggression: defensive and offensive, with different hormonal patterns and neural mechanisms. While offensive aggression is approach motivated and associated with high levels of T and lower levels of C, defensive aggression is fear motivated and related to high plasma C concentrations and extreme asymmetric right frontal activity (discussed below).

Focusing on CNS, specifically in asymmetrical frontal brain activity related to emotions, there are two main conceptual models. The first view (valence model) has posited that the left frontal brain region is involved in the experience and expression of positive emotions and that the right frontal brain region is involved in the expression and experience of negative emotions (e.g. Ahern and Schwartz, 1985; Silberman and Weingartner, 1986). The second view (motivational direction model) has posited that the left frontal brain region is involved in the experience and expression of approach-related emotions and that the right frontal brain region is involved in the experience and expression of withdrawal-related emotions (e.g. Fox, 1991; Harmon-Jones et al., 2006; Sutton and Davidson, 1997).

Positive emotions are usually associated with approach-related motivation (e.g. happiness), whereas negative emotions are associated with withdrawal-related motivation (e.g. sadness and fear); but not all emotions behave in accord with this presumed relationship. Anger is one of the best examples of a violation of the relationship, because anger is experienced as negative (Harmon-Jones, 2004a; Lazarus, 1991; Watson et al., 1999), but it often evokes approach motivation (Adams et al., 2006; Berkowitz, 1999; Carver, 2004; Darwin, 1872/1965; Harmon-Jones, 2004b).

In examinations of anger and asymmetrical cortical activity, several electroencephalographic (EEG) studies have shown that trait anger relates to relatively greater left frontal activity when measured at resting baseline (Harmon-Jones, 2004a; Harmon-Jones and Allen, 1998; Rybak et al., 2006; Stewart et al., 2008), and also after anger-eliciting pictorial stimuli (Harmon-Jones, 2007). Experiments have also revealed that manipulated increases of left frontal cortical activity via repetitive transcranial magnetic stimulation augment vigilant attention towards and memory for angry facial expressions (D'Alfonso et al., 2000; Van Honk and Schutter, 2006). Moreover, some other studies using Positron Emission Tomography (PET) (Dougherty et al., 1999; Marci et al., 2007) or EEG (Aftanas et al., 2006; Harmon-Jones and Sigelman, 2001) have shown that the induction of an anger mood state evokes relatively greater left frontal brain activity, specially when this anger state is approach-related (Harmon-Jones et al., 2006; Wacker et al., 2003).

Finally, some studies have explored the cognitive-behavioral outcome of an induced affect when taking into account the lateralization of the task evaluated. Bartolic et al. (1999) found that dysphoria, generated by means of the Velten Mood Induction Procedure (VMIP) (Velten, 1968) yielded better figural (right hemisphere task) than verbal (left hemisphere task) fluency outcomes. Gadea et al. (2005) showed an increase in left ear (LE) items and a decrease in right ear (RE) items reported in dichotic listening following a sadness mood induction using also the VMIP. The above effects could be explained in terms of Kinsbourne's (1970) model of attention and perceptual asymmetry, with the induced negative affect (sadness or fear) increasing right brain activation, which in turn facilitated right hemisphere tasks and caused an attentional bias to the left hemi-space. This effect could also be explained by Hugdahl (2003) model of attention. From a top-down (instruction-driven information processing) perspective, a bottom-up (stimulus-driven information processing) asymmetry effect could be modulated or switched through cognitive means (e.g. an emotional induction).

In the present study the anger induction (AI) method by Engebretson et al. (1999) was applied to induce anger in healthy men. According with above mentioned studies, we expected increases in HR, SBP and DBP after the anger induction. We hypothesized an increase in T levels and a decrease in C levels too. A consonant–vowel dichotic listening (DL) test was also applied before and after the anger induction. On the basis of past research about anger mood and asymmetrical brain activity (for a review see Harmon-Jones, 2004b), we also predicted that the anger induction would increase left hemisphere activity (motivational model) so a facilitation of RE performance on the DL task would occur.

Method

Participants

Thirty right-handed men, undergraduate volunteers between 18 and 30 years old (mean age: 22.93, S.D.: 2.68) were recruited from the University community. Participants had no self-reported history of a major depression or other psychiatric disorders, medical illness, chronic pharmacological treatment or drug consumption. If they were smokers, their tobacco consumption was limited to five or less cigarettes a day. In order not to affect T measurements, none of them were top-class gymnasts, gym addicted or anabolic–androgenic steroid users. None of them had previous knowledge of the DL technique. All subjects were treated in accordance with "Ethical Principles of Psychologists and Code of Conduct" (American Psychological Association, 1992). The study was also approved by the local ethics committee. Hearing acuity was determined by a Lafayette 15014 C screening audiometer. Subjects included were those with no imbalance in hearing levels of more than 10 dB (at the frequencies of 500, 1000, 2000, 3000 and 6000 Hz).

Procedure

The subjects were told not to eat, drink, smoke or brush their teeth for 1 h prior to testing. On arrival at the laboratory, all subjects were informed that they would be providing saliva for hormonal analyses, and doing some behavioral tasks in a sound-attenuated room in our laboratory. After obtaining written informed consent, the subjects filled in a general information questionnaire and a handedness questionnaire (Olfield, 1971), and were tested for hearing acuity. Then, we started collecting baseline measures (dependent variables). The timing of the protocol was as follows: 3 measures of BP and HR (6 min), PANAS and Anger Subscale (POMS) questionnaires (5 min), collection of saliva sample directly from mouth to tube — Unitek R (5 min), and performance of the DL test (15 min). A short break of 10 min is the maximum. After the break the anger induction lab task was carried out for 20 min (independent variable). Immediately after the mood induction procedure all dependent variables (post-anger measures) were collected again. At the end of the experiment subjects were told

about the purpose of the study and thanked for participation. All sessions were carried out in the afternoon, between 1600 to 1800 h in order to control circadian rhythms and over a 2-month period.

Materials

Anger induction procedure: AI, Engebretson et al. (1999)

A modified Spanish translation of the anger induction (AI) (Engebretson et al., 1999) was used, with the aim of generating an anger affect experience. This AI laboratory task is similar in format to the Velten Mood Induction Procedure for depression (VMIP-D) and elation (VMIP-E) (Velten, 1968) and involves reading descriptors of anger experience, recalling relevant personal memories, and evoking the mood suggested by the sentence/memory. The AI exhibited good sensitivity and specificity in that it induced moderate to greater increases in anger (>1 SD change) in 68% of the sample (Engebretson et al., 1999).

