Anger in brain and body: the neural and physiological perturbation of decision-making by emotion

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Abstract

Emotion and cognition are dynamically coupled to bodily arousal: the induction of anger, even unconsciously, can reprioritise neural and physiological resources toward action states that bias cognitive processes. Here we examine behavioural, neural and bodily effects of covert anger processing and its influence on cognition, indexed by lexical decision-making. While recording beat-to-beat blood pressure, the words ANGER or RELAX were presented subliminally just prior to rapid word/non-word reaction-time judgements of letter-strings. Subliminal ANGER primes delayed the time taken to reach rapid lexical decisions, relative to RELAX primes. However, individuals with high trait anger were speeded up by subliminal anger primes. ANGER primes increased systolic blood pressure and the magnitude of this increase predicted reaction time prolongation. Within the brain, ANGER trials evoked an enhancement of activity within dorsal pons and an attenuation of activity within visual occipitotemporal and attentional parietal cortices. Activity within periaqueductal grey matter, occipital and parietal regions increased linearly with evoked blood pressure changes, indicating neural substrates through which covert anger impairs semantic decisions, putatively through its expression as visceral arousal. The behavioural and physiological impact of anger states compromises the efficiency of cognitive processing through action-ready changes in autonomic response that skew regional neural activity.

Key words: systolic blood pressure; subliminal; lexical decision task; fMRI; emotion

Introduction

Affective states are expressed in body and brain, and these neural and physiological changes can influence subsequent cognitive processes. Different emotion types generally fall into prescribed and distinct patterns of autonomic and central activation (Harrison *et al.*, 2013). These, in turn, can lead to

predictable effects on cognition and behaviour, such as enhanced attentional capture and facilitated reaction time. Much research focuses on the effect of emotions such as fear, sadness and happiness, which align with psychopathological states of anxiety, depression and hypomania. However anger remains relatively understudied (in terms of neuronal and physiological mechanisms of action) and it is harder to predict the likely influences of anger on cognition and behaviour.

Exposure to most emotion categories can result in a mirroring, or embodiment, of that emotion in the perceiver (Harrison *et al.*, 2006; Singer and Lamm, 2009; Harrison *et al.*, 2010). Observing anger has the potential to elicit either an anger state, or a 'complementary' fear state (Harrison *et al.*, 2013), reflecting the dual response of fight or flight. In accordance with its negative status, (subliminal) anger priming decreases liking judgments of neutral stimuli (Murphy and Zajonc, 1993) and enhances avoidance behaviours (Marsh *et al.*, 2005). Anger nevertheless possesses motivating properties normally expressed by positive emotions and which facilitate behaviour. As a consequence of such observations, anger is also viewed as an approach emotion (van Honk and Schutter, 2006; Carver and Harmon-Jones, 2009; Lewis, 2010). Overt or subliminal anger primes elicited a greater desire to possess associated everyday objects compared to neutral or fear primes (Aarts *et al.*, 2010). Anger is linked to 'high optimism, positive expectations and experiences of high coping potential' (Lerner and Keltner, 2001) and, importantly, anger is associated with perceived task ease, a status which again diverges from other negatively valenced emotions, such as sadness, which are associated with greater perceived task difficulty (Gendolla and Silvestrini, 2011).

Both facilitatory and impairing effects of anger thus have the potential to occur, with the direction and the magnitude of anger effects sensitive to context (e.g. obtainable reward, Aarts *et al.*, 2010) and individual differences (e.g. testosterone levels) (Wirth and Schultheiss, 2007).

The physiological expression of anger and hostility has been recognised as important in psychosomatic medicine and health (Smith *et al.*, 2004). Effects of anger on physiological parameters are potentially pathoaetiological in the genesis of cardiovascular disease and hypertension, yet are subject to individual differences. Systolic blood pressure correlates with questionnaire measures of trait anger (Francis *et al.*, 1991). However the process of inducing anger may determine the extent and direction of cardiovascular responses (Kreibig, 2010): Anger elicited in the context of harassment or personalized recall increases cardiac output (Prkachin *et al.*, 2001) along with a shortening of the pre-ejection period, indicating sympathetic effects (Neumann and Waldstein, 2001; Herrald and Tomaka, 2002). In contrast, physiological responses to angry facial expressions presented both supra and subliminally can induce cardiac states more reminiscent of fear, such as heart rate decreases instead of increases, as well as increases in heart rate variability (HRV) and decreases in galvanic skin response (Jonsson and Sonnby-Borgstrom, 2003; Vrana and Gross, 2004; Dimberg and Thunberg, 2007), reflecting potential parasympathetic activation. Subliminal primes displaying the word ANGER have been shown to increase both systolic and diastolic blood pressure, relative to a control group exposed to the subliminal prime RELAX (Hull *et al.*, 2002). Using subliminal priming thus presents a valuable tool to explore the interplay between physiology and brain in an anger context and provides an insight into the mechanisms through which anger may exert effects on cognition and behaviour. Research to date has attempted to delineate the effects of emotions such as fear, happiness and sadness on

