Available online at www.sciencedirect.com
ScienceDirect
International Journal of Psychophysiology 64 (2007) 157–164
INTERNATIONAL JOURNAL OF PSYCHOPHYSIOLOGY
www.elsevier.com/locate/ijpsycho

Sensorimotor gating and anxiety: Prepulse inhibition following acute exercise

Aaron R. Duley [a,*], Charles H. Hillman [b], Stephen Coombes [c], Christopher M. Janelle [c]

[a] University of Central Florida, Institute for Simulation and Training, 3100 Technology Parkway, Orlando, FL 32826, United States
[b] University of Illinois Urbana-Champaign, United States
[c] University of Florida, United States

Received 29 August 2006; received in revised form 6 December 2006; accepted 24 January 2007
Available online 2 February 2007

Abstract

This investigation examined whether gating related deficits among individuals with high trait anxiety could be moderated by an acute bout of exercise. Low (LA) and high (HA) trait anxious participants engaged in either a quiet rest or an exercise session on separate occasions. Replicating previous findings, HA participants exhibited significantly reduced PPI at lead intervals of 30 and 60 ms relative to LA controls. HA and LA participants were also found to occasion similar PPI following exercise relative to quiet rest. This finding was found to be independent of the order in which quiet rest or exercise occurred, and was not a function of differences in raw startle blink amplitude between sessions. The current results highlight the potential for PPI to index the potential anxiolytic effects of an acute exercise bout.

Keywords: Anxiety; Prepulse inhibition; Startle; Exercise

1. Sensorimotor gating and anxiety: prepulse inhibition following acute exercise

Prepulse inhibition (PPI) refers to the unlearned attenuation in startle reflex magnitude observed when a startling stimulus (a pulse) is closely preceded by a transient sensory event (a prepulse or lead stimulus) relative to conditions where the pulse is presented in isolation (Graham, 1975; Hoffman and Ison, 1980). The temporal window for prepulse–pulse pairing to observe PPI ranges from approximately 30 to 500 ms prior to pulse presentation, with maximum reflex inhibition occurring between 100 to 150 ms for an acoustic lead and startle stimulus when measured electromyographically underneath the eye (for reviews, see Blumenthal, 1999; Braff et al., 2001; Filion et al., 1998). In addition to startle modulation via acoustic–brainstem pathways, PPI appears across a combination of sensory channels, including visual and tactile (Fendt et al., 2001; Filion et al., 1998), making it a powerful tool to study the convergence of different sensory pathways on common psychological phenomena (Berg and Balaban, 1999). PPI has been observed in several infrahuman species, providing great flexibility in experimental protocol and design (Braff et al., 2001; Hoffman, 1997). These translational opportunities, taken together with contemporary knowledge concerning the basic neural and chemical substrates involved in the primary startle pathway (Davis et al., 1999; Koch, 1999; Koch and Fendt, 2003), has permitted the robust employment of PPI to evaluate both the physiological components of cognition, and the pharmacological effectiveness of chemical compounds used to treat certain neuropsychiatric disorders (Swerdlow and Geyer, 1999; Swerdlow et al., 2001).

Graham (1975) postulated that PPI reflects the effectiveness of a protective mechanism, instantiated in the early processing of lead stimulus recognition that invokes inhibitory influences on subsequent sensory information, such as a startling stimulus. Similarly, PPI is frequently interpreted as an operational measure of sensorimotor gating, referring to the attributes of the lead *sensory* event that inhibits the *motor* reflex initiated by a startle stimulus (Braff and Geyer, 1990; Braff et al., 2001; Swerdlow et al., 2001). This gating mechanism is hypothesized to regulate the stream of sensory data available for information

* Corresponding author. Tel.: +1 407 882 0313.
E-mail address: aaronduley@gmail.com (A.R. Duley).

0167-8760/$ - see front matter © 2007 Elsevier B.V. All rights reserved.
doi:10.1016/j.ijpsycho.2007.01.006

processing and may serve to reduce distractibility by irrelevant or erroneous stimuli (Blumenthal, 1999). Moreover, because PPI is susceptible to attentional manipulations (e.g., a participant may be instructed to determine if the prepulse is high or low in tone), it can be used to index both automatic and controlled attentional processing (Filion et al., 1993; Graham, 1975; Hackley and Graham, 1983, 1987).

The application of PPI has proven to be an extremely valuable tool in evaluating a host of neuropsychiatric disorders demarcated by "high distractibility, sensory overload, and reduced habituation" (Blumenthal, 1999, p. 65; Braff et al., 2001). For example, disorders such as schizophrenia, Parkinson's disease, Huntington's disease, Tourette's syndrome, and attention deficit hyperactivity disorder to name a few, are routinely accompanied by a reduction in PPI compared to control subjects (for a review, see Braff et al., 2001). From a clinical perspective, a common attribute to each of these disorders is a reduced ability to filter out irrelevant internal or external stimuli, intrusive thoughts, or the inability to suppress undesirable impulses (Dawson et al., 2000; Braff et al., 2001).

1.1. PPI and anxiety

Evidence for the coupling of reduced PPI and certain anxiety disorders has also been noted. These data provide evidence that PPI is attenuated or enhanced for anxious subgroups compared to controls for investigations where simple lead stimuli or negatively valenced stimuli serve as prepulses, respectively. For example, using simple lead stimuli, Swerdlow et al. (1993) exposed individuals diagnosed with obsessive–compulsive disorder to four different auditory prepulse intensities (2, 4, 8, or 16 dB above background) and found impaired sensorimotor gating for the 4–8 dB pair. According to the researchers, this finding contrasts PPI effects observed in schizophrenic patients, for example, who display increased sensorimotor deficits with more intense lead stimuli. Grillon et al. (1996) found a trend for reduced PPI (120 ms lead interval) in combat veterans suffering from post-traumatic stress disorder (PTSD) compared to combat veterans without PTSD, but observed significantly attenuated PPI when compared to normal controls. Likewise, Ludewig et al. (2002), exposed patients with clinically diagnosed panic disorder (PD) and controls to 40 ms of 115 dB white noise pulse paired with prepulse stimuli at lead intervals of 30, 60, 120, 240, and 2000 ms. PD patients exhibited significantly reduced PPI at 30, 60, and 240 ms compared to controls. When the PD group was subdivided to determine whether these differences varied as a function of the state or trait dimensions of anxiety, a group by condition interaction emerged, with high trait anxious patients occasioning reduced PPI at the 240 ms condition compared to low trait anxious PD patients.