The procedure designed to induce an anger affect consisted of 50 self-referent statements gradually progressing from relative mood neutrality ("Today is no different from any other day") to extremely angry ("I can feel my body getting tense with anger", "I feel like striking out at someone who has angered me", "I am consumed with hatred") connoting irritability, hostility, rage and anger.

The subjects were given a loose-leaf binder each page of which contained one of 50 anger statements. The instructions were to read each sentence silently, imagine what the sentence is saying, recall any relevant memories, and generally try as much as possible to get into the mood suggested by the sentence. After 20 s the experimenter continued with the next sentence.

Mood scales

PANAS scales. A Spanish translation (Sandín et al., 1999) of the short PANAS (Positive and Negative Affect Schedule) scales (Watson et al., 1988) was used to assess self-reported mood. The short PANAS consists of two 10-item mood scales that comprise positive (PA) and negative (NA) affect states. High PA reflects a state of high energy, full concentration and pleasurable engagement, whereas high NA subsumes a variety of aversive mood states including upset, guilt, fear, anger and nervousness. The PANAS was rated on a 5-point scale (from *not at all* to *very much*) to document the extent to which the subject experienced each mood state immediately after the anger induction. Psychometric properties of the PANAS scales have shown to have internal consistency, low correlation, and stability over time (Watson et al., 1988).

Anger–Hostility subscale of the P.O.M.S. The P.O.M.S. (Profile of Mood States) inventory consists of 58 items (adjectives) that describe feelings and mood. There are six subscales: Tension–Anxiety; Depression–Dejection; Anger–Hostility; Vigor–Activity; Fatigue–Inertia; and Confusion–Bewilderment (McNair et al., 1971). A Spanish translation (Balaguer et al., 1993) of the Anger–Hostility subscale of the P.O.M.S. was used to assess self-reported anger. This subscale consists of 12 adjectives (angry, grouchy, spiteful, annoyed, ready to fight, furious…) related to an anger and hostility affect state, and was rated on a 5-point scale (from 0 = *not at all* to 4 = *extremely*) to document the extent to which the subject experienced subjective anger immediately after the anger induction.

Cardiovascular response

The analyzed cardiovascular measures were SBP, DBP and HR. These physiological measures represent general cardiovascular indices that are most commonly measured in other studies about emotions. Participants had 3 baseline and 3 post-anger mood induction BP and HR measurements taken by a Dinamap Procare 100 Vital Signs Monitor (Critikon, Tampa, FL) which uses the oscillometric principle for pressure determinations; it has digital displays (thus reducing observer error) and an electrical pump. For all the measures, the BP cuff was placed on the non-dominant arm. Baseline SBP, DBP, and HR were each calculated as an average of the 3 readings, and the same for post-anger mood cardiovascular measures.

Hormonal determinations: salivary testosterone (T) and cortisol (C)

Salivary samples were centrifuged (5000 rpm, 15 ± 2 °C) and frozen at −20 °C at the end of each experimental session until determination by an experienced radioimmunoassay (RIA) technician (Central Research Unit, Faculty of Medicine, University of Valencia, Spain) who was unaware of the hypotheses being tested.

All samples from a given subject were run in duplicate in the same assay. The salivary T assay required a previous extraction phase with ether. Afterwards, ^{125}I-testosterone tracer was added and decanted into a coated tube with a highly specific antibody provided by a commercial kit (Immunotech SA, Marsella, France). Samples were counted by gamma counter for 1 min. Duplicate internal and external control tubes were routinely included. Intra-assay variation coefficient was lower than 5% and sensitivity was below 6 pmol/l. T levels were expressed in pmol/l.

Salivary C was determined by a commercial kit adapted to salivary levels after dilution of the standard curve in the buffer, as recommended in the protocol (Orion Diagnostica, Espoo, Finland). The salivary sample (150 µl) was mixed with ^{125}I-cortisol tracer and the tube coated with highly specific antibody. Finally, samples were decanted and counted for 1 min. C levels were expressed in nmol/l and internal and external controls were included in the assays. Good precision was obtained with intra-assay variation coefficients below 5% with a sensitivity of 1 nmol/l. More details about hormonal determination have been previously described elsewhere (Salvador et al., 2003).

Perceptual asymmetry: dichotic listening (DL) test

The dichotic stimuli consisted of the six stop consonants paired with the vowel /a/ to form six consonant–vowel syllables (ba, da, ga, ka, pa, ta). The syllables were paired with each other in all possible combinations to form 36 different syllable pairs. From these, the homonymic pairs (ba-ba, etc.) were included in the test as a perceptual control, but they were not considered in the statistical analyses. The other 30 syllables were duplicated and recorded randomly; giving 60 test trials, with a maximum correct score of 60. The DL test used in this study has achieved a test–retest reliability of 0.86 (for details and further descriptions of the DL test, see Gadea et al., 2003). The DL test was replayed to the subjects from a Sony Walkman D-EJ985 portable CD player with Technics rp-DJ1210 stereo covered outer ear headphones. The output from the CD player was calibrated at a level of 75 dB.

The subjects were informed that different syllables would be presented to each ear simultaneously and were asked to report only the syllable perceived most clearly. The data were acquired as the number of correctly reported items from the right (RE) and left (LE) ear. In addition, a laterality index (LI) score was calculated for each subject and condition (before and after anger induction), according to the formula: $LI = [(RE - LE) / (RE + LE)]$.

Statistical analyses

All variables were normally distributed (Kolmogorov–Smirnov test>0.05). A Student's *t* test for related variables was applied to all the variables (PA scale, NA scale, Anger subscale, HR, SBP, DBP, T, C, and LI) except DL raw scores to see differences between pre and post-anger induction (AI). An Analysis of Variance (ANOVA) 2×2 (Ear× Moment) with repeated measures and a subsequent Student's *t* test for related variables was applied to DL direct scores. Pearson correlation coefficients were also performed for post AI measures, searching for possible relations between hormones (T and C) and the

other variables. All statistical analyses were performed on a PC, using the SPSS 15 statistical package set. Data are presented as means and standard deviations and confidence intervals for the 95% of the mean are presented when appropriate.

