



## Effects of mood induction via music on cardiovascular measures of negative emotion during simulated driving

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### HIGHLIGHTS

- Cardiovascular correlates of negative mood were captured during simulated driving.
- Low activation music reduced systolic reactivity compared to control condition.
- Negative music influenced cardiovascular responses compared to other groups.
- Mood induction via music moderates cardiovascular correlates of negative mood.

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### ABSTRACT

A study was conducted to investigate the potential of mood induction via music to influence cardiovascular correlates of negative emotions experience during driving behaviour. One hundred participants were randomly assigned to one of five groups, four of whom experienced different categories of music: High activation/positive valence (HA/PV), high activation/negative valence (HA/NV), low activation/positive valence (LA/PV) and low activation/negative valence (LA/NV). Following exposure to their respective categories of music, participants were required to complete a simulated driving journey with a fixed time schedule. Negative emotion was induced via exposure to stationary traffic during the simulated route. Cardiovascular reactivity was measured via blood pressure, heart rate and cardiovascular impedance. Subjective self-assessment of anger and mood was also recorded. Results indicated that low activation music, regardless of valence, reduced systolic reactivity during the simulated journey relative to HA/NV music and the control (no music) condition. Self-reported data indicated that participants were not consciously aware of any influence of music on their subjective mood. It is concluded that cardiovascular reactivity to negative mood may be mediated by the emotional properties of music.

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### 1. Introduction

The experience of negative affect has been associated with coronary heart disease (CHD) [27], particularly for those with high trait negative affectivity [1]. Adverse cardiac events linked to CHD have been associated with anxiety [37], depression [5] and anger [35]. The precise impact of each category of negative affect on CHD is complicated by overlap between affective constructs and their related measures [36].

Cardiovascular markers may be used to measure the state of heightened sympathetic activation deemed to be a potential pathway between negative affect and CHD [36]. However, considerable overlap exists between the types of cardiovascular change that are deemed to

characterise distinct types of negative affect. The experience of anger, for example, is typified by elevated sympathetic activation coupled with increased respiration whereas a similar pattern (increased sympathetic activation, faster/shallower breathing) is associated with increased levels of anxiety [13]. The cardiovascular response to anger induction was correlated with increased heart rate and systolic blood pressure with a reduction of the pre-ejection period (PEP) [32]. The same pattern of cardiovascular activation has been associated with the experience of anxiety during preparation for public speaking [38] and performance of mental arithmetic [39]. It is likely that commonality between cardiovascular manifestation of negative affect is directly influenced by heightened sympathetic activation of the autonomic nervous system.

Driving represents a common activity in everyday life where the experience and expression of negative affect have implications for health and safety [25,41]. For example, the expression of anger is associated with undesirable behaviour on the road, such as physical and verbal

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abuse, speeding, and tailgating [4,19]. Similarly, increased frequency of violations, errors and lapses has been reported for drivers with high levels of trait anxiety [25]. The trigger for anger or anxiety may originate from unexpected or erratic actions from other road users or be provoked from factors associated with the traffic environment, e.g. stop lights and traffic congestion [17]. For instance, early research using ambulatory monitoring revealed an association between high congestion and elevated systolic blood pressure [24,34] due to a loss of control over journey schedule [10].

The experience of negative emotion and its cardiovascular manifestation may be moderated by environmental factors. Music is one of the most potent techniques for mood regulation and has been found to ameliorate a variety of negative moods [30,42,43]. There is also evidence that people tend to use music to regulate mood states when they are alone [30]. Previous studies have indicated that music is capable of inducing and sustaining positive mood states during the driving task [40]. In this study, participants were exposed to positive and negative music during low- and high-demand traffic conditions and results indicated that positive mood was sustained in the presence of (positive) music despite high traffic density and negotiation of narrow lanes. In addition, the 2000 study by Wiesenthal et al. reported that music (compared to no music) can alleviate stress and reduce aggression during high congestion drives.

Previous investigations into the influence of music on cardiovascular activity have yielded mixed results. Several studies reported that arousing music tended to increase heart rate whilst sad music had the opposite effect [6,11]. However, other researchers claimed that the presence of music per se tended to increase heart rate [12,22]. These contradictory findings may stem from a number of methodological variations across the different studies, e.g. the context of music listening (music in the background or as primary activity), the duration of the musical piece, and the interaction between musical stimuli and personal taste. The categorisation of emotional states may be described as a series of distinct categories (happiness, anger etc.) or within the context of a multidimensional space; for example, the circumplex model [23] proposed two orthogonal dimensions of activation (high vs. low) and valence (positive vs. negative) that encompass a wide range of positive and negative emotional states. A number of studies have studied the impact of different types of music to induce specific moods within the context of the circumplex model. For example, Nykliček et al. [18] recorded cardiovascular psychophysiology from participants who listened to music deemed to represent each of the four quadrants of the circumplex model of emotion [23], i.e. high activation/positive valence (HA/PV), high activation/negative valence (HA/NV), low activation/positive valence (LA/PV), and low activation/negative valence (LA/NV). They reported that Respiratory Sinus Arrhythmia (RSA) was lowest during happiness and agitation compared to sadness serenity or a white noise condition. Nykliček and colleagues also reported that left ventricular ejection time (LVET) and PEP increased in the presence of noise and during sad compared to happy music. Krumhansl [14] captured subjective and physiological analysis to various forms of mood music. She reported that heart rate decreased and blood pressure increased during sad music. It is apparent that mood music has an effect on the cardiovascular response but the precise impact is determined by the methodological context.

