

The Impact of Contextual and Non-contextual Noise and Anxiety on Brain Signaling

By

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Introduction

Significance

Noise pollution is a pervasive problem that exists in all areas of our daily lives. Although people can become accustomed to noise levels, it never completely removes its negative ramifications [1]. While urban areas are the major focus, noise has become a ubiquitous exposure that exists in daily life. This is due to the modernization of society which has led to more machines in daily life such as cars. Noise exposure can cause health problems for everyone no matter the environment. The effects of noise pollution can be negative social behavior and annoyance, interference with spoken communication, cardiovascular disturbances, and disturbances in mental health [1-2].

What is Noise?

Noise is described as an unwanted sound and can come in a variety of sources such as traffic, construction, and airports. These are examples of man-made noise which are created in urban environments. Also due to habituation certain noise exposures can become more impactful due to moving into a new noise environment. Noise is measured in decibels (dBA), which is the intensity of the sound, but can also have different frequencies, which is the pitch of the noise which is measured in Hertz (Hz). Previous studies have found that high dBA levels have the greatest impact on health [3]. Details of other noises by comparison are in (Figure 1).

Noise and Health Effects

These findings suggest that chronic or prolonged exposure above a certain noise level negatively impacts health and wellbeing [1,3]. Some of these thresholds include 55 dBA for stress related health, 65dB cardiovascular effects, and 70 dBA for hypertension and heart disease [3,4]. An example of a sound within these thresholds is traffic. The short-term impacts of noise

exposure are increased heart rate, blood pressure, and secretion of stress hormones [1,3,5]. These are our “fight-or flight” responses. This means that noise exposures are consistently putting us in an emergency mode which can cause many negative effects. This increased use of “fight-or flight” and other factors increases the risk of developing cardiovascular disease. Many of these connections were originally made in the occupational setting, but these effects have also been observed in non-occupational settings [6]. While observing sleep data, noise exposure also increases blood pressure and heart rate [5]. The proposed pathway that sleep noise exposure interacts with is through the central nervous system [1]. While this study does not look at those who are asleep, we look at those in a restful environment which experience noise exposure similarly.

EEG

Electroencephalogram or (EEG) is a test that measures brain activity in micro voltages using metal disks around the brain in a headset. It specifically measures the electrical signals that brain cells use to communicate with each other. There is detail of an EEG headset in (Figure 2). EEG, when used as a measure, has different sampling rates from 1-300 Hz which are then used to generate multiple different waves inside the brain [7]. These different waves are beta, alpha, delta and theta, and each wave corresponds to a different Hz range, delta is less than 4 Hz, theta is between 4-7 Hz, alpha is between 8-15 Hz, and beta is between 16-31 Hz [8]. Along with this each brain wave measures different activity, delta waves are associated with slow sleep waves, theta waves are associated with drowsiness and idling, alpha waves are associated with relaxed and reflecting, and beta waves are associated with calm to obsessive reactions, active thinking, and anxious behavior [8]. This information allows us to define noise exposures impact based on brain activity.

EEG and Noise

EEG has been used in multiple studies to examine the impact of noise exposures on brain functioning, particularly neural systems involved in the stress response. For example, EEG has been used to measure the impact of traffic and construction noise on brain functioning [9,10]. Noise from traffic has been linked to EEG activity in the frontal, temporal, and parietal lobes of the brain [9]. In this study of 12 participants 8 males and 4 females ages 22 to 28 years old in New Delhi, India, both delta waves and alpha waves decreased as participants were exposed to traffic noise events such as the use of a horn [9]. Many studies looked at pure tone noise and its effect on EEG signaling. In this 16-participant study with 8 men and 8 women there were changes in delta, beta, and alpha waves [11]. This study observed that delta waves increased based off pure tone and time in all scalp regions, alpha wave activity increased in the right hemisphere of the frontal region, and beta wave activity was higher in all regions of the brain [11]. Another study looked at the subjective power of brain signaling when compared to long exposure white noise. This study of 12 participants ages 20 to 24 found an increase in alpha waves in the occipital region, and an increase in delta waves in the frontal region [12]. Another study investigated the effect of noise on EEG brain wave signaling. In this study of 15 men between the ages of 25 to 30 with no history of hearing impairment, and showed no signs of psychiatric or neurological disorders, beta activity increased in both low frequency and high frequency pure tones, and alpha activity decreased in both low and high frequency tones [13]. The impact of construction noise on physiological stress responses has also been studied. For example, research has linked construction noise to heart rate variability, respiratory rate, and reactivity in the frontal and temporal lobes, which varied depending on the type of construction

noise (e.g., jackhammer, bulldozer, and saw) [10]. These findings establish that EEG is an accurate and appropriate measure of physiological impact of short-term noise exposure. Some of this work establishes a proposed connection between the noise exposure of traffic and construction on our emotional and stress responses.