Broadly speaking, short lead interval startle modification research appears to be a promising tool to evaluate the psychological modulation of anxiety in response to different clinical and pharmacological treatments. Furthermore, as reviewed by Braff et al. (2001), numerous investigations have exploited PPI's parametric sensitivity to the systemic effects of different chemical compounds and their psychiatric veracity in translational and human models of neuropsychiatric illnesses. Given these and previously mentioned reasons, the application of PPI to investigate a host of questions as they pertain to anxiety is a promising physiological vehicle for strong inference toward identification of mechanisms underlying psychological phenomena.

1.2. Exercise and anxiety

The kinesiology literature is replete with empirical and anecdotal evidence touting the anxiolytic effects of acute and chronic exercise. According to Landers and Arent (2000), at least six meta-analyses have been conducted on the subject, all adding similar credence to the claim that exercise is an effective anxiolytic. Exercise's effectiveness appears to span variations in the type (i.e., whether exercise is acute or chronic), duration, and frequency; however, larger effects are typically observed for aerobic exercise (Broman-Fulks et al., 2004; Landers and Petruzzello, 1994). Given that a large portion of the extant data touting the anxiolytic effects of exercise has been self-report in nature, converging evidence from psychophysiological sources may further delineate how exercise operates to reduce anxiety, and would buttress contemporary arguments for alternatives to pharmacological treatment, including the use of exercise therapy to regulate stress and anxiety.

1.3. Current investigation

The purpose of this investigation was to determine whether the anxiety reducing properties of exercise extend to amending sensorimotor gating deficits observed in highly trait anxious participants. On two occasions, low (LA) and high (HA) trait anxious participants engaged in either a quiet rest or an exercise session (at 70% of their age-predicted maximum heart rate) for 30 min prior to testing. Following each session, a simple auditory lead stimulus was presented at 30, 60, and 120 ms prior to the pulse signal. Participants were instructed to look at a fixation cross and ignore the sounds that were presented. The specific lead intervals were selected to correspond with the few studies that have systematically evaluated PPI and anxiety. While inclusion of both a 120 and 240 ms lead interval would have been desirable to replicate findings from both the study by Ludewig et al. (2002) and Grillon et al. (1996), testing session length was a factor given the need to obtain enough trials per condition for a representative summary statistic. Therefore, the 120 ms lead interval was selected given that it was included in both of these previous studies.

Based on previous evidence from the expansive literature on exercise and anxiety, as well as findings that suggest attenuated PPI in highly anxious participants to simple lead stimuli, the following hypotheses were made: (1) Highly trait anxious (HA) participants would exhibit reduced PPI across all lead intervals and testing sessions (i.e., quiet rest and exercise) compared to the low trait anxious (LA) group. (2) In contrast to the quiet rest condition, both the HA and LA groups would show a relative increase in PPI following exercise for the 120 ms but not for lead intervals earlier than 60 ms. Accordingly, it was not

predicted that acute exercise would produce marked changes in PPI at lead intervals presumed to be associated with preattentive processing; rather, exercise was expected to modulate later controlled processing. (3) In accord with previous data demonstrating that PPI may often be differentially expressed based on eyeblink laterality (Braff et al., 2001) blink EMG was recorded from both the right and left eye.

2. Methods

2.1. Participants

Participants were 19 male and 26 female students from the University of Florida who were recruited from undergraduate courses in exchange for a small amount of extra course credit. Participants were selected from a larger screening sample of approximately 400 students. The mean STAI trait score was 36.72 (Median=36, SD=8.09; Range=44), with the lower and upper quartile cutoffs at 31 and 42, respectively. Participants were recruited from ascending scores in the lower and descending scores in the upper quartiles of the screening sample. Two participants were excluded from the HA group because they reported taking some form of anxiety medication, and another two HA and LA participants dropped out of the study. Due to equipment problems and/or errors, data was missing for one HA participant. Final sample sizes for each group were: LA (n=23) and HA (n=15). The LA group was comprised of 10 males and 13 females, with a mean age of 21.06 (SD=0.31) years. The HA group was comprised of 5 males and 10 females, with a mean age of 21.35 (SD=0.42) years. Most participants described themselves as Caucasian (n=32). Other racial groups represented in the sample included African–American (n=2), Hispanic (n=2), Asian (n=2).

2.2. Materials

2.2.1. Questionnaires for personality and mood

The following questionnaires were used as general indices of personality and mood: State-Trait Anxiety Inventory (STAI; Speilberger et al., 1983); Beck Depression Inventory (BDI; Beck et al., 1996); Positive and Negative Affect Schedule (PANAS; Watson et al., 1988); Behavior Inhibition/Activation Scale (BIS/BAS; Carver and White, 1994).

2.2.2. Physical activity readiness questionnaire (British Columbia Ministry of Health, 1993; DOS138:\PARQ.795)

The PARQ is a 7-item questionnaire used to evaluate an individuals' ability to exercise safely. Respondents simply check "Yes" or "No" in response items. "Yes" answers are a flag that an individual should consult a doctor before partaking in any exercise protocol.