The current study was designed to investigate the impact of music on the cardiovascular manifestation of negative affect during a simulated driving task. Participants were exposed to an unavoidable delay during a simulated driving journey with a fixed time schedule in order to induce negative affect. This protocol was validated in a previous study conducted by Fairclough and Spiridon [7]. The current study supplemented this protocol by exposing participants to four different categories of music (HA/PV, HA/NV, LA/PV, LA/NV) plus a no-music control group. Our main aim was to investigate how the dimensions of the musical pieces (activation and valence) systematically influenced cardiovascular correlates of negative affect within the context of an

unavoidable delay during the simulated journey. We expected HA/NV music to augment cardiovascular reactivity whereas LA/PV music was presumed to have the opposite effect. Our primary goal was to explore whether changes in activation or valence induced by the music were sufficient to enhance or mitigate cardiovascular reactivity to the experience of negative affect in comparison to the control condition.

## 2. Method

### 2.1. Participants

The study used a between-subject design with each group including 20 volunteers in total (10 males, 10 females) amounting to 100 participants in total. The mean age of the participants was 21.2 years ( $SD = 4.7$  years, see Table 1). Each person received a £10 voucher (\$15/12€) for participation. Two measures of trait anger were taken from the State-Trait Anger Expression Inventory-2 (STAXI-2) [31]: Trait Anger-Reaction (T-Ang/R) and Trait Anger-Temperament (T-Ang/T) to assess any between-group differences in susceptibility to anger. The former refers to the frequency with which anger is experienced during frustration or negative evaluation, whereas the latter represents the disposition of the individual to experience anger in the absence of provocation.

A number of statistical analyses were conducted to test for trait differences between the five independent groups of participants. An ANOVA on age revealed no significant differences between the participant groups. A  $5 \times 2$  MANOVA was conducted on both trait anger scores (T-Ang/R and T-Ang/T) with null findings (Table 1 for descriptive statistics).

### 2.2. Simulated driving task

A simulated car journey was prepared using a STI SIM Driving Simulator software (STI Inc.). This PC-based software allowed interaction via a steering wheel/pedal console and the driving scene was projected onto a large screen (approx.  $3.66 \text{ m} \times 4.57 \text{ m}$ ) yielding a visual angle of approx.  $80^\circ$ . The simulated journey consisted of a two-lane roadway passing through countryside and urban settings. The route was created so that it could not be finished by the participants. Therefore, after 12 min the drive was stopped.

A clock was visible next to the simulated scene and participants were instructed to complete the journey within 8 min in order to earn the £10 payment for participation in the study. If they failed to complete the journey within this schedule, they were instructed that they would not be paid. This deadline was presented to the participants within the context of a scenario where they were told that the purpose of the journey was to collect a child from school (however, they were not told in advance that it was impossible to make the journey on time). If the driver crashed the vehicle more than twice, they were told that they would lose 70% of their total participant payment. In addition, speeding warnings were in operation (a police siren sounded if the speed limit was exceeded) and participants were informed that they

**Table 1**

Trait variables for each participant group including means and standard errors ( $N = 100$ ). Note: HA/PV = high activation/positive valence, LA/PV = low activation/positive valence, HA/NV = High activation/negative valence and LA/NV = low activation/negative valence. Gender represented as a ratio of males:females.

	Age (M, SE)	Gender	T-Ang/R Mean (SE)	T-Ang/T Mean (SE)
HA/PV	20.79 (1.07)	9/11	2.05 (.13)	1.53 (.61)
LA/PV	20.65 (1.04)	10/10	2.08 (.13)	1.53 (.77)
HA/NV	20.95 (1.04)	10/10	2.01 (.13)	1.61 (.72)
LA/NV	20.15 (1.04)	10/10	1.96 (.13)	1.46 (.37)
No music	23.45 (1.04)	10/10	2.42 (.13)	1.61 (.64)

would be fined by £2 (approx. \$3) on every occasion that they broke the speed limit.

The driver encountered a low level of traffic density in both lanes until approximately 4 min (dependent on the driving speed) of the journey had elapsed, at this point participants encountered a traffic jam where extremely slow moving traffic in the lane ahead was combined with high density traffic in the opposite lane, hence participants remained 'trapped' with no opportunity to overtake for a period of 2 min. The drive then continued with varying levels of traffic intensity, and the journey was stopped after 12 min.