cognition and behaviour, or study the relationship between blood pressure and neural activity (Coulson *et al.*, 2015). However, no study has specifically investigated the interplay of neural and physiological pathways through which anger influences cognition. Indeed, propensity for anger to be both a motivating emotion and an emotion with negative status renders the consequential effects of anger on attention and reaction time unclear. A greater understanding of anger effects on blood pressure and neural activity, and their subsequent relationship to behaviour, can help provide insight into the mechanisms through which anger may exert its influence. We thus simultaneously monitored physiology (e.g. blood pressure) and brain (e.g. using fMRI) to comprehensively detail the physiological and neural mechanisms through which subliminal ANGER may affect decision making.

Summary of experiments

The experiments were designed to investigate whether subliminal induction of an emotional state, specifically anger, altered cognitive processing, as exemplified by lexical decision-making. On each experimental trial, the participant saw a target letter-string presented on a video monitor and was required to judge if this string was a word or nonsense non-word. Before each presentation of the target letter-string, the participant was 'emotionally-primed' by the words ANGER, or RELAX, which were presented subliminally at the point of visual fixation. We checked carefully that the ANGER and RELAX words were subliminal (i.e. not consciously seen by the participants) using a similar laboratory threshold task, described below. Participants performed the main task in the laboratory (Experiment 1) using accuracy and reaction time measures of performance, and during functional neuroimaging (Experiment 2) to test how in the brain emotions can alter decisions. We also measured participants' blood pressure during task performance to characterise the physiological expression of emotional state, manifestation in the brain and impact on behaviour.

Methods

Experiment 1: Psychophysiology

Participants. Twenty-one healthy control participants were recruited from advertisements placed around the University and Medical School campus to take part in the laboratory-only physiological investigation. These healthy volunteers (7 men, 14 women; mean age 22.7 years ± s.d. 1.1; all right handed) provided written consent to take part in the study which was approved by the Brighton and Sussex Medical School (BSMS) Research Governance and Ethics Committee. Two participants were excluded due to artefacts within the blood pressure recording. One participant was excluded as an extreme outlier (reaction time differences were greater than 3 s.d. from the mean of the group sample).

Paradigm. Two-hundred neutral seven-letter words were chosen from a selection of word databases (e.g. ASKNEW). Of these, half had a letter substituted to form 100 non-word letter-strings. Thus the total stimulus set comprised 100 words and 100 non-word letter-strings that served as the basis for lexical decisions. During the experiment, these letter-strings were preceded either by the subliminal prime ANGER, or by RELAX to form 50 trials in each condition (50 words preceded by RELAX, 50 non-words preceded by RELAX, 50 words preceded by

ANGER, 50 non-words preceded by ANGER). Each prime was presented in blocks of four. This ensured that a minimum of four ANGER or four RELAX trials were presented consecutively. Within each four-prime block, half of trials were word trials and half were non-word trials, the order of which were randomized. In each trial, the ANGER and RELAX primes were rendered subliminal using forward and backward masking (Hull *et al.*, 2002) (see below). This cognitive task was run using Matlab software and was synchronized with physiological recording using Spike2 6.08 software.

Physiological recording. In Experiment 1, blood pressure was recorded using non-invasive, beat-to-beat monitoring via photoplethysmographic technology (Finometer PRO; Finapres 2300, Ohmeda, Eaglewood, CO, USA). An inflatable finger cuff and infrared plethysmograph were fitted to the index finger of the subject's right hand, allowing measurements of beat-to-beat systolic blood pressure.

