



CREaTE

Canterbury Research and Theses Environment

Canterbury Christ Church University's repository of research outputs

<http://create.canterbury.ac.uk>

Please cite this publication as follows:

Vernon, D., Peryer, G., Louch, J. and Shaw, M. (2014) Tracking EEG changes to alpha and beta binaural beats. *International Journal of Psychophysiology*, 93 (1). pp. 134-139. ISSN 0167-8760.

Link to official URL (if available):

<http://dx.doi.org/10.1016/j.ijpsycho.2012.10.008>

This version is made available in accordance with publishers' policies. All material made available by CReaTE is protected by intellectual property law, including copyright law. Any use made of the contents should comply with the relevant law.

Contact: create.library@canterbury.ac.uk



Tracking EEG changes in response to alpha and beta binaural beats.

Vernon, D^{1*}., Peryer, G²., Louch, J¹., & Shaw, M¹.

¹Department of Applied Social Sciences,
Canterbury Christ Church University,
Canterbury, Kent.
CT1 1QU, UK.

*Email: david.vernon@canterbury.ac.uk

²Faculty of Medicine and Health Sciences
University of East Anglia
Norwich Research Park
Norwich
NR4 7TJ

Abstract

A binaural beat can be produced by presenting two tones of differing frequency, one to each ear. Such auditory stimulation has been suggested to influence behaviour and cognition via the process of cortical entrainment. However, research so far has only shown frequency following responses in the traditional EEG frequency ranges of delta, theta and gamma. Hence a primary aim of this research was to ascertain whether it would be possible to produce clear changes in the EEG in either the alpha or beta frequency ranges. Such changes, if possible, would have a number of important implications as well as potential applications. A secondary goal was to track any observable changes in EEG throughout the entrainment epoch to gain some insight into the nature of the entrainment effects any changes in an effort to identify more effective entrainment regimes. Twenty two healthy participants were recruited and randomly allocated to one of two groups, each of which was exposed to a distinct binaural beat frequency for ten 1-minute epochs. The first group listened to an alpha binaural beat of 10Hz and the second to a beta binaural beat of 20Hz. EEG was recorded from the left and right temporal regions during pre-exposure baselines, stimulus exposure epochs and post-exposure baselines. Analysis of changes in broad-band and narrow-band amplitude, and frequency showed no effect of either binaural beat frequency eliciting a frequency following effect in the EEG. Possible mediating factors are discussed and a number of recommendations are made regarding future studies, exploring entrainment effects from binaural beat presentation.

Introduction

A binaural beat occurs when two carrier tones of distinct frequency are presented separately, one to each ear, and the resulting perception is of a single tone with a frequency midway between the two carrier tones that waxes and wanes in amplitude at a rate equal to the difference between them (Oster, 1973). For instance, if a clear tone of 400Hz is presented to the left ear and a tone of 410Hz is presented to the right ear this produces a perceived frequency of 405Hz which modulates in amplitude with a frequency of 10Hz. This modulated signal is often referred to as a beat and can be produced when the difference in frequency between the two carrier tones ranges from between 2 and 30Hz (Perrott & Nelson, 1969). Furthermore, there are suggestions that carrier tones of a frequency ranging from 200 – 900Hz may be more effective at eliciting binaural beats than carrier tones that exceed 1 KHz (Pratt et al., 2010; Wahbeh, Calabrese, Zwicky & Zajdel, 2007). Nevertheless, it has recently been suggested that the perceptual strength of a binaural beat signal remains relatively weak (Grose, Buss & Hall, 2012).

A number of reports have suggested that listening to binaural beats can influence behaviour and cognition in a variety of ways. For instance, reports show that exposure to binaural beats has led to improved vigilance (Lane, Kasian, Owens & Marsh, 1998) and memory (Kennerly, 1996), increased levels of hypnotic susceptibility (Brady & Stevens, 2000) and led to reductions in self-reports of anxiety (Le Scouarnec et al., 2001; Padmanabhan, Hildreth & Laws, 2005) and severity ratings of tinnitus (David, Naftali & Katz, 2010). However, others report that exposure to binaural beats has failed to influence hypnotic susceptibility (Stevens et al., 2003), produced no change in blood pressure or heart rate (Carter, 2008) and did not reduce the symptoms of children diagnosed with attention deficit hyperactivity disorder (Kennel, Taylor, Lyon & Bourguignon, 2010). It is difficult to know why such different outcomes emerge and this in part highlights a more general lack of research regarding the possible effects of listening to binaural beats. In part, such differences may occur due to the distinct methodological approaches adopted which in turn may influence the entrainment potential of binaural beats. This is further limited by the fact that only two of the above studies (Brady & Stevens, 2000; Stevens et al., 2003) monitored for potential changes in electroencephalographic (EEG) activity as a function of exposure to binaural beats.

A central assumption of binaural beats is that they can elicit an entrainment effect, sometimes referred to as a frequency following response (FFR), in the electrocortical activity of the brain and thereby represent a mechanism for potential behaviour change (for a review see Vernon, 2009). According to Hink et al (1980) the FFR arises from the brain stem. They suggest that the FFR stems from converging input from overlapping neuronal populations and that this can be directly influenced by binaural beats. It is not clear as yet precisely how widespread the changes in electrocortical activity may be following exposure to binaural beats as there are a number of inconsistencies in the literature. For example, some have reported changes amplitude at the central midline region (Cz) following exposure to binaural beats (Schwarz & Taylor, 2005) whilst others have failed to find any evidence of such a change (Wahbeh et al., 2007). These inconsistencies may stem from differences in methodology, in particular the nature and frequency of the carrier tones used to present the binaural beats as well as the duration of exposure. Nevertheless, there is an emerging consensus that exposure to binaural beats may lead to changes in brain activity in the temporal regions of the brain (Karino et al., 2004; Karino et al., 2006; Pratt et al., 2010). For instance, Karino et al (2006) reported dominant activation of both left and right temporal areas, which contain the auditory cortex, following exposure to binaural beats in the theta range (4Hz and 6.6Hz). More recently Pratt et al (2010) noted that the source of the oscillations elicited by exposure to binaural delta (3Hz) and theta (6Hz) beats were predominantly located to the left temporal region. Such findings are consistent with early suggestions that the temporal lobe is electrically more labile than other regions of the cortex (Bear, 1979). Hence, we have focused on possible changes in the EEG at the left and right temporal regions.