2.2.3. Leisure-time exercise questionnaire (LTEQ; Godin et al., 1986)

The LTEQ is a reliable and valid (Jacobs et al., 1993) self-report behavioral measure of leisure-time physical activity in which participants indicate the average amount of time in a typical week they have engaged in strenuous, moderate, and mild exercise for more than 20 min. In addition to serving as a behavioral measure of leisure-time physical activity, the scale can be used to estimate energy expenditure expressed in the form of metabolic equivalents (METS) by computing the product of mild, moderate, and strenuous scores by 3, 5, and 9, respectively.

2.2.4. Prepulse and pulse stimuli

Acoustic stimuli were delivered binaurally using Sony headphones (MDR-V700DJ) connected to a 24-bit Sound Blaster Audigy soundcard (Creative Technologies, Ltd., Milpitas, CA). The prepulse stimulus consisted of a 1000 Hz tone lasting for 40 ms and presented at 70 dB (A). The pulse, or startling stimulus, was comprised of broadband white noise, with near instantaneous rise time, for 40 ms and presented at 102 dB (A). Sound calibration was completed prior to each participant using a RadioShack (33–2055, Fort Worth, TX) digital sound level meter.

2.3. Physiological data collection

A program written in LabVIEW (7.0; National Instruments; Austin, TX) coordinated the trial timing, stimulus events, and digital triggering for the collection of the physiological data. Electromyographic (EMG) activity of the orbicularis oculi muscle was recorded using two 4 mm Ag/AgCl (EL204S; Biopac Systems, Santa Barbara, CA) electrodes positioned according to the guidelines recommended by Blumenthal et al. (2005) for both the right and left eyes. Specifically, the participant's skin was first prepared by gently rubbing a small amount of Omni-Prep gel at the electrode site, and then cleaned with distilled water. The first electrode was placed directly underneath the participant's lower eyelid and the second electrode placed 1 cm lateral and slightly superior to the first. Raw signals were amplified (×5000) and filtered using a passband of 10–500 Hz (80 dB/Octave; EMG100B; Biopac Systems). Acknowledge software (3.7.2, Biopac Systems) was used to interface an MP150 control module (12-bit A/D converter; Biopac Systems) via a cross-over cable and sampled at 2 kHz from 0.5 s prior to prepulse onset and for 1.5 s following pulse offset.

2.4. Procedure

Following completion of a University approved informed consent, participants completed the Physical Activity Readiness Questionnaire (PARQ). If a 'yes' response was given for any item the participant was thanked, informed that they did not qualify for the study, and excused. At this point, a quiet rest or exercise condition ensued, with the two conditions counterbalanced across subjects. Participants were aware to which condition they would be subjected prior to coming to the lab for testing. For the quiet rest condition, participants were asked to bring leisure reading materials (e.g., magazines), but explicitly told they should not bring school related materials. The participants also had an option to rest quietly, so long as they

did not sleep. On the day of testing, participants sat in a chair alone in a quiet room (ambient noise level ~60 dB) with the room temperature at approximately 24 °C. After 30 min, the state version of the STAI was completed. Participants then accompanied the experimenter to a testing room located within the laboratory. Electrodes were affixed and the headphones were situated. Participants were instructed to maintain their gaze on a fixation cross (10 cm × 10 cm) located centrally on a screen approximately 2 m in front of them (Sharp Notevision LCD projector, XG-NV2U, Tokyo, Japan). They were then told that they would hear sounds coming from the headphones, which they should ignore. The entire testing session lasted about 10 min, and was comprised of 4 blocks of 12 trials. Each block contained 2 trials each of prepulse–pulse pairs at 30, 60, and 120 ms discrete lead intervals, 3 pulse only trials, and 3 prepulse only trials (i.e., 48 total). Stimulus orders were randomized for each participant. Following completion of the quiet rest condition, participants completed several questionnaires and were told that they would be contacted to set up their next esting session (average number of days between sessions = 9.18, SD = 9.57).

For the exercise session, participants engaged in 30 min of exercise using a stationary bike, and were asked to maintain 70% of their age-predicted maximum heart rate level verified with a heart rate monitor (A-1, Polar Electro Inc., Lake Success, NY). Participants were asked to maintain a rate of 60 rpm, with the level of resistance increasing and decreasing such that the target heart rate was maintained. Following the exercise session, participants completed the state version of the STAI and the aforementioned procedure was completed. For either visit, the time from administration of the STAI until actual testing began was approximately 10 min. Following the final testing session, participants were debriefed and thanked for their participation.

Table 1
Descriptive statistics and comparisons by group

Scale	Group		t	α
	Low anxious	High anxious		
Body Mass Index	25.3 (3.85)	24.4 (4.32)	t (37) = −0.18	n/a
Leisure-time exercise				
Mild	10.43 (5.64)	6.92 (4.48)	t (34) = 1.92	n/a
Moderate	17.39 (10.43)	13.46 (8.75)	t (34) = 1.15	n/a
Strenuous	42.26 (20.20)	37.28 (20.73)	t (34) = 0.69	n/a
Beck Depression Inventory	3.72 (3.28)	10.58 (4.36)	t (32) = −5.83**	.82
State-Trait Anxiety Inventory				
Trait anxiety	31.05 (4.52)	47.86 (4.90)	t (34) = −10.54**	.91
State anxiety				
Quiet rest	25.39 (3.79)	33.69 (8.49)	t (34) = −4.07**	.85
Exercise	27.05 (4.51)	31.69 (8.44)	t (29) = −1.79	.88
Positive–Negative Affect Scale				
Positive	41.01 (5.31)	36.03 (5.69)	t (34) = 3.20**	.87
Negative	16.68 (4.69)	23.28 (5.34)	t (34) = −4.48**	.89
Behavioral Activation Scale				
Drive	11.61 (2.35)	11.50 (1.09)	t (34) = −0.12	.68
Fun seeking	12.50 (2.48)	11.42 (2.35)	t (34) = 1.01	.72
Reward acceptance	17.79 (2.05)	17.83 (1.95)	t (34) = −0.23	.70
Behavioral Inhibition Scale	18.94 (2.05)	23.83 (1.75)	t (34) = −4.34**	.88

$p < .01$**.