Procedure. After a 5 min calibration period, the participant was invited to start the task of 200 experimental trials. Each trial began with a 1500 ms fixation cross, followed by a forward mask of seven scrambled letters (17 ms), the ANGER or RELAX prime (17 ms), a blank screen (17 ms), a backwards mask of seven scrambled letters (50 ms) and a blank screen (100 ms). This rapid series of events was immediately followed by a target word/non-word seven letter-string that remained on the screen until participants made a lexical decision, responding by key-press either '1' to denote a word, or '2' to signify a non-word. Participants were encouraged to answer as quickly and accurately as possible. Lexical decision strings remained on the screen until the participant made their response. Reaction times (RTs) and correctness were recorded. The order in which these blocks were presented was randomized.

Experiment 2: Neuroimaging

Participants. A second group of twenty healthy control participants were recruited from advertisements placed around the Medical School and University campus. One participant was removed from all analyses due to non-compliance with experimental procedures and excessive movement in the scanner, resulting in 9 male and 10 female participants (mean age 24.6 years ± s.d. 5.0; all right handed).

Physiological recording. Blood pressure was recorded using a Portapres device (Finapres Medical Systems) adapted in-house to be fully fMRI compatible (Gray *et al.*, 2009). Due to equipment failure within the scanning environment, complete sets of blood pressure recordings could not be obtained for five participants. An fMRI compatible inflatable finger cuff and infrared plethysmograph were fitted to the thumb of the subject's left hand, allowing measurements of beat-to-beat systolic blood pressure. This was recorded and synchronized with stimulus presentation and scan volumes using Spike 2 6.08 software.

Paradigm and procedure. The stimuli and experimental protocol were identical to that followed in the psychophysiological lab, with a few minor modifications made to better optimize task design for fMRI. Lexical decisions were now blocked in groups of six by prime (RELAX, ANGER) instead of four. Within each six-prime block, half of trials were word trials and half were non-word trials, the order of which were randomized. In addition, each trial was fixed at a 3500 ms to create block durations of

21 s. Previously, all prime decisions remained on the screen until a lexical decision was made before switching to a fixation cross. To ensure a fixed trial length, this cross remained on screen until a total trial duration of 3500 ms was reached. If no decision was made within the 3500 ms period, the experiment proceeded to the next trial. Participants were warned to not let this occur, and instructed to make their responses as quickly and as accurately as possible.

MRI acquisition and preprocessing. Participants underwent functional magnetic resonance imaging (fMRI) in a Siemens Avanto 1.5 Tesla MRI scanner (Siemens, Erlangen, Germany). Prior to acquisition of the functional dataset, a whole brain T1-weighted structural scan was acquired for each participant (MPRAGE, 0.9 mm^3 voxels, 192 slices, 1160/4.24 TR/TE, 300 ms inversion time, 230×230 mm^2 FOV, 5 min acquisition time). Then, during performance of the experimental task, functional echo-planar datasets sensitive to blood oxygen level dependent contrast were acquired using axial slices (anteriorposterior phase encode direction) tilted 30° from intercommissural plane to minimize T2* signal dropout from orbitofrontal and anterior temporal regions. Thirty-five 3-mm slices with a 0.75 mm interslice gap provided whole-brain coverage with an in-plane resolution of 3×3 mm (TE 42 ms, volume TR 2620 ms). Images were preprocessed using SPM 8 (http://www.fil.ion.ucl.ac.uk/spm/). The initial four functional volumes were discarded to allow for equilibration of net magnetization. Images were then spatially realigned and unwarped, and spatially normalized to standard MNI space (Montreal Neurological Institute). Normalized functional scans were then smoothed with an 8 mm Gaussian smoothing kernel. For small volume correction analyses focusing on brainstem, a 15-mm sphere functional ROI for dorsal pons [2 −30 −32] was derived from previous work demonstrating its sensitivity to first order representations of bodily state (Critchley *et al.*, 2001), implicating it in anger processing (Damasio *et al.*, 2000) and core aspects of emotion (Damasio, 2010).

Experiment 2 a: Subliminal threshold check

To verify that all ANGER and RELAX primes were presented below the subjective (and objective) threshold; participants were given an additional series of 32 trials in the psychophysiological laboratory as the final experimental procedure (for participants in Experiment 2 only). All task parameters remained the same, but instead of making word/non-word judgments, participants were now instructed to press '1' if the word/non-word was preceded by ANGER or '2' if it was proceeded by RELAX. After this forced choice decision, their confidence was assessed on a scale of 1 (complete guess) to 50 (complete confidence) using a computerized VAS.