Despite such encouraging findings, to date changes have only been reported in three of the main traditional EEG frequency ranges. This includes changes in delta (Kennerly, 2004; Pratt et al., 2010), theta (Brady & Stevens, 2000; Karino et al., 2004; Karino et al., 2006; Pratt et al., 2010) and gamma (Schwarz & Taylor, 2005). However, no changes have yet been reported in the key EEG frequencies of alpha and beta. Hence, the primary aim of this study was to examine the effects of alpha and beta binaural beats on the EEG. By doing this we hoped to ascertain whether such a technique has the potential to increase activity in specific targeted components of the EEG which would have a number of practical applications with regards to performance enhancement, as well as clinical treatment (for a review see Vernon, 2009).

The possible entrainment and modification of alpha and beta EEG activity would present with some interesting possibilities. For instance, there is a growing body of research showing that alpha activity, far from representing cortical idling, may in fact provide an index of neural efficiency (see Vernon et al., 2009). This idea is supported by research showing that individuals classified as more intelligent exhibit greater levels of alpha power (Anokhin & Vogel, 1996; Doppelmayr et al., 2005; Jausovec, 1996) and that induced increases in alpha power using EEG biofeedback (Hanslmayr et al., 2005) and repetitive transcranial magnetic stimulation (Klimesch, Sauseng & Gerloff, 2003) have been associated with improved attentional processing. In addition, beta EEG activity has been implicated in attentional processing, much of which is based on attempts to normalise the EEG of those with attentional dysfunction (see e.g., Lubar, Swartwood, Swartwood & O'Donnell, 1995; Lubar, Swartwood, Swartwood & Timmerman, 1995). Support for this comes from research focusing on enhancing beta power using EEG biofeedback which has been shown to improve attentional processing in both clinical populations (Fuchs et al., 2003; Monastra et al., 2006) and healthy participants (Egner & Gruzelier, 2004; Rasey et al., 1996). In addition, research has shown increases in beta (12-30Hz) amplitude when participants attend to a stimulus and plan a response (Saleh, Reimer, Penn, Ojakangas & Hatsopoulos, 2010). These findings would support the use of binaural beats as a plausible mechanism for enhancing EEG power within a targeted frequency in an attempt to normalise and/or enhance cognition, if exposure to such sounds elicited clear FFR's in the EEG.

A secondary goal of the current research was to track the changes in the EEG throughout the period of exposure to the binaural beats in an attempt to obtain some insight into the possible nature of the brain's response to such sounds. Such information could prove helpful in designing and refining future entrainment protocols. It is not clear as yet if and how, the brain will respond to such audio stimulation. For instance, previous research is inconsistent with some showing that theta (6.6Hz) binaural beats are capable of eliciting changes when measured using a very short exposure window of 1000ms (Karino et al., 2004), whilst others have failed to find any evidence of cortical entrainment when theta (7Hz) binaural beats were constantly presented for 2 minutes (Goodin et al., 2012), or for periods of up to 30mins (Wahbeh et al., 2007). It may be that continuous and prolonged exposure to a binaural beat leads the brain to habituate to the signal, reducing the possibility of identifying a FFR as time progresses. Such a possibility would be consistent with research showing habituation of

evoked potentials to repeating auditory stimuli (e.g., Barry, Cocker, Anderson, Gordon & Rennie, 1992). In an attempt to address this we devised a novel exposure procedure which involved presenting the binaural beat signal in ten 1-minute segments interleaved with a single carrier tone set at the same amplitude and frequency as the perceived frequency of the binaural beats. Given that the alpha binaural beat was created using carrier tones of 395 and 405Hz, producing a perceived frequency of 400Hz modulating at 10Hz, this meant that each 1-minute segment of binaural beats was interleaved with a single pure tone played at 400Hz. Tones centred around 400Hz were used as Oster (1973) suggested that binaural beats may be better perceived with the carrier tones are close to 400Hz (see also Goodin et al., 2012). Such a procedure would not only counteract the possible habituation effects of continuous and prolonged exposure to the binaural beats but would allow us to track any possible changes in the EEG over time.

Method

Participants

Twenty two participants were recruited from the Psychology undergraduate cohort at Canterbury Christ Church University. Participants ages ranged from 18 to 32 (mean 20y) with 13 females and 9 males. Participants were randomly allocated to either the *alpha-entrainment* group or the *beta-entrainment* group¹, however, due to timing issues this resulted in 12 participants in the *alpha-entrainment* group and 10 in the *beta-entrainment* group. All participants had normal or corrected to normal vision, no reported hearing deficits/difficulties and were screened to exclude individuals with a history/family history of epilepsy, psychological problems and those taking prescribed medication. Participants received course credits for completing the study.

Binaural Beats

Previous research has suggested that binaural beats may be perceived with greater clarity when the carrier tones are higher than 100Hz (Perrott & Nelson, 1969) and closer to 400Hz (Oster, 1973). Hence, two binaural beat recordings were created using Adobe Audition. The *alpha* entrainment recording consisted of ten one-minute segments containing two sinusoidal

¹ Participants were exposed to a single binaural beat frequency to avoid potential confounding carry over effects whereby entrainment at one frequency influences the EEG outside of that frequency range (see e.g., Egner & Gruzelier, 2003; Lavalley, Koren & Persinger, 2011).

tones, one at 395Hz and one at 405Hz to produce a 10Hz binaural beat, which was interspersed 10 one-minute segments of a single carrier tone at 400Hz. The *beta* entrainment recording consisted of ten one-minute segments containing two sinusoidal tones, one at 390Hz and one at 410Hz to produce a 20Hz binaural beat, which was interspersed with 10 one-minute segments of a single carrier tone at 400Hz. All sound segments contained a 10ms attack and decay period.