Results

Mood scale scores

As can be seen in Table 1, PANAS positive scores diminished significantly from baseline to post-anger induction (t (29) = 5.38, p<0.001; confidence intervals for baseline versus post-anger induction were 33.2/36.4 and 26.7/31.38, respectively) while PANAS negative scores increased significantly (t (29) = −7.60, p<0.001; confidence intervals for baseline versus post-anger induction were 12.4/14.4 and 19.3/24.3, respectively). These results were seen despite PA being higher than NA for both conditions.

Table 1 shows that Anger–Hostility subscale scores increased significantly from baseline to post-anger induction (t (29) = −8.64, p<0.001; confidence intervals for baseline versus post-anger induction were 4.5/6.5 and 15.6/23.1, respectively).

Cardiovascular response

With regard to HR levels (see Table 1), there was a significant increase from baseline to post-anger induction (t (29) = −2.18, p<0.03; confidence intervals for baseline versus post-anger induction were 67.7/75.7 and 70/79.8 bpm, respectively).

Regarding BP, there were no differences in SBP levels between baseline and post-anger induction (t (29) = −1.37, p = 0.18), as shown in Table 1. On the contrary, DBP levels increased significantly (see Table 1) after the anger induction (t (29) = −2.72, p<0.01; confidence intervals for baseline versus post-anger induction were 68.2/72.5 and 69.7/75.5 mm Hg, respectively).

Testosterone and cortisol levels

Changes in T levels are shown in Fig. 1. There was an increase in T levels (t (29) = −2.492, p<0.01; confidence intervals for baseline versus post-anger induction were 79.7/95 and 90/102.2 pmol/l, respectively) from baseline to post-anger induction. On the other hand, C levels (see Fig. 2) decreased significantly (t (29) = 3.604, p<0.001; confidence intervals for baseline versus post-anger induction were 5.6/10.3 and 3.8/5.4 nmol/l, respectively) after the anger induction.

Table 1
Mean and standard deviation for all the dependent variables before and after the anger induction and t test for related variables p value.

	Baseline	Anger induction	p
	Mean (SD)	Mean (SD)	
PANAS			
Positive	34.83 (4.22)	29.07 (6.19)	<0.001
Negative	13.43 (2.71)	21.83 (6.77)	<0.001
POMS			
Anger subscale	5.53 (2.80)	19.37 (10.09)	<0.001
Cardiovascular reactivity			
HR	71.73 (10.73)	74.93 (13.08)	<0.03
SBP	123.60 (10.03)	125.13 (11.18)	n.s.
DBP	70.37 (5.78)	72.63 (7.74)	<0.01
Dichotic listening test			
RE items	32.17 (5.90)	34.57 (5.86)	<0.001
LE items	17.50 (4.42)	16.90 (4.69)	n.s.
LI (REA)	0.29 (0.18)	0.34 (0.18)	<0.04

Note. PANAS (Positive and Negative Affect Schedule), POMS (Profile of Mood States), HR (heart rate), SBP (systolic blood pressure), DBP (diastolic blood pressure), RE (right ear), LE (left ear), LI (Laterality Index), and REA (right ear advantage).

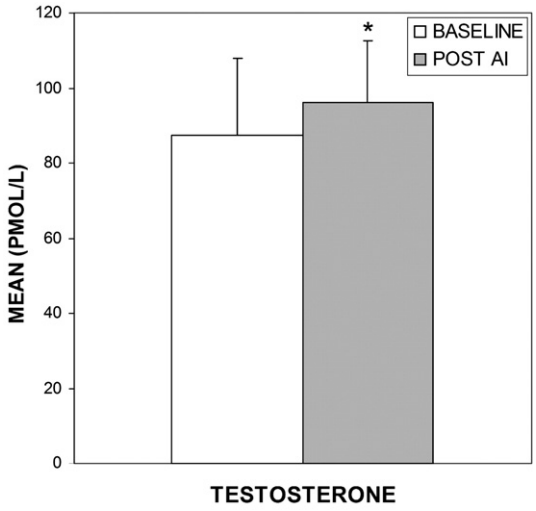

Fig. 1. Increased salivary testosterone levels after anger induction. Bars depict the mean +/− standard deviation of salivary testosterone (pmol/l) measured in 30 men before (empty bar) and after (grey bar) anger induction. * = p<0.01, Student's t test for related variables.

Dichotic listening

An ANOVA (2 × 2) for repeated measures was carried out with the variables Ear (RE versus LE) and Moment (baseline versus post-anger induction). Mean correct responses for both ears are displayed in Fig. 3. There was a significant main effect of Ear (F (1, 29) = 92.54, p<0.001), indicating a right ear advantage (REA) in both conditions, before and after the anger induction; and a significant main effect of Moment (F (1, 29) = 11.80; p<0.002), indicating a higher number of correct syllables after the anger induction in both ears (see Fig. 3). The interaction of Ear × Moment was significant (F (1, 29) = 6.76, p<0.01). As can be seen in Table 1, the analysis post hoc revealed a significant increase in RE items from baseline to post-anger induction (t (29) = −3.79, p<0.001; confidence intervals for baseline versus post-anger induction were 30/34.3 and 32.3/36.7, respectively), whereas there were not differences in LE items (t (29) = 0.944, p = 0.353).

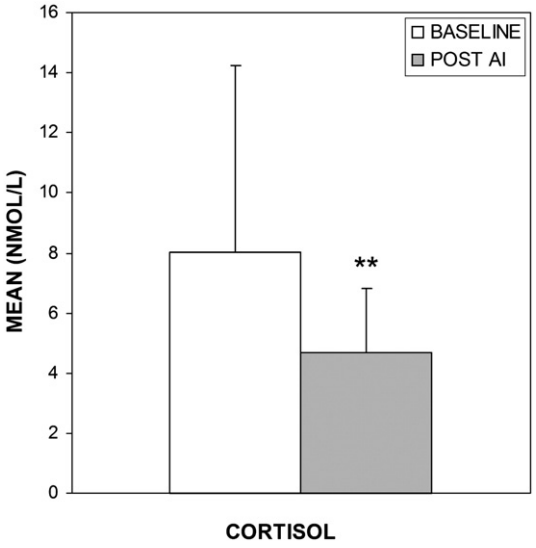

Fig. 2. Decreased salivary cortisol levels after anger induction. Bars depict the mean +/− standard deviation of salivary cortisol (nmol/l) measured in 30 men before (empty bar) and after (grey bar) anger induction. ** = p<0.001, Student's t test for related variables.