All instructions to participants were presented in a written form and it should be noted that threats to withhold participant payment or enforce speeding fines were a deception and all participants were fully debriefed and paid the full amount once the experiment had been completed. The University Ethics Research Committee approved this protocol and all associated methods prior to data collection.

### 2.3. Experimental design

The study employed a mixed design where music group functioned as a between-subjects factor whilst induction and exposure to the traffic jam were manipulated on a within-subject basis. Participants were randomly assigned to one of five groups, a control group who were not exposed to any music and four groups who heard four types of music during the induction and the simulated drive; the four types of music were designed to induce different mood states in the individual, these were: HA/PV = high activation/positive valence, LA/PV = low activation/positive valence, HA/NV = high activation/negative valence, LA/NV = low activation/negative valence.

### 2.4. Dependent variables

#### 2.4.1. Self-report questionnaires

The State scale from the STAXI-2 [31] was administered to participants before and after the simulated journey. This scale is designed to capture the subjective experience of anger by measuring participant responses on a 4-point Likert scale to a series of 15 statements; the score varies from 15 to 60 and reliability is high (0.85). Participants completed two scales (energetical arousal, hedonic tone) to represent activation and valence from the Mood Adjective Checklist developed by Matthews et al. [16] before and after the simulated journey. Both components are captured by 5-point Likert scales in response to eight items and scores range from 5 to 20; the reliability scores for energetical arousal and hedonic tone are 0.78 and 0.89 respectively.

#### 2.4.2. Measures of cardiovascular reactivity

Cardiovascular activity was recorded using band electrodes (each containing two electrodes) placed on the back of the neck and the thorax [26]. These signals were processed via the NICO100C Noninvasive Cardiac Output Module (BIOPAC Systems Inc.) in conjunction with the MP150 data recording system (BIOPAC Systems Inc.). This module delivered impedance magnitude ( $Z_o$ ) and derivatives ( $dZ/dt$ ) at 1000 Hz. The impedance signals were analysed using an algorithm<sup>1</sup> developed

<sup>1</sup> An algorithm was developed to detect significant points (e.g. Q, B, X) in the impedance signals and ECG based on existing research (e.g. [27]). This algorithm provided a calculation for impedance measures based on the Kubicek formula. The C point was defined as the maximum point in the  $dZ/dt$  signal in a time window 60–200 ms from the R peak, the X point was defined as the minimum point over the course of the cycle after the C point whereas B was set as the maximum derivative of the  $dZ/dt$  signal in a time window 150–100 ms before the C point. The algorithm was validated using 10 min baseline data from the current study from 20 participants. These data were scored manually by a trained experimenter and compared to the results from algorithm. With respect to LVET times, the mean deviation between the manual and computerised scores was 82 ms (SD = 30 ms; range = 23–110 ms). For PEP, the mean deviation was 25 ms (SD = 23 ms; range = 5–79 ms). A correlation was conducted across the whole data set to assess PEP between manual scoring vs. computerised analysis; we found that scores were highly correlated ( $r = 0.89$ ).

in our own laboratory in order to detect the following measures for each cardiac cycle: Pre-ejection period (PEP), cardiac output (CO), stroke volume (SV), and left ventricular ejection time (LVET).

The Inter-Beat Interval (IBI) from the heart was calculated from an ECG signal filtered between 0.5 and 35Hz and sampled at 1000Hz. This signal was collected via a two-lead electrode sensor connected to the TEL100C data capture signal (BIOPAC Systems Inc.) that also worked with the MP150 system (i.e. the earth signal for the ECG was obtained from the cardiovascular impedance apparatus).

Blood pressure was measured using a CARESCAPE Vital Signs Monitor (V100) (DINAMAP Inc.) which involved placement of an inflatable cuff on the upper left arm. Readings of systolic blood pressure, diastolic blood pressure, heart rate and mean arterial pressure were all obtained using the oscillometric method. Recordings from the CARESCAPE were taken three times during the journey and the first measurement coincided with the middle point of the traffic jam. Total peripheral resistance (TPR) was calculated by combining information from the CARESCAPE and the NICO100C, i.e.  $TPR = MAP/CO * 80$ .

### 2.5. Procedure

The experiment consisted of two distinct sessions, one concerned with the selection of music for use during the test session and the simulated drive itself. During the former, all participants were exposed to a selection of music via a website and asked to deliver ratings in order to create four music groups where the selection of music could be personalized to the individual. The second session took approximately 60 min in a laboratory and involved a baseline measurement, a mood induction section (when participants were exposed to music only) and the simulated driving task.