EEG acquisition

EEG signals were acquired using a Nexus-10 DC-coupled EEG portable amplifier incorporating a 24-bit A/D converter with BioTrace software (MindMedia, Netherlands). EEG recording was sampled at 256Hz using two monopolar/referential sensors, one attached to T3 (according to international 10/20 system: Jasper, 1958) and referenced to the left mastoid and the other attached to T4 and referenced to the right mastoid, with a ground sensor was placed at Fz. Targeted scalp regions were prepared using NuPrep abrasive gel and sensors attached using Ten20 electrode conducting paste. All sampled data were initially subject to off-line voltage threshold artefacting to help remove possible interference from ocular, head and muscle movement. EEG data analysis was conducted off-line with the raw trace digitally filtered using an IIR Bandpass Butterworth (3rd order) filter to extract broadband alpha (8-12Hz), narrow-band alpha (9.5-10.5Hz), broadband beta (18-22Hz) and narrow-band beta (18.5-20.5Hz) mean amplitude exported at 32 samples/second. Peak frequency was determined using a fast four transform to identify the dominant frequency using 4 second epochs within a range of 8-12Hz for alpha and 18-22Hz for beta. Means were then computed for each of the target EEG components of interest for each minute of the recording.

Procedure

Participants were informed of the purpose of the study and were randomly allocated to one of the two auditory conditions (*Alpha, Beta*). They then completed each of the three stages whilst remaining seated. In the initial stage a 2 minute eyes open resting EEG baseline and a 2 minute eyes closed EEG resting baseline was recorded, with order counterbalanced across participants. In stage 2 participants were shown a nature DVD with the sound muted whilst simultaneously presented with a binaural beat recording via on-ear headphones. Each binaural beat recording consisted of ten 1-minute segments of binaural beats interspersed with ten 1-minute segments of a single carrier tone. The order of these was counterbalanced

across participants. Throughout the auditory exposure period participants' EEG was recorded from T3 and T4. During the final stage a 2-min eyes open and 2-min eyes closed resting EEG baselines were again recorded.

Results

Mean amplitude and frequency measures are reported separately for each group and for baseline and entrainment measures. For all analyses a Greenhouse-Geisser correction was used if Mauchley's test of sphericity was significant and corrected df are reported to two decimal places.

Alpha EEG

A 2 (Time: *Pre* vs. *Post*) x 2 (Hemisphere: *Left* vs. *Right*) repeated measures analysis of variance (ANOVA) was conducted on mean broadband (8-12Hz) amplitude, mean narrow-band (9.5Hz-10.5Hz) amplitude and peak alpha (8-12Hz) frequency separately for eyes open and eyes closed resting baselines.

Eyes open

For broadband amplitude there was a marginal effect of Time, $F(1,11)=4.394$, $p=0.06$, $Mse=17.23$, $\eta^2=0.285$, showing a drop in amplitude from pre to post entrainment from $8.58\mu V$ to $7.34\mu V$. For both narrow-band amplitude and peak frequency there were no main or interaction effects (all $ps>0.18$).

Eyes closed

For both broadband and narrow-band amplitude there were no main or interaction effects (all $ps>0.14$). For peak alpha frequency there was a main effect of Hemisphere, $F(1,11)=5.065$, $p<0.05$, $Mse=0.083$, $\eta^2=0.315$, showing a higher peak alpha frequency in the left hemisphere (9.81Hz) compared to the right (9.72Hz). There were no other main or interaction effects (all $ps>0.45$).

Alpha Entrainment

A 2 (Hemisphere: *Left* vs. *Right*) x 10 (Time: *1min* to *10min*) x 2 (Signal: *BB-on* vs *BB-off*) repeated measures analysis of variance (ANOVA) was conducted on mean broadband (8-12Hz) amplitude, mean narrow-band (9.5Hz-10.5Hz) amplitude and peak alpha (8-12Hz) frequency separately. Descriptives for each of these variables can be seen below in Table 1.

Table 1. Showing means (SD) for alpha broadband (8-12Hz), narrow-band (9.5-10.5Hz) amplitude and peak frequency (8-12Hz) for the ten one-minute binaural-beat on signal entrainment periods and the ten one-minute binaural-beat off carrier tone baseline periods.

			Time Periods									
			1	2	3	4	5	6	7	8	9	10
Broadband amplitude μV	Right hemisphere	BB-on	6.80(2.65)	6.56(2.83)	6.67(3.23)	6.34(2.84)	6.08(2.56)	5.66(2.43)	6.75(3.45)	5.91(3.11)	5.85(2.44)	6.46(3.12)
		BB-off	6.89(2.26)	6.95(2.45)	6.24(2.77)	6.41(3.19)	6.37(3.18)	6.57(2.22)	6.31(3.07)	6.16(3.46)	5.53(1.57)	6.06(3.05)
	Left hemisphere	BB-on	7.54(2.87)	6.61(1.18)	6.92(2.39)	6.29(2.00)	6.56(1.85)	6.30(1.87)	7.52(2.35)	6.56(2.05)	6.24(1.73)	6.32(1.41)
		BB-off	7.54(2.84)	7.36(3.02)	6.19(1.55)	6.62(2.29)	6.49(1.70)	6.75(1.95)	6.72(1.83)	6.85(1.84)	6.37(1.64)	6.73(1.78)
Narrow-band amplitude μV	Right hemisphere	BB-on	3.39(1.75)	3.05(1.44)	3.32(1.93)	3.08(1.54)	2.91(1.32)	2.81(1.45)	3.40(2.28)	2.81(1.55)	2.86(1.34)	3.16(1.77)
		BB-off	3.20(1.11)	3.40(1.39)	3.10(1.65)	3.15(1.79)	3.04(1.82)	3.16(1.15)	2.99(1.52)	2.91(1.85)	2.56(0.66)	2.98(1.79)
	Left hemisphere	BB-on	3.46(1.55)	3.01(0.57)	3.24(1.21)	2.97(0.97)	3.01(0.83)	3.00(0.95)	3.85(1.78)	3.05(1.02)	3.01(0.96)	3.08(0.84)
		BB-off	3.67(1.74)	3.52(1.79)	2.94(0.85)	3.03(1.09)	3.06(0.90)	3.16(0.86)	3.18(1.01)	3.45(1.05)	3.05(0.75)	3.22(0.87)
Peak frequency Hz	Right hemisphere	BB-on	10.0(0.26)	9.96(0.29)	9.84(0.54)	9.89(0.44)	9.85(0.59)	10.0(0.39)	9.94(0.51)	9.92(0.49)	9.97(0.52)	9.95(0.32)
		BB-off	10.0(0.28)	9.96(0.28)	9.93(0.46)	9.97(0.36)	9.84(0.47)	10.0(0.48)	9.91(0.49)	9.92(0.59)	10.0(0.35)	9.98(0.49)
	Left hemisphere	BB-on	10.0(0.39)	10.0(0.50)	10.0(0.61)	9.94(0.55)	9.97(0.47)	9.93(0.46)	10.0(0.33)	9.96(0.41)	10.1(0.52)	9.99(0.51)
		BB-off	10.0(0.37)	10.1(0.44)	10.0(0.52)	9.90(0.57)	9.96(0.44)	10.0(0.42)	10.1(0.42)	10.1(0.43)	9.90(0.28)	9.97(0.44)