Data analyses

Physiological data processing. Beat-to-beat values of systolic blood pressure (mmHg) recorded during both the laboratory and fMRI experiments were processed as follows: First the heartbeat values were interpolated over time (100 Hz) and smoothed using a Gaussian function (set to 1) implanted in Matlab (MATLAB and Statistics Toolbox Release 2012b, The MathWorks, Inc., Natick, Massachusetts, United States). These created a constant signal over systolic peaks and averaged across potential spike artefacts. Mean systolic blood pressure levels were then derived by averaging systolic levels over each block of trials and the

baseline value (defined by the average systolic level from the preceding 2000 ms from block onset) was subtracted to index average evoked blood pressure change. These average block values were then analysed according to emotion prime type to give mean systolic levels separately for blocks containing ANGER versus RELAX primes.

Behavioural data. To limit the effect of outliers, all reaction time (rt) analyses were performed on the reciprocal of the reaction time (1/rt), transforming data so that higher values signify a faster response. Mean correct responses were calculated for each emotion prime condition (total number of correct responses during subliminal anger prime blocks and relax prime blocks). A repeated measures 2×2 ANOVA containing prime (ANGER, RELAX) and word status (word, non-word) was performed for reciprocal rt within the psychophysiological study, and, to mirror subsequent fMRI analyses, a targeted within-participants *t*-test (2-tailed) investigated the main effect of prime during the neuroimaging study behavioural replication. To investigate the relationship between blood pressure change and task performance, blood pressure elevations due to ANGER primes (ANGER – RELAX) were correlated with changes in reciprocal reaction time due to ANGER primes (1/rt for ANGER – RELAX primes). The strength of the correlation was measured using Pearson's r. For behavioural and blood pressure analyses, effect sizes are reported using Cohen's *d* (*d* for within-participant *t*-tests), partial eta squared (η^2_p for ANOVA) (Cohen, 1988).

Trait anger was assessed using the anger dimension of the Buss/Perry Aggression Questionnaire (Buss and Perry, 1992).

Neuroimaging analyses. Functional imaging data were analysed using SPM 8 within a mixed effects design to investigate the influence of ANGER and RELAX primes on brain activity. In individual first-level analyses, neural activity was modelled for blocks containing ANGER primes and contrasted to blocks containing RELAX primes. Blocks were convolved with a canonical haemodynamic response function and regressed in a multiple regression GLM analysis against voxel-wise time-series data from the preprocessed echoplanar images. Six movement regressors derived at pre-processing were included as confounding covariates. Subject-wise contrast images were tested for experimental effects of interest in second-level group analyses focusing on the main effect of prime (ANGER versus RELAX). To specifically isolate the neural substrates through which affective primes impact on behaviour, the degree to which participants were susceptible to the affective primes was calculated (a reciprocal correction was applied to limit the

influence of outliers (Whelan, 2008): average 1/rt for ANGER – average 1/rt for RELAX primes). This value was regressed against subject contrast images at the second level, modelled as the only variable of interest. Individual threshold maps were set to $P < 0.005$, and all results reported were cluster-level corrected using family wise error rate (FWE) $P < 0.05$.

To investigate neural coupling with systolic blood pressure changes, the average systolic blood pressure level was calculated during each scan volume. These were then convolved to the hemodynamic response and regressed at first level for each participant, along with convolved standard deviation for these measures and raw systolic values. Movement regressors were also included in the model, and contrasts at the second level explicitly modelled positive and negative activity associated with rises and falls in convolved systolic blood pressure levels. Individual threshold maps were again set to $P < 0.005$ and all results are reported at $P < 0.05$ cluster level.

As there are known differences between males and females in the reaction to anger (Stemmler and Wacker, 2010) and in neural activation in reaction to emotional manipulations (Sacher *et al.*, 2013), sex was added as a nuisance regressor in all fMRI analyses.

Results

Experiment 1: Psychophysiological laboratory experiment

Behavioural analyses. A main effect of prime [F(1,20) = 13.031, $P = 0.002$; $\eta^2_p = .395$] signified that reaction times for lexical decisions following the subliminal prime ANGER were significantly impaired relative to lexical decisions preceded by the subliminal prime RELAX (Figure 1A). Reaction times were also delayed when making lexical decisions to non-words relative to words, as indicated by a main effect of word type [F(1,20) = 65.960, $P < 0.001$; $\eta^2_p = .767$]. There was no interaction between prime and word type, thus the ANGER prime impaired reaction time irrespective of whether the lexical decision was in response to a word or a non-word stimulus [$\eta^2_p = .055$].