#### 2.5.1. Music selection

The selection of music designed to induce specific mood in the participants was personalized for each individual via an online survey exercise. Participants were asked to rate activation and valence for 80 pieces that had been preselected to vary with respect those dimensions. The classification of the music with respect to these labels was performed using an algorithm that automatically classified music into the four mood classes of the valence–activation model using 83 music features derived from the audio signal (e.g., tempo and mode) [28,29]. The initial music database comprised 1800 songs and included a large variety of pieces ranging from classical music to popular radio music. The 80 pieces that best fitted the four mood dimensions according to the automatic classification were selected for the participant to rate. Participants were asked to listen to each of these pieces at different places within the music to get a general impression of the piece and to rate the level of activation (high to low) and valence (unpleasant to pleasant) using 7-point Likert scales. For each participant six music pieces were selected (i.e., two songs were used for mood induction and four songs were presented during the simulated drive). These were the pieces with the most prototypical scores for a specific quadrant and that had been given the same label following the automatic classification, i.e. for a participant in the HA/PV condition songs with the maximal scores for valence and activation were selected, for those in the LA/NV, those songs with minimal ratings on both dimensions were selected. Table 2 shows four example songs of each music mood dimension to illustrate the variation of the songs. The duration of the selected songs was shortened to 3 min each using Audacity software (version 1.2.6). The volume of the music was fixed, being the same for each participant. The music was presented via two speakers that were positioned in the simulator room and were positioned left and right in front of the participant. The location of two speakers mimicked the location where car music would come from in a car.

**Table 2**

A selection of 4 songs from each of the music categories that were selected for the participants. Note: HA/PV = high activation/positive valence, LA/PV = low activation/positive valence, HA/NV = high activation/negative valence and LA/NV = low activation/negative valence.

HA/NV	HA/PV	LA/NV	LA/PV
Euromasters – everybody clap your hands	Corona – rhythm of the night	J Brahms: Rias Kammerchor – Vergangen ist mir Glueck und Heil	Dinah Washington - what a difference a day makes
Slipknot – wait and bleed	Deep Blue Something – breakfast at Tiffany	Hans Zimmer – the house of spirits	The Temptations - just my imagination
Linkin Park – one step closer	Depeche mode – just can't get enough	Rachmaninoff: Vespers – now let thy servant depart	Jay Ungar & Molly Mason – bound for another harvest home
Pantera – goddamn electric	Kate Nash – foundations	Townes Van Zandt – silver ships of Andilar	Moriarty – Jimmy

### 2.5.2. Simulator trial

In the lab, participants were randomly assigned to one of the five experimental groups. A timeline is provided in Fig. 1 to illustrate the sequence of events and collection of self-reported questionnaire data. After providing informed consent and being seated in the simulator, participants were allowed a short practise drive before the psychophysiological apparatus was attached. Once all signals had been checked, a baseline period commenced and participants were asked to relax and watch a neutral video for 6 min [20]. The data acquired during this time provided the baseline measurements. Participants were then asked to complete the STAXI and the mood scale before listening to the music presented for 6 min; this was the music mood induction session. To ensure that the participants paid attention to the music they were told that questions would be asked about the music at the end of the experiment (this requirement was not applied to the control condition where participants sat in silence during the induction phase). The music mood induction condition ensured that the participants were in the targeted mood from the start of the drive onward. After music induction, participants completed the state anger scale and the mood scale and were presented with written text describing the driving scenario. If the participants had no further questions the drive began. The state anger and mood scales were presented for completion following the simulated drive. Finally, there was a recovery period in which the participants were given the debriefing form and the money voucher.

### 2.5.3. Statistical analysis

All statistical analyses were conducted using SPSS V.12 using multivariate statistical testing (ANOVA and MANOVA). The alpha level for significance was  $p = <.05$  and violations of sphericity were examined using the Mauchly's Test; if this test was significant, the degrees of freedom for that test were adapted via the Greenhouse-Geisser adjustment. Effect sizes are also included calculated as eta-squared, which provides a representation of the amount of variance in the data explained by the effect, i.e.  $0.2 = 20\%$  of variance. The Bonferroni test was used for post-hoc analysis of significant main effects. All data were examined for the presence of outliers (defined as any value  $+/- 3$  SD from the mean) and outliers were omitted from statistical testing.

## 3. Results

Data was derived from self-reported questionnaires and measures of cardiovascular reactivity. The analysis of the former is included in the first section of the results and relied principally on MANOVA analyses to test the effects of music and the simulated drive on subjective

anger and mood. The analysis of cardiovascular reactivity is divided into two sections, an initial test of between-participant differences at baseline and a series of  $5 \times 2$  ANOVA models where music (HA/PV, HA/NV, LA/PV, LA/NV, no music) and test phase (induction, drive) functioned as independent variables.

### 3.1. Analysis of self-reported questionnaire data

State anger was captured using the STAXI where anger is captured with reference to three sub-scales: (a) feelings of anger, (b) feel like expressing anger verbally and (c) feel like expressing anger physically. All three STAXI variables collected after the baseline (STAXI 1 in Fig. 1) were subjected to a  $5 \times 3$  MANOVA to check for differences between the participant groups at baseline and no significant differences were found. Change scores were derived by subtracting post-baseline STAXI scores (STAXI 2 in Fig. 1) from post-induction score (STAXI 1 in Fig. 1) values to capture the effect of music only, i.e. a positive number is associated with increased anger during the music mood induction. These data were subjected to an analysis that revealed a main effect for music ( $F(4,95) = 5.02, p < .01, \eta^2 = .18$ ); post-hoc tests indicated that total anger (a combination of feelings, verbal anger and physical anger) was significantly higher in the HA/NV condition ( $M = 0.19$ ) compared to: HA/PV ( $M = -.03$ ), LA/NV ( $M = .01$ ), LA/PV ( $M = .02$ ) and no music ( $M = .02$ ).