There were no main or interaction effects for broadband amplitude (all $p > .107$), narrow-band amplitude (all $p > .112$) or peak alpha frequency (all $p > .027$).

Given the failure to find any effects of entrainment we re-examined the initial eyes-open resting baseline alpha amplitudes and conducted a median split to identify those with low ($< 8.5 \mu\text{V}$) resting baselines as research focusing on audio-visual entrainment has suggested that only those with low baseline alpha activity may exhibit entrainment effects in the EEG (Rosenfeld, Reinhart & Srivastava, 1997)².

A re-analysis utilising a 2 (Signal: *BB-on* vs. *BB-off*) x 10 (Time: *1min* to *10min*) repeated measures analysis of variance (ANOVA) was conducted on mean broadband (8-12Hz) amplitude for those with a low resting baseline level. This showed a main effect of Signal, $F(1,4)=13.952$, $p < 0.05$, $Mse=0.203$, $\eta^2=0.777$, showing greater alpha amplitude during the *BB-off* phase compared to the *BB-on* phase ($4.84\mu\text{V}$ and $4.75\mu\text{V}$ respectively)³. There were no other main or interaction effects (all $p > 0.23$)⁴.

Beta EEG

A 2 (Time: *Pre* vs. *Post*) x 2 (Hemisphere: *Left* vs. *Right*) repeated measures analysis of variance (ANOVA) was conducted on mean broadband (18-20Hz) amplitude, mean narrow-band (19.5Hz-20.5Hz) amplitude and peak beta (18-20Hz) frequency separately for eyes open and eyes closed resting baselines.

Eyes open

For both broadband and narrow-band amplitude there was a Time by Hemisphere interaction. Broadband: $F(1,9)=22.224$, $p < 0.01$, $Mse=13.47$, $\eta^2=0.712$; Narrow-band: $F(1,9)=6.776$, $p < 0.05$, $Mse=3.091$, $\eta^2=0.43$. Post hoc comparisons using a Bonferroni correction ($0.05/2 = 0.025$) examined the changes in amplitude from *Pre* to *Post* for *Left* and *Right* hemispheres separately. These showed a marginal increase in amplitude from *Pre* to *Post* for *Left*

² Given the lack of any effects of hemisphere we collapsed the data across right and left hemispheres and focused only on eyes open pre entrainment baselines as the entrainment was also conducted with eyes open. Re-analysis of the entrainment effects was also conducted collapsing across hemisphere given the initial null effects and also to reduce variability given the smaller N.

³ A corresponding analysis on those with high resting baselines showed no evidence of any effect of Signal ($p=0.81$).

⁴ A similar analysis conducted on narrow-band amplitude and frequency revealed no main or interaction effects (all $p > 0.25$).

hemisphere (Broadband: 7.69 μ V and 9.65 μ V $p=0.041$; Narrow-band 3.54 μ V and 4.56 μ V $p=0.032$) but not the *Right* (Broadband: 7.56 μ V and 7.20 μ V $p=0.65$; Narrow-band: 3.74 μ V and 3.65 μ V $p=0.87$). There were no other main effects for amplitude (all $p_s>0.20$) and no main or interaction effects for peak beta frequency (all $p_s>0.54$).

Eyes closed

For both broadband and narrow-band amplitude there were main effects of Time. Broadband: $F(1,9)=20.73$, $p<0.001$, $Mse=34.93$, $\eta^2=0.70$; Narrow-band: $F(1,9)=24.33$, $p<0.001$, $Mse=15.34$, $\eta^2=0.73$. Showing a drop in amplitude from pre to post entrainment (Broadband: from 8.72 μ V to 6.85 μ V; Narrow-band from 4.65 μ V to 3.41 μ V). For narrow-band amplitude only there was also a marginal effect of Hemisphere, $F(1,9)=4.69$, $p=0.058$, $Mse=11.97$, $\eta^2=0.34$, with less amplitude in the *Left* hemisphere compared to the *Right* (4.58 μ V to 3.48 μ V respectively). There were no other main or interaction effects for amplitude (all $p_s>0.11$) and no main or interaction effects for peak beta frequency (all $p_s>0.18$).

Beta Entrainment

A 2 (Hemisphere: *Left* vs. *Right*) x 10 (Time: *1min* to *10min*) x 2 (Signal: *BB-on* vs *BB-off*) repeated measures analysis of variance (ANOVA) was conducted on mean broadband (18-22Hz) amplitude, mean narrow-band (19.5Hz-20.5Hz) amplitude and peak beta (18-22Hz) frequency separately. Descriptives for each of these variables can be seen below in Table 2.