2.5. Data reduction and analysis

Following data collection, raw EMG waveforms were first rectified and then smoothed using an FIR windowed filter (i.e., Hamming Window, 101 Coefficients) with a low-pass cutoff frequency set at 40 Hz (van Boxtel and Blumenthal, 2003). The waveform was subsequently baseline corrected by subtracting the mean 250 ms prestimulus baseline from each data point in the waveform.

To establish the dependent measures of interest the following information was obtained using computer-assisted scoring. For a given trial, response magnitude and amplitude were determined by locating the waveform peak within a 20–200 ms window following the acoustic startle stimulus. Once this value was determined, PPI was converted to proportion of difference scores by subtracting the average response on pulse alone trails from each prepulse condition and dividing that value by the average response on control trials (Blumenthal et al., 2004). Response probability was determined by dividing the total number of trials in which a response was detected by the total number of trials in which the startle stimulus was delivered.

Prior to data analysis, rejection, non-response, and exclusion criteria were established for both trial and participant data. Trials in which an unstable baseline was detected were discarded. While both amplitude and magnitude express the intensity of the muscle contraction associated with the eye-blink response, the summary statistic for trials in which a detectable EMG response did not occur within a 20–200 ms window were scored as zero for magnitude, while amplitude excluded these trials.

Separate general linear models were used to determine differences in PPI for proportion of difference scores computed for blink magnitude, blink amplitude, response probability, and raw blink magnitude for the pulse only trials. Separate 2 (Group: HA, LA) × 2 (Gender: M, F) × 2 (Session: quiet rest, exercise) × 3 (Lead Interval: 30, 60, 120) × 2 (Eye: left, right) analysis of variance (ANOVA) with repeated measures on the last three factors was used to assess PPI computed using blink magnitude. A 2 (Group: HA, LA) × 2 (Gender: M, F) × 2 (Session: quiet rest, exercise) × 3 (Lead Interval: 30, 60, 120) analysis of variance (ANOVA) with repeated measures on the last two factors was used to evaluate response probability. For all ANOVAs, multivariate tests were used to avoid violations due to sphericity. Follow-up analyses were conducted where appropriate using Student–Neuman–Keuls (SNK) corrected paired samples t-tests to control for inflation of the Type I error rate, and simple effects tests were used as follow-up procedures for significant interactions. For all analyses, the probability value was set at $p < 0.05$.

3. Results

3.1. Questionnaires

Table 1 presents means, standard deviations, and simple comparisons for Body Mass Index (BMI), and the various questionnaires for each group. The HA group reported significantly greater depression, trait anxiety, state anxiety following the quiet rest session only, greater negative and less positive affect, as well as greater behavioral inhibition compared to the LA group. The groups did not differ on BMI, LTEQ, or on any of the BAS subscales.

3.2. Prepulse inhibition

Consistent with previous findings demonstrating reduced PPI computed from blink magnitude for high trait anxious samples, a Group × Gender × Lead Interval interaction was obtained, F (2, 30) = 5.05, $p < .02$, $\lambda = 0.748$. Both the Group × Lead Interval and the Gender × Lead Interval interactions were also significant, F (2, 30) = 5.79, $p < 0.01$, $\lambda = 0.722$ and F (2, 30) = 4.47, $p < .05$, $\lambda = 0.770$, respectively. Fig. 1 depicts these interactions. Follow-up pairwise comparisons using the SNK procedure revealed that the three-way interaction was primarily attributed to differences at both the 30 and 60 ms lead intervals. HA females exhibited significantly less PPI at each of these lead intervals (30 ms: $M = -0.655$, SE = 0.06; 60 ms: $M = -0.66$, SE = 0.7), as compared to their LA counterparts (30 ms: $M = -0.76$, SE = 0.05; 60 ms: $M = -0.751$, SE = 0.06). HA males also occasioned significantly less PPI at the 30 and 60 ms lead intervals, (30 ms: $M = -0.64$, SE = 0.11; 60 ms: $M = -0.51$, SE = .12), but were also found to have significantly less PPI at the 120 ms lead interval ($M = -0.71$, SE = 0.12) relative to their LA counterparts ($M = -0.77$, SE = .06), ($M = -0.81$, SE = 0.07), and ($M = -0.80$, SE = .07), while the pairwise comparison for HA females ($M = -0.661$, SE = 0.06) and LA females ($M = -0.72$, SE = 0.06) participants failed to reach significance.

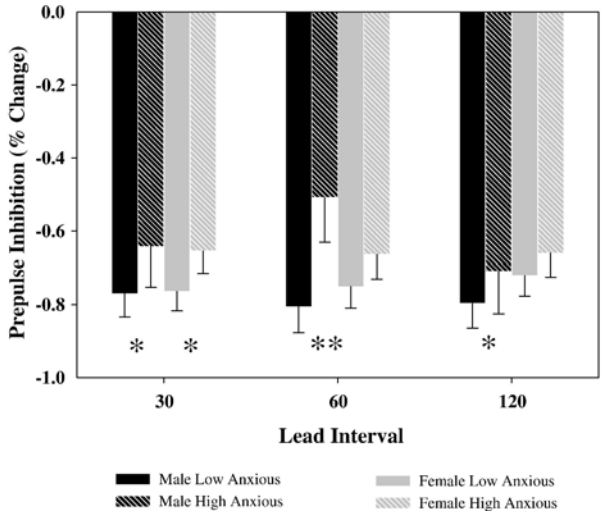

Fig. 1. Depicts the mean PPI percent change as a function of Group and Lead Interval. High trait anxious males occasioned significantly reduced PPI at all lead intervals compared to male low anxious controls. Although high anxious females exhibited a similar pattern, reduced PPI was noted only for the 30 and 60 ms lead intervals.