A series of change scores were created for the simulated drive by subtracting post-drive STAXI scores (STAXI 3 in Fig. 1) from post-induction scores (STAXI 2 in Fig. 1). All three scales were subjected to a  $5 \times 3$  MANOVA (Group  $\times$  State Anger). This analysis revealed a significant effect for Group with respect to feelings of anger ( $F(4,94) = 4.15, p < .01, \eta^2 = .15$ ) and feel like expressing anger verbally ( $F(4,94) = 3.36, p < .05, \eta^2 = .13$ ). There was no significant effect for the scale related to expression of physical violence. All descriptive statistics are presented in Table 3. Post-hoc comparisons revealed that feeling angry scores were significantly higher for the HA/NV group relative to all other groups ( $p < .05$ ). A post-hoc analysis of the verbal anger scale indicated higher scores for the HA/NV group compared to all groups except for those in the Control (no music) group.

Two unidimensional scales from the UMACL, activation (energetical arousal: active vs. tired) and valence (hedonic tone: happy vs. sad) were converted to change scores, i.e. post-test minus pre-test during both the music induction (Mood 2 – Mood 1 in Fig. 1) and driving (Mood 3 – Mood 1 in Fig. 1) parts of the test session. These data were analysed as a manipulation check that four categories of music had the predicted effects on subjective mood. A  $5 \times 2 \times 2$  MANOVA was conducted on these data (Group  $\times$  induction/driving  $\times$  Act/Val),

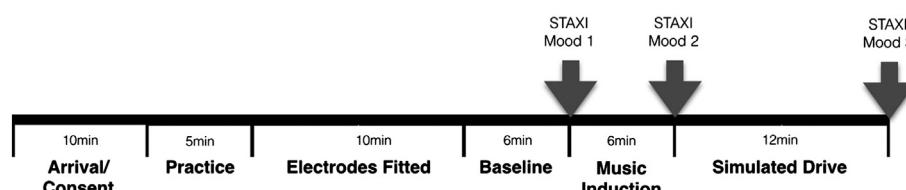


Fig. 1. Timeline of events during the simulator trial.

**Table 3**

Means and standard errors for three sub-scales of the STAXI-2 based on baselined scores (post\_drive minus pre\_drive) (N = 100).

Group	Feeling angry	Verbal anger	Physical anger
HA/PV	0.62 [.11]	0.21 [.11]	0.07 [.06]
LA/PV	0.39 [.12]	0.05 [.11]	0.01 [.06]
LA/NV	0.60 [.12]	0.25 [.11]	0.08 [.06]
HA/NV	1.05 [.12]	0.57 [.11]	0.18 [.06]
Control	0.67 [.12]	0.32 [.11]	0.09 [.06]

which revealed significant interaction effects for Group  $\times$  Act/Val ( $\Lambda(4,95) = .81$ ,  $p < .01$ ,  $\eta^2 = .19$ ), induction/driving  $\times$  Act/Val ( $\Lambda(1,95) = .44$ ,  $p < .01$ ,  $\eta^2 = .57$ ) and a three-way interaction ( $\Lambda(4,95) = .83$ ,  $p < .01$ ,  $\eta^2 = .17$ ). Post-hoc analyses (Bonferroni tests) revealed that activation was significantly higher for HA/PV and HA/NV groups compared to all other groups during the music induction session ( $p < .01$ ). The post-hoc analysis of valence during music induction revealed that both negative valence categories of music (HA/NV and LA/NV) resulted in significantly lower valence scores compared to all other groups ( $p < .01$ ), i.e. lower valence scores are indicative of negative affect. It was also found that activation significantly increased and valence decreased during the simulated drive compared to the music induction session ( $p < .01$ ). By contrast, the analysis of subjective mood data during the driving task failed to reveal any significant differences between the five groups of participants. All descriptive statistics for the mood data during both music induction and the simulated drive are illustrated in Fig. 2.

### 3.2. Analysis of cardiovascular reactivity data

Baseline cardiovascular data was tested via MANOVA to test for a priori differences between the five participant groups. Data from 6 to 9 participants were lost due to erroneous data resulting from excessive movement during the baseline phase. There were no significant differences between the five groups at baseline for any of the cardiovascular variables and all descriptive statistics are presented in Table 4.