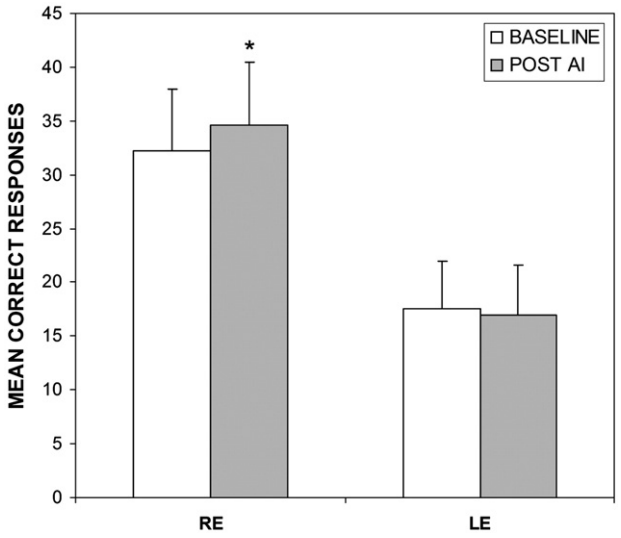

Fig. 3. Increased right ear correct responses after anger induction. Bars depict the mean +/− standard deviation of correct responses for both ears (RE: right ear, LE: left ear) obtained in 30 men for the DL test, before (empty bars) and after (grey bars) anger induction. * = $p<0.01$, ANOVA (2×2) for repeated measures.

Regarding LI, there was a significant increase in REA (t (29) = −2.118; $p<0.04$; confidence intervals for baseline versus post-anger induction were 0.225/0.358 and 0.274/0.409, respectively) after the anger mood induction (see Table 1).

Correlational analysis

Several correlations for post AI measures were observed. Regarding mood scales, we found an inverse association ($r=-0.47$; $p<0.009$) between PANAS positive and negative scores. A strong correlation ($r=0.80$; $p<0.001$) between anger subscale scores and PANAS negative scores was also seen. The more negative state you feel the angrier you get.

With regard to cardiovascular measures, both systolic and diastolic BP levels were positively correlated ($r=0.57$; $p<0.001$), and an interesting inverse correlation ($r=-0.52$; $p<0.03$) was found between post-anger DBP and C levels. In other words, participants who showed a decrease in C levels increased their DBP levels after anger induction.

Strong correlations between post AI dichotic listening measures (RE and LE raw scores and REA) were also observed. On the one hand, there was a positive association ($r=0.87$; $p<0.001$) between RE and REA and, on the other hand, LE was inversely related to both RE raw scores ($r=-0.72$; $p<0.001$) and REA ($r=-0.96$; $p<0.001$).

Discussion

The main finding of this study was that an anger induction procedure, applied to healthy right-handed men, elicited profound changes in different psychobiological parameters. We found greater self-reported anger mood, an increase in cardiovascular reactivity, higher salivary T levels, lower salivary C levels and an increase in RE items reported in DL together with an enhanced REA which suggested greater left asymmetrical brain activity. Several correlations for post-anger induction measures were also reported.

The analysis of scores on mood scales, which were used to assess self-reported mood, showed, on the one hand, an increase in Anger–Hostility subscale scores which means that participants were subjectively angrier after the mood induction. On the other hand, there was a decrease in PA scores as well as an increase in NA scores. This is congruent with previous research considering anger as a negative emotion (Harmon-Jones, 2004a; Lazarus, 1991; Watson et al., 1999). Correlational analyses were also in accordance with the literature. We found an inverse association between positive and negative mood scores after the anger induction procedure together with a strong correlation between Anger scale and negative mood. Participants who got angrier felt a more negative state immediately after the anger induction.

Regarding emotion-specific peripheral physiological responses, we focus on cardiovascular and hormonal reactivity when anger is experienced. Several studies have consistently found that anger evokes significant increases in HR and BP (e.g. Ax, 1953; Foster and Harrison, 2004; Prkachin et al., 2001; Rainville et al., 2006; Schwartz et al., 1981; Sinha et al., 1992; Weerts and Roberts, 1976). In our study, there was an increase in HR as well as in DBP. Also, SBP and DBP were positively related. Subjects who responded to the anger induction with high SBP levels showed increases in DBP. This effect of anger is different from the effect observed for fear, high SBP levels and low DBP levels (Ax, 1953; Christie and Friedman, 2004; Ekman et al., 1983; Schwartz et al., 1981; Sinha et al., 1992). However, significant differences in SBP have not been found, in spite of a higher mean in SBP after the anger induction. Bongard et al. (1997) found a similar cardiovascular reactivity pattern after anger provocation which particularly affected HR and DBP.

Considering hormonal changes after inducing anger and controlling circadian rhythms, we have found an increase in T levels as well as a decrease in C levels. Traditionally, higher T levels have been associated with anger and/or approach behavior (Hohlagschwandtner et al., 2001; Kalin, 1999; Persky et al., 1971; Thompson and George, 2003; Van Honk et al., 1999, 2000; Wirth and Schultheiss, 2007). Regarding C, our results showed a decrease in salivary C levels in response to anger induction. Many authors have associated lower C levels with anger, aggressive behavior and approach behavior (Lerner et al., 2007; Roelofs et al., 2005; Roy, 2004; Van Goozen et al., 1998; Van Honk et al., 2003). However, acute and relatively short term C elevations may facilitate approach behavior, including anger and aggression (Putman et al., 2007; Salvador, 2005; Salvador et al., 2003; Van Honk et al., 2000). So, the relationship between anger and C levels remain more controversial than with T.

We have not found any study on changes in T and/or C levels during an anger mood induction laboratory task, so the present study represents an unique contribution to this field, reporting changes in hormonal reactivity (T and C) when anger is evoked in healthy men by the AI method.

The most interesting finding of the correlational analysis was an inverse relationship between post-anger salivary cortisol and DBP levels which means that participants who showed a decrease in C levels increased their DBP after anger induction. Both, lower C levels (Kalin, 1999; Roelofs et al., 2005; Roy, 2004; Van Goozen et al., 1998; Van Honk et al., 2003) and higher DBP (e.g. Ax, 1953; Foster and Harrison, 2004; Prkachin et al., 2001; Rainville et al., 2006; Schwartz et al., 1981; Sinha et al., 1992; Weerts and Roberts, 1976) were documented in anger and aggression research. Nevertheless, none of these studies have combined both psychobiological parameters in their research paradigms.