For both broadband and narrow-band amplitude there was a marginal effect of Time: Broadband, $F(9,81)=2.675$, $p=0.082$, $Mse=15.88$, $\eta^2=0.23$; Narrow-band, $F(2.62,23.65)=2.879$, $p=0.063$, $Mse=5.50$, $\eta^2=0.24$. Showing a drop in amplitude from *time-1* to *time-10* (Broadband: 7.52 μ V to 5.65 μ V; Narrow-band 3.41 μ V to 2.19 μ V). There was also a marginal effect of Hemisphere: Broadband, $F(1,9)=4.654$, $p=0.059$, $Mse=159.51$, $\eta^2=0.34$; Narrow-band, $F(1,9)=4.152$, $p=0.072$, $Mse=51.77$, $\eta^2=0.31$. Showing greater amplitude in the *Left* hemisphere compared to the *Right* (Broadband: 6.55 μ V to 5.29 μ V; Narrow-band 2.90 μ V to 2.18 μ V). For narrow-band amplitude only there was also a significant Time by Hemisphere interaction, $F(9,81)=2.149$, $p<0.05$, $Mse=0.83$, $\eta^2=0.19$ [Broadband interaction was $p=0.131$]. This was explored by conducting a 10 (Time: *1min* to *10min*) x 2 (Signal: *BB-on* vs. *BB-off*) repeated measures analysis of variance (ANOVA) on mean narrow-band (19.5Hz-20.5Hz) amplitude for *Left* and *Right* hemisphere separately. For *Left* hemisphere this revealed a main effect of Time, $F(2.35,21.15)=3.684$, $p<0.05$, $Mse=3.95$, $\eta^2=0.29$ with

Table 2. Showing means (SD) for beta broadband (18-22Hz), narrow-band (19.5-20.5Hz) amplitude and peak frequency (18-22Hz) for the ten one-minute binaural-beat on signal entrainment periods and the ten one-minute binaural-beat off carrier tone baseline periods.

			Time Periods									
			1	2	3	4	5	6	7	8	9	10
Broadband amplitude μV	Right hemisphere	BB-on	7.26(4.41)	5.64(3.58)	5.34(3.08)	5.05(2.39)	5.08(2.33)	4.66(1.84)	4.85(2.00)	5.20(2.43)	5.57(2.53)	4.81(1.49)
		BB-off	5.75(2.38)	5.51(3.36)	5.34(3.48)	5.20(2.46)	5.00(2.20)	4.58(2.11)	4.83(2.19)	5.62(2.91)	5.71(2.64)	4.71(1.70)
	Left hemisphere	BB-on	8.75(3.28)	7.00(2.63)	6.37(2.46)	6.11(2.11)	6.87(2.04)	6.61(1.98)	6.02(2.41)	5.53(1.96)	6.30(3.45)	6.05(2.61)
		BB-off	8.32(3.33)	6.79(2.37)	6.24(2.56)	6.60(2.23)	6.70(2.16)	6.23(1.79)	6.11(1.83)	6.23(3.61)	6.59(3.27)	5.56(2.11)
Narrow-band amplitude μV	Right hemisphere	BB-on	3.09(1.87)	2.33(1.29)	2.19(1.05)	2.17(1.08)	2.09(0.85)	1.82(0.67)	1.94(0.64)	2.26(0.96)	2.27(1.05)	1.92(0.54)
		BB-off	2.52(1.05)	2.30(1.36)	2.20(1.30)	2.07(0.84)	1.99(0.74)	1.81(0.66)	1.93(0.95)	2.32(1.42)	2.45(1.09)	1.87(0.53)
	Left hemisphere	BB-on	4.18(1.83)	3.12(1.37)	2.79(1.20)	2.70(0.86)	3.09(1.17)	2.97(1.18)	2.55(1.16)	2.47(0.95)	2.73(1.74)	2.59(1.25)
		BB-off	3.86(1.70)	2.98(1.13)	2.79(1.13)	2.91(1.00)	3.01(1.02)	2.66(0.75)	2.73(0.75)	2.62(1.78)	2.84(1.49)	2.36(0.93)
Peak frequency Hz	Right hemisphere	BB-on	20.1(0.46)	20.1(0.50)	20.3(0.45)	20.3(0.44)	20.4(0.55)	20.5(0.47)	20.4(0.58)	20.4(0.42)	20.4(0.54)	20.4(0.66)
		BB-off	20.2(0.41)	20.3(0.45)	20.3(0.60)	20.5(0.48)	20.5(0.53)	20.4(0.51)	20.4(0.51)	20.3(0.52)	20.3(0.47)	20.2(0.59)
	Left hemisphere	BB-on	20.2(0.45)	20.0(0.60)	20.1(0.52)	20.1(0.51)	20.1(0.53)	20.2(0.40)	20.0(0.44)	20.1(0.46)	20.1(0.46)	20.1(0.41)
		BB-off	20.3(0.42)	20.1(0.47)	20.0(0.56)	20.1(0.53)	20.1(0.49)	20.0(0.47)	20.1(0.43)	20.2(0.43)	20.2(0.40)	20.1(0.43)

pairwise comparisons showing a reduction in amplitude from *time-1* to *time-8* ($4.02\mu\text{V}$ to $2.55\mu\text{V}$; $p<0.05$) and marginal reductions from *time-1* to *time-9* ($4.02\mu\text{V}$ to $2.79\mu\text{V}$; $p=0.087$) and from *time-1* to *time-10* ($4.02\mu\text{V}$ to $2.48\mu\text{V}$; $p=0.095$). There were no main or interaction effects for *Right* hemisphere (all $p_s>0.15$).

For beta peak frequency there was a significant Time by Hemisphere interaction, $F(9,81)=2.117$, $p<0.05$, $Mse=0.082$, $\eta^2=0.19$ but no other main or interaction effects (all $p_s>0.13$). The interaction was explored by conducting a 10 (Time: *1min* to *10min*) x 2 (Signal: *BB-on* vs. *BB-off*) repeated measures analysis of variance (ANOVA) on mean peak beta frequency for *Left* and *Right* hemisphere separately. However, this produced no main or interaction effects for either *Left* hemisphere (all $p_s>0.21$) or *Right* hemisphere (all $p_s>0.13$).