Cardiovascular data was extracted from three periods: baseline (Fig. 1), music induction (Fig. 1) and the 2 min period of the simulated drive when participants encountered the traffic jam. A set of baselined data were created for music induction (MI) and traffic jam (TJ) where cardiovascular measures at baseline were subtracted from measures obtained during MI and TJ, i.e. positive numbers are equated with an increase of each measure during MI and TJ. The two phases of the experiment, MI and TJ, were collectively labelled as the test phase and all cardiovascular data were subjected to  $5 \times 2$  ANOVA (Group  $\times$  test phase) testing.

The presence of movement artefacts in the data resulted in the removal of six participants from the blood pressure data. The analysis of systolic reactivity (SBP) yielded main effects for both Group ( $F(4,89) = 4.08$ ,  $p < .01$ ,  $\eta^2 = .34$ ) and test phase ( $F(1,88) = 19.54$ ,  $p < .01$ ,  $\eta^2 = .17$ ). Post-hoc Bonferroni tests indicated that systolic reactivity was significantly reduced for the LA/PV and LA/NV groups compared to both HA/NV and Control groups ( $p < .01$ ). This effect is illustrated in Fig. 3. The main effect for test phase indicated that systolic reactivity was highest during TJ ( $M = 6.17$ ,  $s.e. = 0.95$ ) compared to MI ( $M = 1.81$ ,  $s.e. = 0.49$ ) ( $p < .01$ ). The same ANOVA model was applied to diastolic reactivity (DBP). This analysis yielded a significant effect for test phase ( $F(1,89) = 14.91$ ,  $p < .01$ ,  $\eta^2 = .13$ ); as with SBP, diastolic reactivity was significantly higher during TJ1 ( $M = 4.08$ ,  $s.e. = 0.92$ ) compared to MI ( $M = 0.65$ ,  $s.e. = 0.38$ ) ( $p < .01$ ).

A number of participant data records from the ECG and impedance data were corrupted due to movement artefacts in the driving simulator environment; this loss of data resulted in only 81 participants being eligible for subsequent data analysis of heart rate and all markers of cardiovascular impedance (PEP, CO, LVET, SV, TPR), i.e. 16 participants

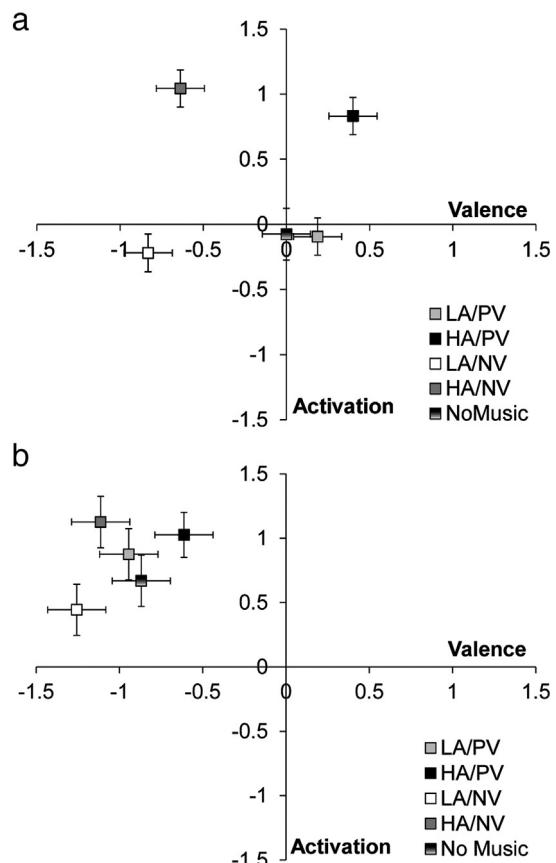


Fig. 2. Self-reported mood data (activation and valence) during music induction (a) and simulated drive (b) for all conditions (N = 100).

remained in all groups with the exception of the HA/PV group where data for 17 participants were available.

The analysis of baselined heart rate (extracted from ECG trace) revealed a significant effect for Test Phase ( $F(1,80) = 6.58$ ,  $p < .05$ ,  $\eta^2 = .09$ ). Post-hoc tests indicated that baselined heart rate was significantly higher during TJ ( $M = 3.85$ ,  $s.e. = 1.54$ ) compared to MI ( $M = -0.17$ ,  $s.e. = 0.92$ ). The same ANOVA model was applied to the baselined values of PEP, which also revealed a significant main effect for Test Phase ( $F(1,80) = 16.30$ ,  $p < .01$ ,  $\eta^2 = .18$ ), i.e. baselined PEP was significantly reduced at TJ ( $M = -11$  ms,  $s.e. = 3$  ms) compared to MI ( $M = 1$  ms,  $s.e. = 2$  ms). There was evidence of an interaction effect for Cardiac Output ( $F(4,77) = 2.69$ ,  $p < .05$ ,  $\eta^2 = .13$ ); post-hoc testing indicated an increase of CO from induction ( $M = -0.51$ ,  $s.e. = 0.13$ ) to the experience of the traffic jam ( $M = 0.76$ ,  $s.e. = 0.15$ ) but only for the HA/NV group.