While these studies provide important information, many do not account for other covariates such as habituation or other mental illness. They also do not directly test the frequencies alone of traffic and construction to examine their effect on EEG signaling.

Mental Illness and Habituation

Mental illness such as anxiety could cause stronger reactions to noise exposures which would modify the impact of noise exposure on brain activity [14]. This was observed in a study in which those with generalized anxiety had higher gamma wave activation, and when treated this gamma activation dropped to comparable levels of the control group [14]. While habituation, defined as the diminishing of a physiological or emotional response to a frequently repeated stimulus has not been studied as broadly, it has been indicated that the negative ramifications of the exposure remain [1]. When studying noise exposures and their impact on EEG, these two variables- noise habituation and mental illness- should be accounted for when studying EEG because of the indirect impact that both have on brain activity.

Thesis Study

Given the previously mentioned limitations of prior studies examining the impact of noise exposure on brain activity, this study seeks to expand on the prior research by: (1) considering how both contextual and non-contextual sound may impact brain activity, (2) accounting for participants noise environment a potential proxy for habituation and (3) how anxiety may modify that impact.

Methods

Study Population

This study is an analysis of a non-randomized experiment performed in Boston, Massachusetts from February to March 2020. This study included 22 men and women aged 18 years and older who resided in the Greater Boston Area. Participants were recruited on a volunteer basis in which they contacted us through either setting up a phone appointment through scanning a QR code or sending an email. Each participant received a pre-screening interview that screened for eligibility. People with present or past psychiatric, neurological, or audiological disorders, and doctor diagnosed cardiovascular diseases were excluded. Also anyone who currently used drugs that would effect EEG were excluded from the experiment, and that list included: Marijuana/THC, LSD, PCP, Barbiturates, Morphine, Opiates, Heroin, Alcohol, Neuroleptics (e.g., chlorpromazine, thioridazine, clozapine, Clozaril), Anxiolytics, Hormones, Antidepressants (e.g., Amitriptyline, Imipramine, Iproniazid, MAO Inhibitors, SSRIs), Stimulants (e.g., Amphetamines, Methylphenidate, Caffeine, Nicotine, Cocaine), Antimanics (any drug that stabilizes mood by controlling symptoms of mania, the abnormal psychological state of excitement), and Tuberculostatic (any drug that halts the growth of tubercle mycobacteria). This list was included due to certain drugs ability to effect EEG data [15,16].

Study Procedures

This human subjects study was approved by the Boston University Medical Center Institutional Review Board. Participants attended a single study visit to measure EEG activity in response to noise. Participants while wearing the EEG headset were required to listen to

contextual and non-contextual noises at 55 dBA, and these noises were “annoying (high pitch buzzing sound)”, “traffic”, “construction”, “nature”, “airport”, and “ambulance”. The present study focused on traffic, construction, and nature noises. Contextual noise are noises which sound exactly as described, while non-contextual noise takes the dominate frequencies of those noises and distorts the context. Before exposure, participants had 2 minutes of silence before the first noise of interest started. The noise exposure was continuous and lasted for 2 minutes which made a soundtrack of 26 minutes and 22 seconds. EEG captured data at the rate of one three hundredth of a second or 300Hz. This gave the study 474,600 data points per participant. Details of study soundtrack are in (Figure 3). Brain activity was measured with a Wearable Sensing (16744 West Bernardo Drive. San Diego, CA 92127) DSI 24 with 21 nodes and 3 auxiliary channel (eyes, muscle, and heart rate variability) dry EEG headset in Hz and microvolts. Measurement of brain activity started to be recorded as soon as the EEG headset was fitted, and the soundtrack began to play.

In addition to the experiment described above, at the beginning of the study participants also completed a self-report survey at the visit. The questions were sociodemographic and include questions about address, length of stay at current address sex, age, race, educational attainment, children, any disabilities, any medications being taken, how much time spent working, type of employment, industry of employment, if you are in a relationship, if you live with your partner, religious views, total income, and social class.