Blood pressure analyses. Average systolic blood pressure levels were significantly elevated during ANGER prime blocks relative to RELAX prime blocks [t(18) = 2.5 $P = 0.02$; $d = 0.579$], in which ANGER primes caused an increase in blood pressure relative to baseline, and RELAX a relative decrease (Figure 1B).

Behaviour and physiological interactions. The relationship between blood pressure reactivity and reaction time was assessed. Changes in blood pressure between ANGER and RELAX priming
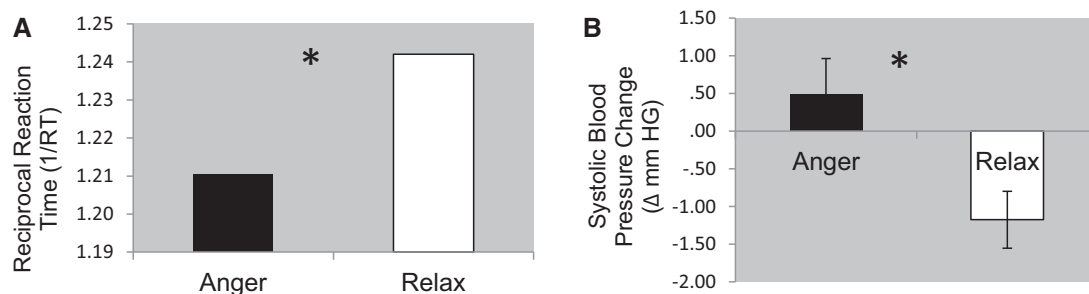

Fig. 1. (A) Reciprocal reaction times against prime type: Reaction times for word/non-word decisions were significantly longer when proceeding ANGER primes and faster following RELAX primes. (B) Systolic blood pressure change against prime type: ANGER primes raised blood pressure while RELAX primes reduced it, relative to preceding baseline.
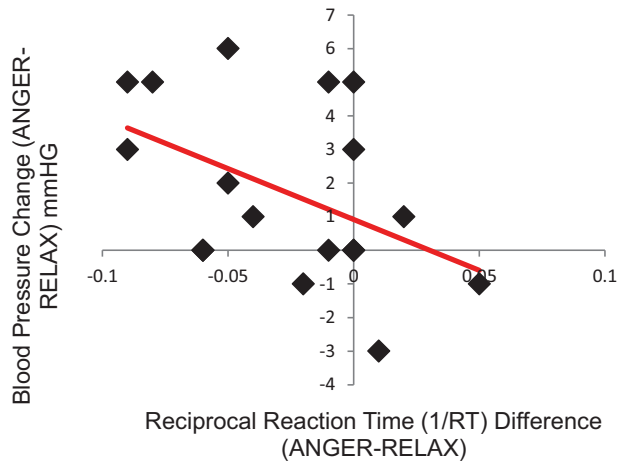
Fig. 2. Relationship between systolic blood pressure elevation and augmented RT to ANGER versus RELAX primes. Individuals with greater blood pressure variation between ANGER and RELAX primes also had a larger difference in reaction time for lexical decisions preceded by ANGER versus RELAX primes. Specifically, individuals with greater increases in blood pressure to ANGER versus RELAX primes displayed a greater slowing in reaction time for lexical decisions to ANGER primes.

conditions were analysed against reciprocal reaction time differences between the two priming conditions, separately for each participant. This relationship was significant ($r = -0.498$, $P = 0.038$) (Figure 2), demonstrating that ANGER primes were associated with increased reaction times and elevated blood pressure response, relative to RELAX primes.

Experiment 2: Neuroimaging experiment

Behavioural replication. Reaction times for lexical decisions following ANGER primes were significantly slower relative to those following RELAX primes [$t(18) = -3.646$, $P = 0.002$; $d = -0.898$], replicating the previous ANGER impairment effect.

Trait anger. Trait anger was related to the difference in reaction time between ANGER and RELAX trials ($r = 0.5$, $P = 0.047$), signifying that individuals high on trait anger were more likely to be sped up by subliminal ANGER primes relative to RELAX primes (despite the group-level propensity for subliminal anger primes to impede reaction time).