The ANOVA analyses of left ventricular ejection time (LVET), stroke volume (SV) and total peripheral resistance (TPR) failed to reveal any significance effects from either Group or test phase.

### 4. Discussion

The experiment was designed to induce negative affect in participants by impeding their journey schedule during a simulated drive. The protocol was successful in this respect where participants indicated a mood state that coupled high activation with negative valence as illustrated in Fig. 2b. The cardiovascular changes observed during the traffic jam (increased blood pressure, higher heart rate, reduced PEP) indicate an increase of sympathetic activation, which was found to be indicative of anger in one earlier study [32]. However, the pattern of subjective mood and cardiovascular psychophysiology observed during the protocol are consistent with anxiety in response to a perception of failure

**Table 4**

Mean and standard errors for all cardiovascular variables during the baseline session: SBP (systolic blood pressure), DBP (diastolic blood pressure), HR (heart rate), PEP (pre-ejection period), LVET (left ventricular ejection time), SV (stroke volume), CO (cardiac output), TPR (total peripheral resistance).

Group (units)	SBP mmg/Hg	DBP mmg/Hg	HR bpm	PEP ms	LVET ms	SV ml	CO L/min	TPR dyne-s * cm <sup>-5</sup>
HA/PV	118.50 [2.49]	75.07 [1.56]	74.98 [2.71]	108 [.03]	298 [.01]	45.93 [9.06]	3.45 [.68]	1603.56 [292.31]
LA/PV	120.18 [2.70]	78.24 [1.69]	75.59 [2.94]	105 [.03]	309 [.01]	65.22 [9.06]	4.85 [.68]	1123.44 [292.31]
HA/NV	121.97 [2.56]	78.54 [1.60]	71.67 [2.78]	116 [.03]	316 [.01]	48.11 [9.06]	3.16 [.68]	1540.35 [317.06]
LA/NV	122.17 [2.63]	79.56 [1.64]	75.28 [2.86]	149 [.03]	302 [.01]	54.17 [9.06]	3.85 [.68]	1177.06 [299.91]
Control	120.70 [2.49]	77.53 [1.56]	68.75 [2.71]	141 [.03]	322 [.01]	43.49 [8.82]	3.77 [.66]	1655.50 [292.31]
N	94	94	94	91	91	91	91	91

(being unable to complete the journey on schedule) and the impact of real-world consequences (the threat of not being paid for their participation), which was equally capable of inducing states of anxiety and fear as well as states closely related to anger, such as frustration and annoyance. The experiment was not designed to discriminate between different categories of negative emotion, such as anger and fear [32,33], and the specificity of our manipulation suffered as a result.

It was hypothesised that HA/NV music would enhance cardiovascular reactivity during the simulated journey whilst LA/PV music would have the opposite effect. We found no evidence for the former; levels of systolic reactivity during HA/NV music were similar to those observed during the no-music control condition (Fig. 2). In addition, none of the cardiovascular parameters significantly increased due to the presence of HA/NV music with respect to the control group. It was found that cardiac output significantly rose during the traffic jam compared to music induction only for the HA/NV group. This finding suggests that prior exposure to HA/NV music during the induction session may have primed the cardiovascular system to enhance blood flow during the traffic jam as this effect was not observed in any of the other four groups.

The second hypothesis concerning the capacity for LA/PV music to reduce cardiovascular reactivity was generally supported but with a number of caveats. The first proviso concerns the fact that only one cardiovascular parameter (systolic reactivity) was significantly reduced

in the presence of LA/PV music compared to the control condition and all other parameters remained unaffected. Secondly, it is important to note that LA/NV music caused a similar reduction of systolic reactivity as LA/PV (Fig. 1) and therefore it was the activation value of the musical piece rather than valence that was responsible for a reduction of systolic reactivity. It is known that the force of myocardial contraction is the main influence on systolic reactivity, which is governed primarily by the level of beta-adrenergic activity in the autonomic system [8]. The same trend was observed in the PEP data, which is widely regarded as the most robust marker of beta-adrenergic activation [21,26], but this trend did not reach statistical significance. The sensitivity of systolic blood pressure to the activating properties of the music seems to suggest that an observed effect was chiefly mediated by beta-adrenergic activation.

With respect to subjective self-assessment, we found that the presence of HA/NV tended to increase feelings of anger (Table 2) compared to other conditions; however, this finding may reflect demand characteristics as the prevailing mood of the musical piece is inevitably perceived by participants. The ability of the music to influence subjective mood states in the expected direction was demonstrated during the music induction task (Fig. 2a) but between-group differences were rendered insignificant by the simulated drive (Fig. 2b), presumably because the primacy of the simulated drive neutralized the subjective impact of music on mood.