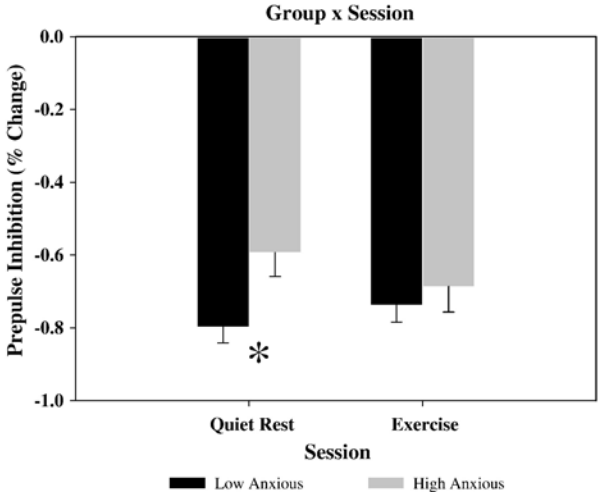

Fig. 2. The figure illustrates the mean PPI percent change as a function of Group and Session. PPI was significantly reduced for high anxious participants following quiet rest as compared to low anxious controls. No differences in PPI were observed following exercise or between quiet rest and exercise between or within groups.

Central to determining whether exercise was effective at moderating the PPI-related deficits for high trait anxious participants, the critical Group × Session interaction emerged, F (1, 31) = 6.27, $p < .02$, $\lambda = 0.83$ (see Fig. 2). SNK post-hoc analyses on these data showed significantly less PPI was occasioned following quiet rest for HA ($M = -0.593$, SE = 0.067) relative to LA ($M = -0.80$, SE = 0.44) participants, while no difference was observed between HA ($M = -0.74$, SE = 0.046) and LA ($M = -0.74$, SE = .046) participants following exercise.

For PPI proportion of difference scores computed from blink amplitude, a significant Group × Session interaction was obtained, F (1, 28) = 4.23, $p < .05$, $\lambda = 0.869$, but post-hoc comparisons were not significant. The two-way interaction was superseded by a significant Group × Session × Eye interaction, F (1, 28) = 4.71, $p < .05$, $\lambda = 0.856$. Follow-up comparisons confirmed that the LA group exhibited significantly greater PPI for both the left ($M = -0.772$, SE = 0.053) and right ($M = -0.758$, SE = 0.051) eye following quiet rest as compared to the HA group, ($M = -0.496$, SE = 0.078) and ($M = -0.539$, SE = 0.075), respectively. Following exercise, however, this group difference remained for the right eye only (LA = −0.750, SE = 0.057 vs. HA = −0.598, SE = 0.083), with the HA group showing significantly greater PPI for the left eye following exercise ($M = -0.627$, SE = 0.095) relative to quiet rest. Whereas the HA group displayed a significant increase in PPI for their left eye for exercise versus quiet rest, LA participants showed the opposite pattern; exhibiting a significant decrease in PPI for their left eye following exercise ($M = -0.653$, SE = 0.053) relative to quiet rest ($M = -0.772$, SE = 0.053).

3.3. Order effects and pulse only data

To evaluate whether the present findings were a consequence of the session order in which participants engaged in exercise or quiet rest, an Order (quiet rest first, exercise first) × Session (quiet rest, exercise) multivariate ANOVA with repeated measures for the last factor indicated that no such effect of session order emerged, F (1, 34) = 0.182, $p > 0.05$, $\lambda = 0.995$.

In addition to evaluating whether exercise or quiet rest differentially moderated PPI, a separate analysis was conducted to determine whether either treatment altered raw blink magnitude for pulse only trials. A Group × Gender × Session × Eye ANOVA with repeated measures on the last two factors indicated that differences in raw blink magnitude were not significant, F (1, 31) = 0.431, $p > 0.05$, $\lambda = 0.986$. Low anxious and high anxious males had mean blink magnitudes of (M = 0.366, SE = 0.92) and (M = 0.210, SE = (0.16), while low anxious and high anxious females had mean blink magnitudes of (M = 0.412, SE = 0.76) and (0.258, SE = (0.87), respectively. All other interactions and main effects were also not significant.

3.4. Response probability

Table 2 reports the blink response probabilities obtained for the current study. While a Group × Gender × Lead Interval × Session interaction approached significance, F (2, 30) = 3.64, $p = 0.054$, $\lambda = 0.823$, a significant Gender × Lead Interval × Session interaction was observed, F (2, 30) = 3.64, $p < 0.05$, $\lambda = 0.805$. Follow-up pairwise comparisons for the significant three-way interaction using the SNK procedure revealed that the males exhibited a significantly lower response probability at the 30 (M = 0.78, SE = 0.06) and 120 ms (M = 0.722, SE = 0.06) lead intervals compared to females, (M = 0.87, SE = 0.04) and (M = 0.81, SE = 0.04). These findings are consistent with previous data by Blumenthal and Gescheider (1987) who found women had significantly greater response probability than males when using tactile prepulses. Moreover, response probability for females significantly decreased for each lead interval following exercise (Lead 30: M = 0.80, SE = 0.04; Lead 60: M = 0.82, SE = 0.03; Lead 120: M = 0.83, SE = 0.04) compared to the same lead interval following quiet rest (Lead 30: M = 0.87, SE = 0.04; Lead 60: M = 0.86, SE = 0.04; Lead 120: M = 0.88, SE = 0.04). Males exhibited a similar pattern for response probabilities at 30 ms (exercise: M = 0.75, SE = 0.06) and (quiet rest: M = 0.78, SE = 0.07), however exhibited a significantly greater response probability at 60 ms (exercise: M = 0.85, SE = 0.05) and (quiet rest: M = 0.71, SE = 0.06).