Systolic blood pressure replication. Systolic blood pressure was significantly enhanced on trials preceded by an ANGER prime relative to a RELAX prime [$t(13) = 2.30$, $P = 0.038$ $d = 0.711$].

Neuroimaging data. Results demonstrate that ANGER primes relative to RELAX primes activated brainstem, specifically dorsal pons/parabrachial nucleus ([$-8\ -36\ -30$], 48 voxels, $T = 5.34$), an area sensitive to sympathetic arousal (Figure 3). ANGER trials induced hypoactivation of parietal lobe extending into postcentral gyrus and precuneus ([$56\ -32\ -44$], 1106 voxels, $T = 5.76$, P-FWE < 0.001), and visual cortex/cuneus ([$24\ -88\ 32$], 352 voxels, $T = 4.64$, P-FWE $= 0.019$).

The impairing effect on reaction time of ANGER, relative to RELAX primes was also associated with attenuation of activity across visual areas, including cuneus, middle occipital gyrus and lingual gyrus, also parietal cortices including superior parietal lobule (see Figure 4 and Table 1 for full listing of brain activity inversely correlated with 1/rt).

Fig. 3. Main effect of ANGER (*vs* RELAX) prime in dorsal pons/parabrachial nucleus, an area containing reciprocal relationships with extended central amygdaloid nucleus and hypothalamus, while also encompassing nuclei with ascending noradrenergic projections to midbrain periaqueductal grey and relays of descending autonomic pathways.
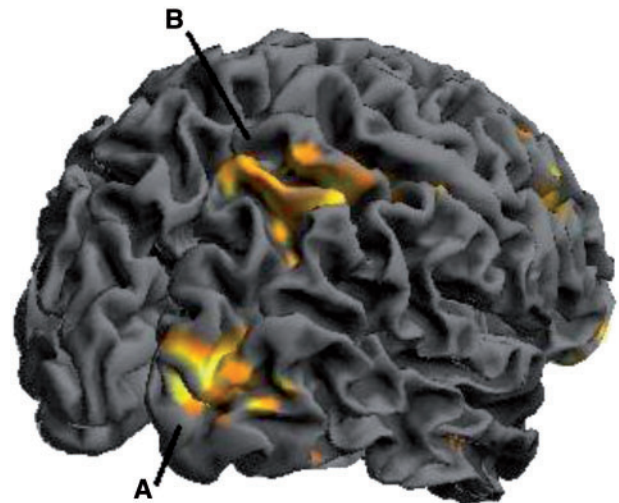

Fig. 4. Brain activity inversely correlated with 1/rt: The impairing effect on reaction time of ANGER relative to RELAX primes was also associated with attenuation of activity across visual areas including (A) cluster peaking the middle occipital gyrus [$40\ -86\ 8$] and (B) right parietal lobe [$34\ -48\ 54$].

When investigating neural activity coupled with elevations in systolic blood pressure, hypoactivation within a number of regions (Table 2).

Cardiac analysis. In Experiment 1 and Experiment 2 respectively, heart rate [$t(17) = -0.05$, $P = 0.96$; $t(13) = -0.94$, $P = 0.37$] and HRV [$t(17) = 0.72$, $P = 0.48$; $t(13) = 1.54$, $P = 0.16$] did not differ between the blocks of ANGER and RELAX primes, suggesting the effects on blood pressure, mediated sympathetically, were associated with suppression of the baroreflex.

Subliminal threshold check

Objective threshold. The mean amount of correct responses in a forced choice test (ANGER prime versus RELAX prime) revealed a mean of 18.1 (SEM = 0.79) correct out of 32 trials (16 would be expected by chance). This did not significantly differ using a 1-way *t*-test.

Table 1. Neural areas correlated with the facilitated RT due to prime

Brain region	Side	Voxels	T-Score	Coordinates(mm)			P-FWE
				x	y	z	
Middle occipital gyrus	R	369	6.12	32	56	−8	0.012
Middle occipital gyrus	R	831	6.09	40	−86	8	<0.001
Cuneus	R		5.82	22	−90	6	
Middle frontal gyrus extending into superior frontal gyrus	R	384	4.89	46	24	30	0.009
Parietal lobe	R	808	4.83	34	−48	54	<0.001

Note: Reciprocal rt was regressed against voxel-wise activity for the contrast RELAX > ANGER, demonstrating brain areas sensitive to the impairing effect of ANGER primes on speeded reaction time. Table thresholded at $P < 0.05$ FWE corrected.