Discussion

Exposure to ten 1-minute segments of either alpha (10Hz) or beta (20Hz) binaural beats produced no clear frequency following effects in the EEG. For those exposed to the alpha binaural beat there was some evidence of a decrease in resting baseline amplitude from pre to post entrainment and during the session participants exhibited greater alpha activity during the binaural-beat off phase compared to the on phase. For those exposed to the beta binaural beat there was some evidence of an increase in resting eyes open baseline beta amplitude for the left hemisphere only. However, during eyes closed resting baseline sessions there was a drop in amplitude from pre to post entrainment. In addition, during the entrainment session there was a drop in amplitude over time with more robust reductions seen for the narrow-band of amplitude in the left hemisphere.

These results provide no clear indication that a frequency following effect was elicited in the EEG following exposure to either alpha or beta binaural beats. A central assumption of the binaural beats technique is that listening to the sounds elicits an entrainment effect in the EEG and in this way various behaviours may be modified (see Vernon, 2009). However, the results of this study would suggest that exposure to 10 interleaved one-minute segments of either alpha or beta binaural beats at the perceived frequency of 400Hz is not sufficient to elicit any such change in the EEG and as such would be expected to have no influence on behaviour. This lack of any clear change in either the alpha or beta EEG frequencies following exposure to binaural beats is consistent with those reporting similar null effects following exposure to theta (Wahbeh et al., 2007) and alpha (Goodin, et al., 2012) binaural

beats. However, this is in contrast to others who have reported changes in the EEG following exposure to delta/theta (Brady & Stevens, 2000; Karino et al., 2004; Karino et al., 2006; Pratt et al., 2010) and gamma (Grose & Mamo, 2011; Schwarz & Taylor, 2005) frequency ranges. These inconsistent findings suggest that it may be too early to label the technique of binaural beats as completely ineffective as there may be a number of factors that mediate the effectiveness of such an entrainment technique. For instance, it may not be possible for each frequency component of the EEG to be equally entrained, or it may be that participants need to attend to the stimuli for an entrainment effect to occur. In addition, the possible changes elicited by the binaural beats may be more widespread and/or not visible at the temporal regions, or the specific nature and duration of the tones themselves may influence the outcome. Each of these points is discussed in turn below.

It is possible that only certain EEG frequencies exhibit a frequency following effect when exposed to binaural beats. Such a possibility would be consistent with those showing changes in the EEG following exposure to low frequency delta (3 and 4Hz) and theta (~6Hz) binaural beats (Karino et al., 2004; Karino et al., 2006; Pratt et al., 2010). In addition, Pratt et al (2010) reported greater changes in the EEG following exposure to a 3Hz binaural beat compared to 6Hz. Such findings could imply that the lower EEG frequencies may be more amenable to change. However, others have reported clear changes in the EEG following exposure to higher gamma (40Hz) frequency binaural beats (Grose & Mamo, 2011). In addition, combined audio *and* visual entrainment techniques have been shown to elicit changes in both the alpha (Moridis et al., 2010; Teplan, Krakovska & Stolc, 2006) and beta (Timmerman, Lubar, Rasey & Frederick, 1999) frequency ranges of the EEG. Such findings suggest a level of cortical flexibility that is evident across all frequency ranges of the EEG and as such it should be possible to elicit entrainment effects in these frequencies.

Alternatively, it is possible that the effects of binaural beats may be attenuated if the tones are not directly attended to. However, Lane et al (1998) reported changes in behaviour following exposure to beta binaural beats when participants remained blind to the presence of the sounds. Unfortunately no EEG measures were taken so it is not clear whether any changes occurred in the EEG. In contrast, Goodin et al (2012) had participants focus their attention directly on the sounds of the alpha binaural beats and respond when they were able to perceive the beats. Nevertheless, such focused perception had no effect on the binaural beats which failed to elicit any entrainment effects in the EEG. Interestingly, Schwarz and Taylor

(2005) found that when a few of their participants who were musically trained attempted to assign musical intervals to the binaural beat carrier tones by attending to them separately no change was evident in the EEG. However, when the same participants later attended to both carrier tones simultaneously a change in the EEG in response to the binaural beats was evident. This would suggest that individuals, at least those trained in music, may need to ‘attend’ to the two tones together rather than separately and as such a ‘degree’ of attention may be helpful. As yet there has been no reported comparison between the effects of attending to binaural beats versus non-attending and as such this represents a possible avenue of exploration for future research.

A further possibility is that the binaural beats used in this study were able to elicit a change in the EEG but that this was simply not evident at the temporal (T3/T4) regions examined here. Despite some reports that exposure to binaural beats leads to changes in the temporal regions EEG (Karino et al., 2006; Pratt et al., 2010) others have found changes at both frontal (Fz: Grose & Mamo, 2011) and central regions (Cz: Hinke et al., 1980; Schwarz & Taylor, 2005). Given the disparity in tones it could of course be that distinct frequencies elicit changes in the EEG at different locations. Nevertheless, future research could address this issue by conducting full scalp recordings to monitor for possible changes across different regions.

A final possibility examined here is that the presentation of continuous 1-minute binaural beat tones is either insufficient or incapable of eliciting changes in the EEG. Such an idea is consistent with the findings of Kennerly (2004) who found evidence of entrainment in the EEG occurring on average five minutes after the presentation of the binaural beats. Hence, a continuous tone of 1-minute may be insufficient in length. However, others have shown that prolonged exposure to binaural beats for 20 minutes and more has also failed to elicit any evidence of a change in the EEG (Goodin et al., 2012, Stevens et al., 2003, Wahbeh et al, 2007). An alternative to the use of continuous tones of varying durations may be to present the binaural beats in short intense bursts. In this way it is the ‘intensity’ of the tone which facilitates the frequency following effect in the EEG. It should be noted that this is a speculative proposition as no research has yet directly addressed this issue. Nevertheless, a number of reports have found that short bursts of binaural beats can elicit clear changes in the EEG. For instance, Hink et al (1980) played tones in short bursts of 100 milliseconds, Schwarz and Taylor (2005) played binaural beats in short bursts of 1200ms, Pratt et al (2010) presented binaural beats for bursts of 2000ms and Grose and Mamo (2011) presented tones in

bursts of a little under 2 seconds. It may be that presenting the binaural beats in short bursts provides a stronger more robust auditory stimulus and as such is more capable of eliciting a response in the EEG. As noted above, this represents a speculative suggestion as no research has directly addressed this question. Nevertheless, future research could address this by directly comparing the effectiveness of binaural beats delivered using continuous tones versus short intense bursts.