Table 2
Response probability by group and gender for each PPI and pulse condition

Condition	Gender	Low trait anxious		High trait anxious	
		Quiet rest	Exercise	Quiet rest	Exercise
Pulse alone	M	0.94 (0.04)	0.96 (0.02)	0.96 (0.02)	0.96 (0.04)
	F	0.98 (0.01)	0.97 (0.02)	0.97 (0.02)	0.97 (0.01)
Lead 30	M	0.75 (0.06)	0.81 (0.07)	0.81 (0.10)	0.69 (0.12)
	F	0.85 (0.05)	0.80 (0.06)	0.89 (0.05)	0.82 (0.05)
Lead 60	M	0.70 (0.06)	0.80 (0.05)	0.71 (0.10)	0.90 (0.10)
	F	0.83 (0.05)	0.82 (0.04)	0.89 (0.06)	0.82 (0.07)
Lead 120	M	0.74 (0.06)	0.76 (0.06)	0.71 (0.11)	0.71 (0.11)
	F	0.83 (0.05)	0.85 (0.05)	0.92 (0.06)	0.81 (0.06)

4. Discussion

The purpose of this study was to determine whether an acute exercise bout moderates the sensorimotor gating deficits observed among individuals with high trait anxiety. Prepulse inhibition was assessed within a simple auditory short lead interval startle modification protocol following both a quiet rest and a moderately intense exercise session. Though PPI magnitude was inhibited for the highly anxious participants relative to low anxious controls following a quiet rest session, no group differences were observed following the exercise session. When evaluating blink magnitude, high trait anxious males occasioned reduced PPI at all lead intervals compared to low anxious male controls. Although high anxious females exhibited a similar pattern, reduced PPI was noted only for the 30 and 60 ms lead intervals. For PPI data computed from blink amplitude, highly trait anxious participants were found to exhibit reduced PPI for both 30 and 60 ms lead intervals relative to LA controls when collapsed across both the quiet rest and exercise sessions. Further, HA participants displayed an increase in PPI for exercise compared to quiet rest, which was significant for the left eye only, whereas reduced PPI persisted for quiet rest relative to exercise for the right eye. Conversely, LA participants exhibited significantly decreased PPI for the left eye following exercise relative to quiet rest, with no observable differences for PPI measured from the right eye.

Our findings corroborate other reports that highly trait anxious participants' exhibit reduced PPI at early lead intervals relative to controls (e.g., Ludewig et al., 2002; Grillon et al., 1996; Swerdlow et al., 1993). Attenuated PPI in this context is thought to partially reflect deficits in a regulatory system in the brain which assists in reducing the information processing burden of irrelevant or erroneous sensory stimuli. Braff, Geyer, Swerdlow, and their colleagues (Braff et al., 2001; Swerdlow and Geyer, 1999; Swerdlow et al., 2001) argue that operational measures of sensorimotor function, namely prepulse inhibition, but also including the P50 component of the event-related potential, are sensitive to gating related impairments often coupled with certain neuropsychiatric illnesses.

An assumption for this investigation was that sensorimotor gating deficits are indicative of an active anxiety condition, but whose effects could be diminished following treatment. For example, as first suggested by Aitken et al. (1999), there appears to be a logical relationship between the large body of attentional bias literature, which suggests that anxiety is linked with biases in information processing, and the ability of short lead interval startle modification to resolve these effects, given its sensitivity to automatic and controlled attentional processes. From this body of literature, there is evidence from interference and probe detection paradigms that the biases observed in clinically anxious participants often are reduced or eliminated following treatment (e.g., Williams et al., 1996). However, it is not entirely clear whether the results from treatment will

manifest in operational measures of sensorimotor function. Moreover, while several studies have evaluated the pharmacological consequences of different anxiolytic compounds (e.g., clonidine and diazepam) with normal human subjects (e.g., Abduljawad et al., 1997), there are few studies that have directly investigated sensorimotor gating before and after either talk-based therapies or following pharmacological remedies in clinically anxious participants. For those studies that have examined the latter, confounds as a product of reduced startle amplitude are often an issue (e.g., Grillon et al., 1994). In spite of these disparities, the working hypothesis for the present study was that the gating related deficits associated with high trait anxiety could be altered with treatment, and therefore theoretically permit the anxiolytic properties of exercise to be expressed with an increase in PPI relative to quiet rest. This prediction was supported, with high anxiety and low anxiety participants showing similar PPI following exercise relative to quiet rest. Nevertheless, this finding only partially supports the prediction that exercise has a moderating effect on PPI. That is, PPI could have been reduced for the low anxiety group while the high anxiety group remained constant following exercise. While the pairwise comparison between quiet rest and exercises did not reveal differences in PPI for low anxious participants, the simple comparison which would confirm an increase in PPI following exercise showed only a trend in the predicted direction for the high anxiety group (SNK Obtained = 2.37, $\alpha = 2.77$). All other main effects and interactions for proportion of difference scores computed for blink magnitude were non-significant.

In considering likely explanations for this finding, the potential contributions of state anxiety were initially explored. Perhaps high anxious participants, on average, experienced a greater reduction in state anxiety following exercise relative to quiet rest than the low anxious subjects, leading to the observed increase in PPI? As summarized by Landers and Arent (2000), two meta-analytic reviews (Landers and Petruzzello, 1994; Petruzzello et al., 1991) both describe that larger effect sizes are typically observed for individuals who are highly trait anxious relative to normal controls. As reported, group differences in state anxiety emerged for the quiet rest session only, with the high anxious group reporting more anxiety than low anxious participants. This difference disappeared following exercise however, and a significant net change in self-reported anxiety was also non-existent both between and within groups. Regardless, it may be possible that the level of statistical power necessary to isolate those differences was not present, and that state anxiety may be interacting with dispositional anxiety levels to yield the current results.