Table 2. Neural activation negatively coupled with systolic activation (i.e. hypoactivation with elevations in systolic blood pressure)

Brain region	Side	Voxels	T-Score	Coordinates(mm)			P
				x	y	z	
Posterior temporal lobe extending into occipital lobe	L	233	6.97	−30	−48	6	0.009
Parietal lobe/Precuneus	R	168	6.14	18	−56	42	0.023
Parahippocampus extending into amygdala/fusiform gyrus	L	158	4.88	−26	−4	−28	0.027
Precentral/Postcentral gyrus	L	224	4.72	−64	−2	22	0.010
Precentral/Postcentral gyrus	R	306	4. 64	58	−8	32	0.004
Cuneus	R	180	3.85	20	−64	20	0.013
Parietal lobe/Precuneus		389	3.78	0	−64	48	0.001

Note: Table thresholded at $P < 0.05$ cluster corrected.

Subjective threshold. When correlating confidence with accuracy for each subject, all correlations were non-significant, with the exception of one participant. This participant did not differ significantly in behaviour from the other participants as their average reaction times for both lexical decisions preceded by ANGER (638 ms) and RELAX (625 ms) were less than one standard deviation (159 and 156 ms, respectively) from the group means (766 and 749 ms). In any case, exclusion of this participant from the principal analysis of reaction time differences between ANGER and RELAX conditions retained significance using both raw reaction time [(t(17) = 2.74, P = 0.014)] and reciprocal reaction time [t(17) = −3.40, P = 0.003].

Discussion

Our data show that the implicit manipulation of autonomic state using affective primes impacts directly on cognitive processes. Specifically, the subliminal processing of ANGER primes, compared to RELAX primes, impairs the speed with which lexical decisions are made and raises systolic blood pressure. Within the brain, this effect of subliminal ANGER primes was associated with enhanced brainstem activation, yet also engendered a widespread relative attenuation of activity across parietal and occipital cortices which mostly paralleled the reaction-time signatures of cognitive impairment.

The degree to which individuals were susceptible to the deleterious effect of subliminal ANGER primes was predicted by the magnitude of their blood pressure response: the magnitude of increased systolic blood pressure evoked by subliminal ANGER correlated with the magnitude of impaired reaction time across participants. Functional neuroimaging revealed that ANGER primes elicited brain activation in pons, an area implicated in the regulation of sympathetic arousal. In addition, ANGER primes attenuated activity within cortical regions including occipitotemporal areas implicated in visual object representations (including orthographic and semantic) and subsections of parietal lobe (implicated in visual attention). Together, these results suggest two principal mechanisms through which subliminal anger can impede decision making: First, through autonomic cardiovascular arousal, indexed by heightened blood pressure, where the extent of anger induced elevations in systolic blood pressure was proportionally detrimental to the semantic decisions as reflected in reaction times. Second, through attention and visual processing areas, where underlying activity within cuneus, occipital gyrus and parietal cortex, normally present or facilitated during relaxed states, is compromised by the covert processing of ANGER.

Anger is an interesting emotion: It is fundamentally negative, yet classified as an 'approach' emotion carrying motivation potential (van Honk and Schutter, 2006; Carver and Harmon-Jones, 2009; Lewis, 2010). Anger can facilitate motoric behaviour (Wilkowski and Meier, 2010), yet in our study the implicit viewing of anger raised mean reaction times to semantic decisions indicating an interference effect at the cognitive semantic level, rather than influencing the tuning of motor responses at a more distal level. Notably such interference is also observed with another negative emotion, fear. Processing of fear stimuli prolongs reaction time if they are presented prior to lexical decisions (Calvo and Castillo, 2005) and interferes with performance on a variety of other tasks including Posner cueing paradigms (Fox *et al.*, 2001), Stroop tasks (McKenna and Sharma, 2004) and rapid serial visual presentation paradigms (Barnard *et al.*, 2005). Both fear and anger convey potential threat, hence the extent to which fear interference also varies with anxiety state (Barnard *et al.*, 2005), complements our facilitation effect of anger as a function of trait anger. These findings indicate that manifestations of specific emotions interact with affective priming to facilitate and/or counteract the behavioural impact. Moreover, the

interference effect afforded by the subliminal ANGER prime in the majority of participants appears to support the notion that implicit negative emotions or 'unconscious stress' may enhance physiological levels in daily life and may even play a role in the stress-health link (Brosschot, 2010; Brosschot *et al.*, 2010). While we observed effects in the majority of participants, future work can determine whether the extent of blood pressure reactivity to emotional primes is influenced by resting blood pressure level.