No more relevant correlations regarding hormones were found. This could indicate that the measured variables in our study were relatively independent, although all were part of the psychobiological components of anger.

Therefore, when we viewed from the subjective perception and/or from the neuroendocrine and cardiovascular response of the subjects, our results indicated that the AI method was able to generate an experience of an anger affect. Another question was to analyze the consequences of that experience upon perceptual asymmetry when measured with a non-emotional laterality task. The analysis of DL scores before and after the anger induction lab task, yielded interesting results. On the one hand, a facilitation of RE items after the emotional induction was seen; on the other hand, no differences

in LE items were observed. Two models of attention could explain our results. On the one hand, Kinsbourne (1970, 1982) proposed a model for attentional-activation influences on DL performance. Briefly, the model predicts that a secondary task known to be lateralized in one hemisphere is able to alert, activate or "prime" that hemisphere, which in turns generates an attentional bias to the opposed hemifield, leading to a processing advantage of those items presented there. The model can be tested by analyzing the performance in a primary task (in this case, the DL test) with and without the addition of that secondary task. In this study the secondary task would be the anger mood induction. On the other hand, Hugdahl (2003) attentional model apply to hemisphere asymmetry the frequently used distinction in cognitive psychology and cognitive neuroscience between bottom-up or stimulus-driven information processing versus top-down, or instruction-driven information processing. A bottom-up approach would mean that language stimuli (e.g. syllables) would produce a left hemisphere response, while a top-down approach would ask the question whether a bottom-up asymmetry effect could be modulated or switched through cognitive means. Furthermore, correlational analysis revealed an obvious positive association between RE and REA, while LE was inversely related to both RE raw scores and REA after anger induction.

In our study, the results on DL performance (using both a LI and raw scores from the two ears) showed enhanced REA after anger, but due mainly to an increase in right ear items. So, the anger mood induction modulated the bottom-up asymmetry effect by increasing the REA, which would indicate greater left hemisphere activity. This increase in REA and specifically in right ear items could not be explained by a practice effect. For more details see the classical report of Porter et al. (1976) which observed that the REA remains practically invariable over an eight days period of testing. Moreover, the DL is not an exclusive test of temporal lobe function, as traditionally believed (Spreen and Strauss, 1991). Several studies (Hugdahl et al., 2003; Jäncke and Shah, 2002) have found the importance of a cortical network involving the left frontal lobe which is necessary for focusing attention on speech sounds. Therefore, our findings on brain asymmetry using the DL test agree with the motivational direction model, which has posited that the left frontal brain region is involved in the experience and expression of approach-related emotions (Fox, 1991; Harmon-Jones et al., 2006; Sutton and Davidson, 1997) like anger.

To our knowledge, there are few studies that introduced a negative affect experience and analyzed its consequences on DL performance (e.g. Demaree and Harrison, 1997; Gadea et al., 2005; Shenal and Harrison, 2003). All of them found a decrease in REA or even a LEA (left ear advantage) which suggest greater right hemisphere activity. This is due to the fact that all these studies use tasks that elicit sadness, pain, or anxiety (emotions associated with avoidance motivation) but not anger. Even Demaree and Harrison (1997) and Shenal and Harrison (2003), which took into account high and low hostility of the participants, did not use a method for inducing anger. Therefore, the present study is the first in analyzing the consequences of an anger experience upon perceptual asymmetry measured by a non-emotional DL test.

In examinations of anger state and asymmetrical cortical activity, there are some studies using PET (Dougherty et al., 1999; Marci et al., 2007) or EEG (Aftanas et al., 2006; Harmon-Jones et al., 2006; Harmon-Jones and Sigelman, 2001; Wacker et al., 2003) that have reported relatively greater left frontal activity after the induction of an anger mood state, supporting the motivational direction model too. Harmon-Jones and Sigelman (2001) conducted an experiment to assess whether situationally induced anger would increase relative left frontal activity. Participants were randomly assigned to a condition in which another person insulted them or to a condition in which another person treated them in a neutral manner. Immediately following the treatment, EEG was collected. As predicted, individuals who were insulted evidenced greater relative left frontal activity than individuals who were not insulted. Moreover, they reported being more angry and behave more aggressively towards the person who insulted them. Additionally, regression analyses revealed that relative left frontal activation was associated with more anger and aggression in the condition in which anger was evoked. More recently, Harmon-Jones et al. (2006) have found that an increase in the personal relevance of the anger stimuli coupled with the expectation of approach-related action increased relative left frontal activity in response to anger-producing pictures. Moreover, Aftanas et al. (2006) have reported changes in EEG activity during the experience of anger emotion. Specifically, the anterior and posterior areas of the cortex showed asymmetrical increases in theta-2 power in the left hemisphere and in the beta-1 range, the left hemisphere also showed an increase in response to the emotional film.

Although most theories of emotion support an essential role for physiology and neurobiology (Ekman and Davidson, 1994), few studies have combined different types of measures in research paradigms designed to study emotion. Several studies have examined cardiovascular reactivity and asymmetrical brain activity when anger is experienced. Harmon-Jones and Sigelman (2003) (unpublished data cited in Harmon-Jones, 2003) measured HR, SBP, DBP and EEG before and after participants were insulted. Results reveal that greater left frontal activity was associated with greater cardiovascular reactivity. Using PET, Dougherty et al. (1999) found greater activation of the left orbitofrontal cortex while subjects imagined the content of narrative scripts developed from autobiographical information to induce an anger state; however, no differences in cardiovascular reactivity were observed. Despite that, more recently and using the same procedure for inducing anger, Marci et al. (2007) besides reporting increased activity in the left orbitofrontal cortex and in the left insula, found increased HR too while anger was experienced. Nevertheless, in some studies (e.g. Foster and Harrison, 2004), despite reporting greater activation of frontal lobe and changes in cardiovascular reactivity, asymmetrical brain differences were not evident.