Landers and Arent (2000) also explain that reported effect sizes for changes in anxiety may also vary as a function of baseline fitness levels. In the present study, frequency of leisure-time physical activity was not found to differ between groups. Nevertheless, the instrument used can provide only a retrospective analysis of activity, and conceivably, may not fully yield an accurate account of an individuals' exercise history. This concern highlights and acknowledges a limitation of the present study. Because participants were unselected on the basis of their reported LTEQ scores, it is possible that exercise history interacted with state anxiety such that some individuals may have experienced the predicted reductions in anxiety, while others may have occasioned an increase. Nevertheless, correlations among the different LTEQ activity levels with either self-reported anxiety following quiet rest, exercise, or these sessions net change were all non-significant. Even with an accurate representation of an individual's exercise history, resting level of cardiorespiratory fitness may have varied such that the exercise intensity, based on the age-predicted heart rate maximum values, was not representative of a person's true level of fitness. Unfortunately, verification of whether baseline fitness played a role in the present results was not possible in the current design. Future attempts should carefully consider this possibility.

Future studies should systematically determine the contribution of attentional mechanisms in moderating PPI via exercise. Although the Group × Session × PPI interaction was not significant, in hindsight, this finding could likely be a product of instructions to ignore the prepulse and pulse stimuli. In most other examinations of attentional bias, tasks typically require an active attentional set. For that reason, a simple alteration in the experimental protocol would be to vary the task instructions such that participants are instructed to attend to the prepulse, or to have them detect high versus low pitched tones as in Filion et al. (1993). This may help in identifying differences related to attention allocation between high anxious and low anxious samples, and more systematically evaluate the consequences of acute exercise and attention.

There is a paucity of research documenting laterality differences in PPI with high trait anxious samples (cf. Swerdlow et al., 1997 for an exception). The present study evidenced PPI group differences for both eyes, although increased PPI following exercise was only evidenced from the left eye. In studies which have evaluated PPI deficits with patients diagnosed with schizophrenia, schizotypal personality disorder, Huntington's disease, and OCD, PPI deficits are generally found with eyeblink recordings from the right eye, while in other groups (e.g., Enuretic children) deficits in PPI are found from the left eye only (Swerdlow and Geyer, 1999). This finding may implicate greater right hemisphere lateralization of exercise effects on anxiety. Because PPI computed from blink amplitude does not include information about the presence or absence of a blink, and given the finding that response probability significantly differed across Gender and Session, a second likely explanation is that the laterality differences reflected in PPI computed from blink amplitude represents a complex interaction of physical activity that is sensitive to Gender and Trait Anxiety. However, more research is needed to dissect this interaction.

In conclusion, the current study supported the hypothesis that exercise moderates the sensorimotor gating deficits seen coupled with high trait anxiety. As it applies more broadly to anxiety research, there are many questions that short lead interval startle modification can address, and an opportunity for future investigators to explore how alternative measures of sensorimotor function, such as the P50 of the event-related potential, may shed additional light on psychophysiological

processes underlying anxiety and its disorders. Replication of the current findings are needed to determine the significance of PPI lateralization effects within the broad scope of sensorimotor function and anxiety, as well as to firmly delineate the possible role of state anxiety and attention in the regulation of PPI following exercise.

References

Abduljawad, K.A.J., Langley, R.W., Bradshaw, C.M., Szabadi, E., 1997. Effects of clonidine and diazepam on the acoustic startle response and on its inhibition by 'prepulses' in man. Journal of Psychopharmacology 11, 29–34.

Aitken, C.J., Siddle, D.A.T., Lipp, O.V., 1999. The effects of threat and nonthreat word lead stimuli on blink modification. Psychophysiology 36, 699–705.

Beck, A.T., Steer, R.A., Brown, G.K., 1996. Manual for the Beck Depression Inventory, 2nd ed. The Psychological Corporation, San Antonio, TX.

Berg, W.K., Balaban, M.T., 1999. Startle elicitation: stimulus parameters, recording techniques, and quantification. In: Dawson, M.E., Schell, A.M., Bohmelt, A.H. (Eds.), Startle Modification: Implications for Neuroscience, Cognitive Science, and Clinical Science. Cambridge University Press, Cambridge, UK, pp. 21–50.

Blumenthal, T.D., 1999. Short lead interval startle modification. In: Dawson, M.E., Schell, A.M., Böhmelt, A.H. (Eds.), Startle Modification: Implications for Neuroscience, Cognitive Science, and Clinical Science. Cambridge University Press, Cambridge, pp. 51–71.

Blumenthal, T.D., Gescheider, G.A., 1987. Modification of the acoustic startle response by tactile prepulse: effects of stimulus onset asynchrony and prepulse intensity. Psychophysiology 24, 320–327.

Blumenthal, T.D., Cuthbert, B.N., Filion, D.L., Hackley, S., Lipp, O.V., Boxtel, A.V., 2005. Committee report: guidelines for human startle eyeblink electromyographic studies. Psychophysiology 42, 1–15.

Blumenthal, T.D., Elden, A., Flaten, M.A., 2004. A comparison of several methods used to quantify prepulse inhibition of eyeblink responding. Psychophysiology 41, 326–332.

Braff, D.L., Geyer, M.A., 1990. Sensorimotor gating and schizophrenia: human and animal model studies. Archives of General Psychiatry 47, 181–188.

Braff, D.L., Geyer, M.A., Swerdlow, N.R., 2001. Human studies of prepulse inhibition of startle: normal subjects, patient groups, and pharmacological studies. Psychopharmacology 156, 234–258.

Broman-Fulks, J.J., Berman, M.E., Rabian, B.A., Webster, M.J., 2004. Effects of aerobic exercise on anxiety sensitivity. Behavior Research and Therapy 42, 125–136.

Carver, C.S., White, T.L., 1994. Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: the BIS/BAS scales. Journal of Personality and Social Psychology 67, 319–333.

Davis, M., Walker, D.L., Lee, Y., 1999. Neurophysiology and neuropharmacology of startle and its affective modulation. In: Dawson, M.E., Schell, A.M., Böhmelt, A.H. (Eds.), Startle Modification: Implications for Neuroscience, Cognitive Science, and Clinical Science. Cambridge University Press, Cambridge, pp. 95–113.