Meta-analyses suggest motivationally-salient and emotional stimuli increase activation within visual cortices (Phan *et al.*, 2002; Fusar-Poli *et al.*, 2009; Vytal and Hamann, 2010). This effect is coupled to peripheral autonomic (sympathetic) arousal (Critchley *et al.*, 2000; Critchley, 2005). However, in this subliminal priming paradigm, we did not observe a main effect of the (arguably more salient) negative emotion on the level of activation of visual areas. Indeed, we found that for the contrast of ANGER versus RELAX prime conditions, *reduced* activation of visual areas predicted slower reaction times. This finding is nevertheless in accordance with evidence from a body of event-related potential (ERP) literature for diminished attention and visual activation to anger. In an ERP study, attenuated N400 signal over parietal and occipital regions was observed in children in response to vocal anger (Chronaki *et al.*, 2012), denoting anger-induced suppression of an index of early attentional processing. Similarly for emotion scenes, positive affective stimuli are known also to evoke greater shifts in both oxygenated and deoxygenated haemoglobin within occipital cortices than negative or neutral stimuli (Minati *et al.*, 2009). Our findings in relation to anger (and peripheral autonomic arousal) suggest that the reduced activation in visual areas represents both a central signature of, and a likely basis for, compromised perceptual-cognitive processes reflected behaviourally in reaction time.

A major pathway through which the state of cardiovascular arousal influences brain activity (and associated mental processes) is via arterial baroreceptors. These are stretch-sensitive mechanoreceptors which are engaged in the regulation of arterial blood pressure (Rau *et al.*, 1993; Thrasher, 2002). Increases and decreases in cardiac activity (including blood pressure) excite or inhibit baroreceptors. Stable heart-rate across the task indicates that the baroreflex was suppressed during anger trials (Gianaros *et al.*, 2012). The phasic baroreceptor signalling of the timing and strength of each heartbeat has been viewed over the years as predominantly inhibitory to sensory processes and cortical activity (Lacey and Lacey, 1970; Dworkin *et al.*, 1994). While this inhibition is shown reliably for pain processing; facilitatory effects are present for the processing of certain emotions including fear stimuli (Gray *et al.*, 2012; Garfinkel *et al.*, 2014). Even for pain stimuli concurrent baroreceptor activation is associated with greater amygdala engagement (Gray *et al.*, 2009). Thus the regulatory role of baroreceptors on the brain during blood pressure changes are likely to be more complicated than first anticipated (Minati *et al.*, 2009), potentially incorporating structures isolated in our current paradigm, including visual, attentional and memory networks.

Amygdala and insula have been implicated in stress evoked blood pressure reactivity (Gianaros and Sheu, 2009), and across individuals, those who demonstrate exaggerated blood pressure reactivity also demonstrate enhanced neural activation in a variety of areas implicated in stress-related cardiovascular control, such as posterior cingulate cortex and anterior insula (Gianaros *et al.*, 2007). Attention has been shown to modulate blood pressure reactions and their link to neural activity (Okon-Singer *et al.*, 2014). Interestingly, the study by Okon-Singer *et al.* (2014),

which also implemented beat-to-beat blood pressure within an fMRI scanning environment, suggests areas such as the parietal cortex, parahippocampal gyrus and amygdala are associated with blood pressure reactivity in specific emotional-attentional conditions. These results, in combination with the current findings, point to an involvement of blood pressure reactivity with emotional-attentional systems.

These are the first experiments to investigate how subliminal anger influences semantic decisions via physiological mechanisms in both brain and body. We demonstrate a relationship between systolic blood pressure elevations and impeded reaction time in response to subliminal anger primes, as well as reduced activation in visual and attention networks. Anger possesses the combination of approach and adverse affective properties that typically differentiate positively or negatively valenced emotions. We highlight detrimental effects of anger on a specific relatively 'high-level' cognitive process, semantic decision-making; yet we acknowledge and anticipate that anger states will facilitate other types of cognitive and behavioural processes. Future research should thus build upon these findings, to further delineate, in order to predict, the neural and physiological mechanisms through which behavioural and cognitive processes can be both enhanced and impeded by anger.

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