In summary, to our knowledge, this is the first study that examines in a single investigation different psychobiological parameters, including cardiovascular and hormonal reactivity as well as asymmetrical brain activity immediately after an anger induction. Our results agree with previous research about anger mood and are consistent with the theoretical writings of Darwin (1872/1965) and James (1884). In other words, we have found that anger emotion is accompanied by unique patterns of physiological and neurophysiological activity, and we have provided evidence supporting discrete anger emotion-specific nervous system activity. In our study, the emotion of anger, a negative emotion often associated with approach motivation, elicits an increase in cardiovascular response and in T salivary levels, and a decrease in C levels. Besides these changes in peripheral physiological activity we have evidenced a left asymmetrical brain activation using a DL test when anger is being experienced by right-handed men, supporting the motivational direction model. In future research, it would be interesting to include women and left-handed subjects for greater generalization of these results; and also to see if different response patterns to anger experience are observed.

Acknowledgments

The authors wish to thank all the volunteers which participated in the research. We thank Teresa Fuertes and Concepción Gómez for the revision of the English text and Inmaculada Pérez for her useful contribution to the format of the manuscript. We are also grateful to Dr. Julio Sanjuán for his valuable comments on the manuscript writing.

This study was supported by the Spanish Mental Health Network CIBERSAM(CB07/09/0006) and the Spanish MEC in the framework of the Consolider Eje C Project SEJ2006-1408.

References

Adams, R.B.J., Ambadi, N., Macrae, C.N., Kleck, R.E., 2006. Emotional expressions forecast approach-avoidance behavior. Motiv. Emot. 30, 179–188.

Aftanas, L.I., Reva, N.V., Savotina, L.N., Makhnev, V.P., 2006. Neurophysiological correlates of induced discrete emotions in humans: an individually oriented analysis. Neurosci. Behav. Physiol. 36, 119–130.

Ahern, G.L., Schwartz, G.E., 1985. Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. Neuropsychologia 23, 745–755.

American Psychological Association, 1992. Ethical principles of psychologists and code of conduct. Am. Psychol. 47, 1597–1611.

Archer, J., 2006. Testosterone and human aggression: an evaluation of the challenge hypothesis. Neurosci. Biobehav. Rev. 30, 319–345.

Ax, A.F., 1953. The physiological differentiation between fear and anger in humans. Psychosom. Med. 15, 433–442.

Balaguer, I., Fuentes, I., Meliá, J.L., García-Mérita, M.L., Pérez-Recio, G., 1993. El perfil de los estados de ánimo (P.O.M.S.): baremo para estudiantes valencianos y su aplicación en el contexto deportivo. Rev. Psicología del Deporte 4, 39–52.

Bartolic, E.I., Basso, M.R., Schefft, B.K., Glauser, T., Titanic-Schefft, M., 1999. Effects of experimentally-induced emotional states on frontal lobe cognitive task performance. Neuropsychologia 37, 677–683.

Berkowitz, L., 1999. Anger. In: Dalgleish, T., Power, M.J. (Eds.), Handbook of Cognition and Emotion. John Wiley and Sons, Chichester, England, pp. 411–428.

Bongard, S., Pfeiffer, J.S., al'Absi, M., Hodapp, V., Linnenkemper, G., 1997. Cardiovascular responses during effortful active coping and acute experience of anger in women. Psychophysiology 34, 459–466.

Bradley, M.M., Lang, P.J., 2000. Measuring emotion: behaviour, feeling, and physiology. In: Lane, R.D., Nedal, L. (Eds.), Cognitive Neuroscience of Emotion. Oxford Univ. Press, New York, pp. 142–276.

Buss, K.A., Schumacher, J.R., Dolski, I., Kalin, N.H., Goldsmith, H.H., Davidson, R.J., 2003. Right frontal brain activity, cortisol, and withdrawal behavior in 6-month-old infants. Behav. Neurosci. 117, 11–20.

Cannon, W.B., 1929. Bodily Changes in Pain, Hunger, Fear, and Rage, 2nd ed. Appleton, New York.

Cacioppo, J.T., Berntson, G.G., Larsen, J.T., Poehlmann, K.M., Ito, T.A., 2000. The psychophysiology of emotion, In: Lewis, M., Haviland-Jones, J.M. (Eds.), The Handbook of Emotion, 2nd ed. Guildford Press, New York, pp. 173–191.

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

Christie, I.C., Friedman, B.H., 2004. Autonomic specificity of discrete emotion and dimensions of affective space: a multivariate approach. Int. J. Psychophysiol. 51, 143–153.

Dabbs Jr., J.M., Carr, T.S., Frady, R.L., Riad, J.K., 1995. Testosterone, crime and misbehaviour among 692 prison inmates. Pers. Indiv. Diff. 18, 633–727.

Darwin, C., 1872/1965. The expression of the emotions in man and animals. University of Chicago Press, Chicago, IL.

D'Alfonso, A.A.L., van Honk, J., Hermans, E., Postma, A., de Haan, E.H.F., 2000. Laterality effects in selective attention to threat after repetitive transcranial magnetic stimulation at the prefrontal cortex in female subjects. Neurosci. Lett. 280, 195–198.

De Kloet, E.R., 2004. Hormones and the stressed brain. Ann. N.Y. Acad. Sci. 1018, 1–15.

Demaree, H.A., Harrison, D.W., 1997. Physiological and neuropsychological correlates of hostility. Neuropsychologia 35, 1405–1411.

Dougherty, D.D., Shin, L.M., Alpert, N.M., Pitman, R.K., Orr, S.P., Lasko, M., Macklin, M.L., Fischman, A.J., Rauch, S.L., 1999. Anger in healthy men: A PET study using script-driven imagery. Biol. Psychiatry 46, 466–472.

Ekman, P., Davidson, R.J., 1994. The Nature of Emotion: Fundamental Questions. Oxford Univ. Press, New York.

Ekman, P., Levenson, R.W., Friesen, W.V., 1983. Autonomic nervous system activity distinguishes among emotions. Science 221, 1208–1210.

Engebretson, T.O., Sirota, A.D., Niaura, R.S., Edwards, K., Brown, W.A., 1999. A simple laboratory method for inducing anger: a preliminary investigation. J. Psychosom. Res. 47, 13–26.

Foster, P.S., Harrison, D.W., 2004. The covariation of cortical electrical activity and cardiovascular responding. Int. J. Psychophysiol. 52, 239–255.

Fox, N.A., 1991. If it's not left, it's right. Electroencephalograph asymmetry and the development of emotion. Am. Psychol. 46, 863–872.

Gadea, M., Gomez, C., Gonzalez-Bono, E., Salvador, A., Espert, R., 2003. Salivary testosterone is related to both handedness and degree of linguistic lateralization in normal women. Psychoneuroendocrinology 28, 274–287.