In conclusion, presentation of interleaved one-minute binaural beats of alpha and beta failed to elicit any clear evidence of a change in the EEG. This severely limits the potential applications of binaural beats given that a key assumption is that they mediate behavioural change via entrainment of cortical activity. Nevertheless, future research could help to clarify this picture by conducting full scalp recordings and possibly utilising short bursts of binaural beats rather than the continuous and prolonged tones utilised here.

References

Anokhin, A., & Vogel, F. (1996). EEG alpha rhythm frequency and intelligence in normal adults. *Intelligence*, 23(1-14).

Barry, R. J., Clarke, A. R., & Johnstone, S. J. (2003). A review of electrophysiology in attention-deficit/hyperactivity disorder: I. Qualitative and quantitative electroencephalography. *Clinical Neurophysiology*, 114(2), 171-183.

Barry, R. J., Cocker, K. I., Anderson, J. W., Gordon, E., & Rennie, C. (1992). Does the N100 evoked potential really habituate? Evidence from a paradigm appropriate to a clinical setting. *International Journal of Psychophysiology*, 13(1), 9-16.

Bear, D. M. (1979). Temporal lobe epilepsy: A syndrome of sensory-limbic hyperconnection. *Cortex*, 15(3), 357-384.

Boutros, N. N., Belger, A., Campbell, D., D'Souza, C., & Krystal, J. (1999). Comparison of four components of sensory gating in schizophrenia and normal subjects: a preliminary report. *Psychiatry Research*, 88, 119-130.

Brady, B., & Stevens, L. (2000). Binaural-beat induced theta EEG activity and hypnotic susceptibility. *American Journal of Clinical Hypnosis*, 43(1), 53-68.

Carter, C. (2008). Healthcare performance and the effects of the binaural beats on human blood pressure and heart rate. *Journal of Hospital Marketing & Public Relations*, 18(2), 213-219.

David, J. B., Naftali, A., & Katz, A. (2010). Tinntrain: A multifactorial treatment for tinnitus using binaural beats. *The Hearing Journal*, 63(11), 25-28.

Doppelmayr, M., Klimesch, W., Hodlmoser, K., Sauseng, P., & Gruber, W. (2005). Intelligence related upper alpha desynchronisation in a semantic memory task. *Brain Research Bulletin*, 66, 171-177.

Egner, T., & Gruzelier, J. (2003). Ecological validity of neurofeedback: modulation of slow wave EEG enhances musical performance. *NeuroReport*, *14*(9), 1221-1224.

Egner, T., & Gruzelier, J. (2004). EEG Biofeedback of low beta band components: frequency-specific effects on variables of attention and event-related brain potentials. *Clinical Neurophysiology*, *115*, 131-139.

Fuchs, T., Birbaumer, N., Lutzenberger, W., Gruzelier, J. H., & Kaiser, J. (2003). Neurofeedback treatment for attention-deficit/hyperactivity disorder in children: A comparison with methylphenidate. *Applied Psychophysiology and Biofeedback*, *28*(1), 1-12.

Fuentemilla, L. I., Marco-Pallares, J., & Grau, C. (2006). Modulation of spectral power and of phase resetting of EEG contributes differentially to the generation of auditory event-related potentials. *NeuroImage*, *30*, 909-916.

Goodin, P., Ciorciari, J., Baker, K., Carrey, A., Harper, M., & Kaufman, J. (2012). A high density EEG investigation into steady state binaural beat stimulation. *PLoS One*, *7*(4), e34789.

Grose, J. H., Buss, E., & Hall, J. W. (2012). Binaural beat salience. *Hearing Research*, *285*(1-2), 40-45.

Grose, J. H., & Mamo, S. K. (2011). Electrophysiological measurement of binaural beats: Effects of primary tone frequency and observer age. *Ear and Hearing*, *32*(5), 1-8.

Gruzelier, J., Inoue, A., Smart, R., Steed, A., & Steffert, T. (2010). Acting performance and flow state enhanced with sensory-motor rhythm neurofeedback comparing ecologically valid immersive VR and training screen scenarios. *Neuroscience Letters*, *480*, 112-116.

Hanslmayr, S., Sauseng, P., Doppelmayr, M., Schabus, M., & Klimesch, W. (2005). Increasing individual upper alpha power by neurofeedback improves cognitive performance in human subjects. *Applied Psychophysiology & Biofeedback*, *30*(1), 1-10.

Hink, R. F., Kodera, K., Yamada, O., Kaga, K., & Suzuki, J. (1980). Binaural interaction of a beating frequency-following response. *Audiology*, *19*, 36-43.

Jausovec, N. (1996). Differences in EEG alpha activity related to giftedness. *Intelligence*, *23*, 159-173.

Karino, S., Yumoto, M., Itoh, K., Uno, A., Matsuda, M., Yamakawa, K., Sekimoto, M., Kaneko, Y. & Kaga, K. (2004). Magnetoencephalographic study of human auditory steady-state responses to binaural beat. *International Congress Series*, *1270*, 169-172.

Karino, S., Yumoto, M., Itoh, K., Uno, A., Yamakawa, K., Sekimoto, S. & Kagal, K. (2006). Neuromagnetic responses to binaural beats in human cerebral cortex. *Journal of Neurophysiology*, *96*, 1927-1938.