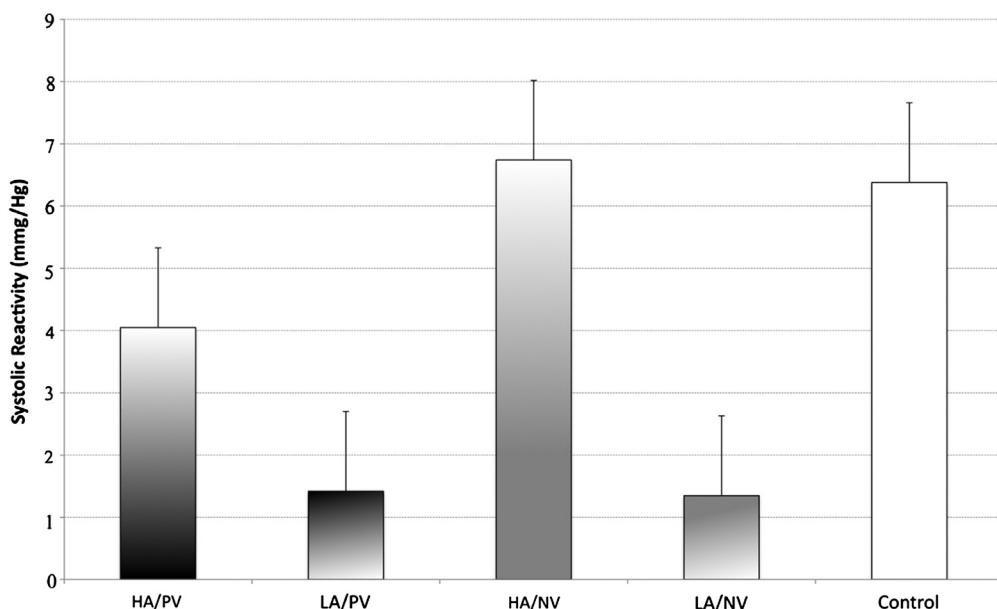


Fig. 3. Systolic reactivity (SBP) for all five groups averaged across both test phases (MI, TJ) (N = 98).

The study demonstrated that relative changes in systolic reactivity in response to the simulated traffic jam were mitigated by music characterised by low levels of psychological activation. The absence of any corroboration of this physiological effect in the subjective self-report data could be a product of different time periods associated with measurement (subjective measures were collected when the simulated drive was over) or it may suggest that music exerted an influence at the hypothalamic level, which is not perceived consciously by the individual; see Brosschot [3] and Lovallo [15] for similar theories related to the physiological impact of stress. This pattern of altered psychophysiology combined with no significant changes in subjective ratings was observed during an earlier study [40]. The dissociation between subjective mood and psychophysiology corresponds to theories where fluctuations in mood must pass a certain level of intensity and saliency before they are consciously noticed [9]. A secondary implication of the current work is that an environmental intervention, such as music, may be deployed to subconsciously alter cardiovascular reactivity to negative mood states. The exploration of conscious and unconscious routes to anger reduction is one possible avenue for further research.

With respect to methodological issues, the protocol [28,29] that enabled personalization of music choice to specific individuals in order to produce a desired effect on mood was validated, i.e. effects of music on subjective mood were as anticipated during induction session (Fig. 2a). A negative mood state was provoked during the study by a specific event (traffic jam) within the context of a long-term challenge, i.e. to complete the journey on schedule and avoid a financial penalty. The negative mood state experienced by our participants during the simulated journey resulted from both short-term boredom/frustration/annoyance/anger at impedance to the journey and implication of this delay with respect to the longer-term goal of completing the journey on schedule, which may have provoked a sense of fear and anxiety. Further research is required to explore the relative contribution of individual elements within the simulated journey to cardiovascular reactivity in order to define the type of emotion experienced by participants with greater specificity. For example, future studies that manipulate short-term factors (presence of traffic jam vs. no traffic jam) and long-term goals (e.g. possibility of financial punishment, presence of a journey schedule) may help elaborate the relationship between cardiovascular reactivity and different categories of negative mood states in greater detail. The design of the current experiment does not permit us to draw strong conclusions about the effect of the traffic jam per se because this factor was not systematically manipulated. It would also be useful to explore how different types of traffic event influence those cardiovascular responses such as comparing an impersonal event (traffic jam) with a personal one (unsafe and inconsiderate behaviour of other road users). This particular study used threat of punishment as one means of inducing negative mood and future work could explore whether the availability of an additional reward had the same effect in this scenario. With respect to the applied work, it would be interesting to explore the effect observed in the current study with respect to the impact of music on cardiovascular measures associated with negative mood states that can generalise to other domains of life [2]. As this study took place in a simulated environment, which represents a simplification of the real driving environment, there is a question over the ecological validity of our findings. For example, systolic reactivity during the traffic jams was high for the control group (Fig. 3) and it is possible that a relative paucity of multisensory stimulation in our experiment may have (a) exaggerated the impact of music on cardiovascular reactivity, and (b) amplified the aversive experience of the traffic jams for participants in the no-music control condition.

To summarise, the study demonstrated that low activation music, regardless of valence, was found to reduce systolic reactivity associated with a state of high-activation and negative valence albeit in a simulated driving environment. Further research is required to explore whether this finding can be replicated in the field and in other domains of anger induction.

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