Gadea, M., Gómez, C., González-Bono, E., Espert, R., Salvador, A., 2005. Increased cortisol and decreased right ear advantage (REA) in dichotic listening following a negative mood induction. Psychoneuroendocrinology 30, 129–138.

Gerra, G., Avanzini, P., Zaimovic, A., Fertonani, G., Caccavari, R., Delsignore, R., Gardini, F., Talarico, E., Lecchini, R., Maestri, D., Brambilla, F., 1996. Neurotransmitter and endocrine modulation of aggressive behavior and its components in normal humans. Behav. Brain. Res. 81, 19–24.

Gray, A., Jackson, D.N., McKinlay, J.B., 1991. The relation between dominance, anger, and hormones in normally aging men: results from the Massachusetts Male Aging Study. Psychosom. Med. 53, 375–385.

Early Career AwardHarmon-Jones, E., 2003. Clarifying the emotive functions of asymmetrical frontal cortical activity. Psychophysiology 40, 838–848.

Harmon-Jones, E., 2004a. On the relationship of frontal brain activity and anger. Examining the role of attitude towards anger. Cogn. Emot. 18, 337–361.

Harmon-Jones, E., 2004b. Contributions from research on anger and cognitive dissonance to understanding the motivational functions of asymmetrical frontal brain activity. Biol. Psychol. 67, 51–76.

Harmon-Jones, E., 2007. Trait anger predicts relative left frontal cortical activation to anger-inducing stimuli. Int. J. Psychophysiol. 66, 154–160.

Harmon-Jones, E., Allen, J.J., 1998. Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. J. Pers. Soc. Psychol. 74, 1310–1316.

Harmon-Jones, E., Sigelman, J., 2001. State anger and frontal brain activity: evidence that insult-related relative left prefrontal activation is associated with experienced anger and aggression. J. Pers. Soc. Psychol. 80, 797–803.

Harmon-Jones, E., Lueck, L., Fearn, M., Harmon-Jones, C., 2006. The effect of personal relevance and approach-related action expectation on relative left frontal cortical activity. Psychol. Sci. 17, 434–440.

Hohlagschwandtner, M., Husslein, P., Klier, C., Ulm, B., 2001. Correlation between serum testosterone levels and peripartal mood states. Acta Obstet. Gynecol. Scand. 80, 326–330.

Hugdahl, K., 2003. Dichotic listening in the study of auditory laterality. In: Hugdahl, K., Davidson, R.J. (Eds.), The Asymmetrical Brain. MIT Press, Cambridge, MA, pp. 441–476.

Hugdahl, K., Bodner, T., Weiss, E., Benke, T., 2003. Dichotic listening performance and frontal lobe function. Cogn. Brain Res. 16, 58–65.

James, W., 1884. What is an emotion? Mind 9, 188–205.

Jäncke, L., Shah, N.J., 2002. Does dichotic listening probe temporal lobe functions? Neurology 58, 736–743.

Kalin, N.H., 1999. Primate models to understand human aggression. J. Clin. Psychiatry 60, 29–32.

Kinsbourne, M., 1970. The cerebral basis of lateral asymmetries in attention. Acta Psychol. (Amst) 33, 193–201.

Kinsbourne, M., 1982. Hemispheric specialization and the growth of human understanding. Am. Psychol. 37, 411–420.

Lazarus, R.S., 1991. Emotion and Adaptation. Oxford Univ. Press, New York.

Lerner, J.S., Dahl, R.E., Hariri, A.R., Taylor, S.E., 2007. Facial expressions of emotion reveal neuroendocrine and cardiovascular stress responses. Biol. Psychiatry 61, 253–260.

Lewis, M., Ramsay, D., 2005. Infant emotional and cortisol responses to goal blockage. Child Dev. 76, 518–530.

Marci, C.D., Glick, D.M., Loh, R., Dougherty, D.D., 2007. Autonomic and prefrontal cortex responses to autobiographical recall of emotions. Cogn. Affect. Behav. Neurosci. 7, 243–250.

Mazur, A., Booth, A., 1998. Testosterone and dominance in men. Behav. Brain. Sci. 21, 353–397.

McNair, D.M., Lorr, M., Droppleman, L.F. (Eds.), 1971. How to Use the Profile of Mood States (POMS) in Clinical Evaluations. Educational and Industrial Testing Service, San Diego.

O'Connor, D.B., Archer, J., Wu, F.C., 2004. Effects of testosterone on mood, aggression, and sexual behavior in young men: a double-blind, placebo-controlled, cross-over study. J. Clin. Endocrinol. Metab. 89, 2837–2845.

Ohira, H., Nomura, M., Ichikawa, N., Isowa, T., Iidaka, T., Sato, A., Fukuyama, S., Nakajima, T., Yamada, J., 2006. Association of neural and physiological responses during voluntary emotion suppression. Neuroimage 29, 721–733.

Olfield, R.C., 1971. The assessement and analysis of handedness: the Edinburg Inventory. Neuropsychologia 9, 97–113.

Persky, H., Smith, K.D., Basu, G.K., 1971. Relation of psychologic measures of aggression and hostility to testosterone production in man. Psychosom. Med. 33, 265–277.

Pope Jr., H.G., Kouri, E.M., Hudson, J.I., 2000. Effects of supraphysiologic doses of testosterone on mood and aggression in normal men: a randomized controlled trial. Arch. Gen. Psychiatry 57, 133–140.

Porter Jr., R.J., Troendle, R., Berlin, C.I., 1976. Effects of practice on the perception of dichotically presented stop-consonant–vowel syllables. J. Acoust. Soc. Am. 59, 679–682.

Prkachin, K.M., Mills, D.E., Zwaal, C., Husted, J., 2001. Comparison of hemodynamic responses to social and nonsocial stress: Evaluation of an anger interview. Psychophysiology 38, 879–885.

Putman, P., Hermans, E.J., van Honk, J., 2007. Exogenous cortisol shifts a motivated bias from fear to anger in spatial working memory for facial expressions. Psychoneuroendocrinology 32, 14–21.

Rainville, P., Bechara, A., Naqvi, N., Damasio, A.R., 2006. Basic emotions are associated with distinct patterns of cardiorespiratory activity. Int. J. Psychophysiol. 61, 5–18.

Robinson, R.G., 1995. Mapping brain activity associated with emotion. Am. J. Psychiatry 152, 327–329.