Dawson, M.E., Schell, A.M., Hazlett, E.A., Nuechterlein, K.H., Filion, D.L., 2000. On the clinical and cognitive meaning of impaired sensorimotor gating in schizophrenia. Psychiatry Research 96, 187–197.

Fendt, M., Li, L., Yeomans, J.S., 2001. Brain stem circuits mediating prepulse inhibition of the startle reflex. Psychopharmacology 156, 216–224.

Filion, D.L., Dawson, M.E., Schell, A.M., 1993. Modification of the acoustic startle reflex eyeblink: a tool for investigating early and late attentional processes. Biological Psychology 35, 185–200.

Filion, D.L., Dawson, M.E., Schell, A.M., 1998. The psychological significance of human startle eyeblink modification: a review. Biological Psychology 47, 1–43.

Godin, G., Jobin, J., Bouillon, J., 1986. Assessment of leisure-time exercise behavior by self-report: a concurrent validity study. Canadian Journal of Public Health 77, 359–361.

Graham, F.K., 1975. Presidential address, 1974. The more or less startling effects of weak pre-stimulation. Psychophysiology 12, 238–248.

Grillon, C., Sinha, R., O'Malley, S., 1994. Effects of ethanol on the acoustic startle reflex in humans. Psychopharmacology 114, 167–171.

Grillon, C., Morgan, C.A., Southwick, S.M., Davis, M., Charney, D.S., 1996. Baseline startle amplitude and prepulse inhibition in Vietnam veterans with posttraumatic stress disorder. Psychiatry Research 64, 169–178.

Hackley, S.A., Graham, F.K., 1983. Early selective attention effects on cutaneous and acoustic blink reflexes. Physiological Psychology 11, 235–242.

Hackley, S.A., Graham, F.K., 1987. Effects of attending selectively to the spatial position of reflex-eliciting and reflex-modulating stimuli. Journal of Experimental Psychology. Human Perception and Performance 13, 411–424.

Hoffman, H.S., 1997. Attentional factors in the elicitation and modification of the startle reaction. In: Lang, P.J., Simons, R.F., Balaban, M. (Eds.), Attention and Orienting: Sensory and Motivational Processes. Lawrence Erlbaum Associates, New Jersey, pp. 185–204.

Hoffman, H.S., Ison, J.R., 1980. Reflex modification in the domain of startle: I. Some empirical findings and their implication for how the nervous system processes sensory input. Psychological Review 87, 175–189.

Jacobs, D.R., Ainsworth, B.E., Hartman, T.J., Leon, A.S., 1993. A simultaneous evaluation of ten commonly used physical activity questionnaires. Medicine and Science in Sports and Exercise 25, 81–91.

Koch, M., 1999. The neurobiology of startle. Progress in Neurobiology 59, 107–128.

Koch, M., Fendt, M., 2003. Startle response modulation as a behavioral tool in neuropharmacology. Current Neuropharmacology 1, 175–185.

Landers, D.M., Arent, S.M., 2000. Physical activity and mental health, In: Singer, R.N., Hausenblas, H.A., Janelle, C.M. (Eds.), The Handbook of Sport Psychology, 2nd ed. Wiley, New York.

Landers, D.M., Petruzzello, S.J., 1994. Physical activity, fitness, and anxiety. In: Bouchard, C., Shephard, R.J., Stephens, T. (Eds.), Physical Activity, Fitness, and Health. International Proceedings and Consensus Statement. Human Kinetics, Champaign, IL, pp. 868–882.

Ludewig, S., Ludewig, K., Geyer, M.A., Hell, D., Vollenweider, F.X., 2002. Prepulse inhibition deficits in patients with panic disorder. Depression and Anxiety 15, 55–60.

Petruzzello, S.J., Landers, D.M., Hatfield, B., Kubitz, K.A., Salazar, W., 1991. A meta-analysis on the anxiety-reducing effects of acute and chronic exercise. Sports Medicine 11, 143–182.

Speilberger, C.D., Gorsuch, R.L., Lushene, R., Vagg, P.R., Jacobs, G.A., 1983. Manual for the State-Trait Anxiety Inventory (STAI). Consulting Psychologist Press, Palo Alto, CA.

Swerdlow, N.R., Geyer, M.A., 1999. Neurophysiology and neuropharmacology of short lead interval startle modification. In: Dawson, M.E., Schell, A.M., Böhmelt, A.H. (Eds.), Startle Modification: Implications for Neuroscience, Cognitive Science, and Clinical Science. Cambridge University Press, Cambridge, pp. 115–133.

Swerdlow, N.R., Benbow, C.H., Zisook, S., Geyer, M.A., Braff, D.L., 1993. A preliminary assessment of sensorimotor gating in patients with obsessive compulsive disorder. Biological Psychiatry 33, 298–301.

Swerdlow, N.R., Hartston, H.J., Zinner, S., 1997. Sensorimotor gating deficits in obsessive compulsive disorder (OCD): lateralized findings. Biological Psychiatry 41, 83S.

Swerdlow, N.R., Geyer, M.A., Braff, D.L., 2001. Neural circuit regulation of prepulse inhibition of startle in the rat: current knowledge and future challenges. Psychopharmacology 156, 194–215.

van Boxtel, A., Blumenthal, T.D., 2003. Analog and digital processing of startle eyeblink responses. Psychophysiology 40, S13 (Abstract).

Watson, D., Clark, L.A., Tellegen, A., 1988. Development and validation of brief measures of positive and negative affect: the PANAS scales. Journal of Personality and Social Psychology 54, 1063–1070.

Williams, J.M.G., Mathews, A., MacLeod, C., 1996. The emotional Stroop task and psychopathology. Psychological Bulletin 120, 3–24.