Kennel, S., Taylor, A. G., Lyon, D., & Bourguignon, C. (2010). Pilot feasibility study of binaural auditory beats for reducing symptoms of inattention in children and adolescents with attention-deficit/hyperactivity disorder. *Journal of Paediatric Nursing*, *25*(1), 3-11.

Kennerly, R. (1996). An empirical investigation into the effect of beta frequency binaural beat audio signals on four measures of human memory. *Hemi-Synch Journal*, *14*(3), 1-4.

Klimesch, W., Sauseng, P., & Gerloff, C. (2003). Enhancing cognitive performance with repetitive transcranial magnetic stimulation at human individual alpha frequency. *European Journal of Neuroscience*, *17*(5), 1129-1133.

Lane, J. D., Kasian, S. J., Owens, J. E., & Marsh, G. R. (1998). Binaural auditory beats affect vigilance performance and mood. *Physiology & Behavior*, *63*(2), 249-252.

Lavallee, C. F., Koren, S. A., & Persinger, M. A. (2011). A quantitative electroencephalographic study of meditation and binaural beat entrainment. *Journal of Alternative and Complementary Medicine*, *17*(4), 351-355.

Le Scouarnec, R. P., Poirier, R. M., Owens, J. E., Gauthier, J., Taylor, A. G., & Foresman, P. A. (2001). Use of binaural beat tapes for treatment of anxiety: a pilot study of tape preference and outcomes. *Alternative Therapies in Health & Medicine*, 7(1), 58-63.

Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioural ratings, and WISC-R performance. *Biofeedback and Self Regulation*, 20(1), 83-99.

Lubar, J. F., Swartwood, M. O., Swartwood, J. N., & Timmerman, D. L. (1995). Quantitative EEG and auditory event related potentials in the evaluation of attention deficit/hyperactivity disorder: Effects of methylphenidate and implications for neurofeedback training. *Journal of Psychoeducational Assessment*, 143-160.

Moridis, C. N., Klados, M. A., Kokkinakis, I. A., Terzis, V., Economides, A. A., Karlovasitou, A., Bamidis, P. D., & Karabatakis, V. E. (2010). The impact of audio-visual stimulation on alpha brain oscillations: An EEG study. In: *Proceedings of the 10th IEEE International Conference on Information Technology and Applications in Biomedicine, Corfu, Greece. 3rd to 5th November.*

Monastra, V. J., Lynn, S. L., Linden, M., Lubar, J. F., Gruzelier, J., & La Vaque, T. J. (2006). Electroencephalographic biofeedback in the treatment of Attention-Deficit/Hyperactivity Disorder. *Journal of Neurotherapy*, 9(4), 5-34.

Oster, G. (1973). Auditory beats in the brain. *Scientific American*, 229(4), 94-102.

Padmanabhan, R., Hildreth, A. J., & Laws, D. (2005). A prospective, randomised, controlled study examining binaural beat audio and pre-operative anxiety in patients undergoing general anaesthesia for day case surgery. *Anaesthesia*, 60, 874-877.

Pantev, C., Okamoto, H., Ross, B., Stoll, W., Ciurlia-Guy, E., Kakigi, R., & Kubo, T. (2004). Lateral inhibition and habituation of the human auditory cortex. *European Journal of Neuroscience*, 19, 2337-2344.

Perrott, D. R., & Nelson, M. A. (1969). Limits for the detection of binaural beats. *Journal of the Acoustical Society of America*, *46*, 1477-1481.

Pratt, H., Starr, A., Michalewski, H. J., Dimitrijevic, A., Bleich, N., & Mittelman, N. (2010). A comparison of auditory evoked potentials to acoustic beats and to binaural beats. *Hearing Research*, *262*, 34-44.

Rasey, H. W., Lubar, J. F., McIntyre, A., Zoffuto, A. C., & Abbott, P. L. (1996). EEG Biofeedback for the enhancement of attentional processing in normal college students. *Journal of Neurotherapy*, *1*(3), 15-21.

Rosenfeld, J. P., Reinhart, A. M., & Srivastava, S. (1997). The effects of alpha (10-Hz) and beta (22 Hz) entrainment stimulation on the alpha and beta EEG bands: Individual differences are critical to prediction of effects. *Applied Psychophysiology and Biofeedback*, *22*(1), 3-20.

Saleh, M., Reimer, J., Penn, R., Ojakangas, C. L., & Hatsopoulos, N. G. (2010). Fast and slow oscillations in human primary motor cortex predict oncoming behaviourally relevant cues. *Neuron*, *65*, 461-471.

Schwarz, D. W. F., & Taylor, P. (2005). Human auditory steady state responses to binaural and monaural beats. *Clinical Neurophysiology*, *116*, 658-668.

Tanaka, H., Monahan, K. D., & Seals, D. R. (2001). Age-predicted maximal heart rate revisited. *Journal of the American College of Cardiology*, *37*, 153-6.

Teplan, M., Krakovska, A., & Stolc, S. (2006). EEG responses to long-term audio-visual stimulation. *International Journal of Psychophysiology*, *59*(2), 81-90.

Teplan, M., Krakovska, A., & Stolc, S. (2011). Direct effects of audio-visual stimulation on EEG. *Computer Methods and Programs in Biomedicine*, *102*, 17-24.

Timmerman, D. L., Lubar, J. F., Rasey, H. W., & Frederick, J. A. (1999). Effects of dominant and twice-dominant alpha audio-visual stimulation on the cortical EEG. *International Journal of Psychophysiology*, 32, 55-61.

Valentino, D. A., & Dufresne, R. L. (1991). Attention tasks and EEG power spectra. *International Journal of Psychophysiology*, 11, 299-302.

Vernon, D. (2009). *Human Potential: Exploring Techniques Used to Enhance Human Performance*. London: Routledge.

Wahbeh, H., Calabrese, C., Zwicky, H., & Zajdel, D. (2007). Binaural beat technology in humans: a pilot study to assess neuropsychologic, physiologic, and electroencephalographic effects. *Journal of Alternative and Complementary Medicine*, 13(2), 199-206.