Roelofs, K., Elzinga, B.M., Rotteveel, M., 2005. The effects of stress-induced cortisol responses on approach-avoidance behavior. Psychoneuroendocrinology 30, 665–677.

Roy, M.P., 2004. Patterns of cortisol reactivity to laboratory stress. Horm. Behav. 46, 618–627.

Rybak, M., Crayton, J.W., Young, I.J., Herba, E., Konopka, L.M., 2006. Frontal alpha power asymmetry in aggressive children and adolescents with mood and disruptive behavior disorders. Clin. EEG Neurosci. 37, 16–24.

Salvador, A., 2005. Coping with competitive situations in humans. Neurosci. Biobehav. Rev. 29, 195–205.

Salvador, A., Costa, R., 2009. Coping with competition: Neuroendocrine responses and cognitive variables. Neurosci. Biobehav. Rev. 33, 160–170.

Salvador, A., Suay, F., Gonzalez-Bono, E., Serrano, M.A., 2003. Anticipatory cortisol, testosterone and psychological responses to judo competition in young men. Psychoneuroendocrinology 28, 364–375.

Sandín, B., Chorot, P., Lostao, L., Joiner, T.E., Santed, M.A., y Valiente, R.M., 1999. Escalas de afecto positivo y negativo: validación factorial y convergencia estructural. Psicothema 11, 37–51.

Schwartz, G.E., Weinberger, D.A., Singer, J.A., 1981. Cardiovascular differentiation of happiness, sadness, anger, and fear following imagery and exercise. Psychosom. Med. 43, 343–364.

Shenal, B.V., Harrison, D.W., 2003. Investigation of the laterality of hostility, cardiovascular regulation, and auditory recognition. Int. J. Neurosci. 113, 205–222.
Silberman, E.K., Weingartner, H., 1986. Hemispheric lateralization of functions related to emotion. Brain Cogn. 5, 322–353.
Sinha, R., Lovallo, W.R., Parsons, O.A., 1992. Cardiovascular differentiation of emotions. Psychosom. Med. 54, 422–435.
Spreen, O., Strauss, E. (Eds.), 1991. A Compendium of Neuropsychological Test: Administration, Norm, Commentary. Oxford University Press, New York.
Stewart, J.L., Levin-Silton, R., Sass, S.M., Heller, W., Miller, G.A., 2008. Anger style, psychopathology, and regional brain activity. Emotion 8, 701–713.
Su, T.P., Pagliaro, M., Schmidt, P.J., Pickar, D., Wolkowitz, O., Rubinow, D.R., 1993. Neuropsychiatric effects of anabolic steroids in male normal volunteers. JAMA 269, 2760–2764.
Sutton, S.K., Davidson, R.J., 1997. Prefrontal brain asymmetry: a biological substrate of the behavioral approach and inhibition systems. Psychol. Sci. 8, 204–210.
Thompson, R.R., George, K., 2003. Testing the relationship between levels of endogenous testosterone and physiological responses to facial expressions in men: An experiment conducted by students in an undergraduate behavioral neuroscience class. J. Undergraduate Neurosci. Education (JUNE) 1, A41–A47.
Van Bokhoven, I., van Goozen, S.H., van Engeland, H., Schaal, B., Arseneault, L., Séguin, J.R., Assaad, J.M., Nagin, D.S., Vitaro, F., Tremblay, R.E., 2006. Salivary testosterone and aggression, delinquency, and social dominance in a population-based longitudinal study of adolescent males. Horm. Behav. 50, 118–125.
Van der Meij, L., Buunk, A.P., van de Sande, J.P., Salvador, A., 2008. The presence of a woman increases testosterone in aggressive dominant men. Horm. Behav. 54, 640–644.
Van Eck, M., Berkhof, H., Nicolson, N., Sulon, J., 1996. The effects of perceived stress, traits, mood states, and stressful daily events on salivary cortisol. Psychosom. Med. 58, 447–458.
Van Goozen, S.H., Matthys, W., Cohen-Kettenis, P.T., Gispen-de Wied, C., Wiegant, V.M., van Engeland, H., 1998. Salivary cortisol and cardiovascular activity during stress in oppositional-defiant disorder boys and normal controls. Biol. Psychiatry 43, 531–539.
Van Honk, J., Schutter, D.J., 2006. From affective valence to motivational direction: the frontal asymmetry of emotion revised. Psychol. Sci. 17, 963–965.
Van Honk, J., Schutter, D.J., 2007. Testosterone reduces conscious detection of signals serving social correction: implications for antisocial behavior. Psychol. Sci. 18, 663–667.
Van Honk, J., Tuiten, A., Verbaten, R., van den Hout, M., Koppeschaar, H., Thijssen, J., de Haan, E., 1999. Correlations among salivary testosterone, mood, and selective attention to threat in humans. Horm. Behav. 36, 17–24.
Van Honk, J., Tuiten, A., van den Hout, M., Koppeschaar, H., Thijssen, J., de Haan, E., Verbaten, R., 2000. Conscious and preconscious selective attention to social threat: different neuroendocrine response patterns. Psychoneuroendocrinology 25, 577–591.
Van Honk, J., Kessels, R.P., Putman, P., Jager, G., Koppeschaar, H.P., Postma, A., 2003. Attentionally modulated effects of cortisol and mood on memory for emocional faces in healthy young males. Psychoneuroendocrinology 28, 941–948.
Velten, E., 1968. A laboratory task for induction of mood states. Behav. Res. Ther. 6, 473–482.
Wacker, J., Heldmann, M., Stemmler, G., 2003. Separating emotion and motivational direction in fear and anger: Effects on frontal asymmetry. Emotion 3, 167–193.
Watson, D., Clark, L.A., Tellegen, A., 1988. Development and validation of brief measures of positive and negative affect: the PANAS scales. J. Pers. Soc. Psychol. 34, 1063–1070.
Watson, D., Wiese, D., Vaidya, J., Tellegen, A., 1999. The two general activation systems of affect: structural findings, evolutionary considerations, and psychobiological evidence. J. Pers. Soc. Psychol. 76, 820–838.
Weerts, T.C., Roberts, R., 1976. The physiological effects of imagining anger-provoking and fear-provoking scenes. SPR Abstr 13, 174.
Wirth, M.M., Schultheiss, O.C., 2007. Basal testosterone moderates responses to anger faces in humans. Physiol. Behav. 90, 496–505.