Measures

The effect modifiers of this experiment were anxiety level and sex. Both measures were collected at the beginning of the experiment through self-report surveys. Sex was measured by asking participants “How do you currently describe your gender identity” and was dichotomized

as male and female. The 7-item Generalized Anxiety Disorder survey (GAD7) was administered to measure symptoms of generalized anxiety occurring in the past 2 weeks on a 4-point scale ranging from not at all (0) to nearly every day (3) [17]. When screening for an anxiety disorder, a recommended cut-off point for further evaluation is 10 or greater [17]. We categorized GAD-7 scores into 4 groups based on thresholds of 5, 10, 15, and 20.

The confounder of our study was neighborhood noise level. Neighborhood noise level was obtained from a Boston sound model provided from a prior study [18]. This model provided predictions of a-weighted sound levels for every parcel in Boston. Which were then averaged to the census block group. The noise of a participant's census block group, which was used as a proxy for neighborhood, was classified into three groups 0,1, and 2. These category ranges were below 55 dBA, 55 dBA and above, and 65 dBA and above respectively. These thresholds were obtained from well-established prior literature in which physiological impact occurred at a minimum of 55 dBA [3].

The outcome of interest was the power of each of the 21 EEG nodes on the headset. Each EEG node measured a specific region such as F for frontal, Fp for prefrontal cortex, P for parietal, T for temporal, and O for occipital, and these were compared to reference nodes such as Cz. Power is the squaring of the micro voltage amplitudes provided by the EEG measurements [9]. Each one of these nodes took continuous measurements of micro voltages inside the brain while the noise exposure was played through earphones.

Statistical Analysis

Separate datasets were created for each exposure type Traffic contextual, Traffic non-contextual, Construction contextual, Construction non-contextual, Nature contextual and Nature

non-contextual. This data was filtered for artifacts such as muscle movements and eye movements. An average was taken of overall activity in each node, and then squared to perform a power analysis on each node in the EEG headset [7,12]. This data for each participant was then combined to form six different data sets each corresponding to what noise exposure produced the results.

We used T-tests and ANOVAs using Tukey analysis to compare EEG measurements within groups of anxiety. All analysis will include adjustment for sex and neighborhood noise level. This gave us a mean difference between each group within analysis for power output for each node on the EEG headset. Significance levels were set to alpha 0.05. Analyses were conducted in R version 1.4.1717 [19].

Results

Sociodemographic characteristics of the study sample are shown in Table 1. In this study most of the population were women (90.9%) compared to (9.1%) being men. The most represented racial group in this study were white people representing (50%) of the study other racial groups such as black, Hispanic, Asian and mixed had (13.6%), (13.6%), (4.5%), and (18.2%) respectively. Most participants had neighborhood noise levels below 55 dBA (45.5%). Above 55 dBA and 65 dBA had (36.4%) and (18.2%) respectively. The most represented GAD7 anxiety score was 0-4 comprised (77.3%) of participants while 5-10 and 11-15 comprised (18.2%) and (4.5%) respectively.

All results are controlled for neighborhood noise level. Women had stronger responses in Cz and O2 nodes when listening to Nature Non-contextual and Construction Non-contextual noises, respectively when compared to men. While in Traffic Contextual noise those with

moderate anxiety had a stronger response in nodes T5 and X2. We can imply that this means anxiety is an effect modifier of the impact of noise exposure on brain activity. There were no statistically significant nodes within any study covariates in traffic non-contextual, construction contextual, and nature contextual noise exposure. These exposures will only be compared to either their contextual or non-contextual counterparts. Only significant results are reported in Table 2.

Discussion

In a study of 20 women and 2 men in greater Boston we were able to: (1) consider how both contextual and non-contextual sound may impact brain activity, (2) account for participants noise environment a potential proxy for habituation and (3) determine how anxiety may modify that impact. Results from this study show that there are differences between contextual and non-contextual exposures to noise.

When exposed to non-contextual construction noise, node O2 was statistically different within levels of sex. This implies that there are differences between men and women when perceiving construction frequencies. When exposed to contextual traffic noise pollution the nodes T5 and X2 were statistically significant within anxiety levels. This means that those with moderate anxiety were more likely to have more activity within these nodes of the EEG headset. This finding aligns with the literature of differences within the temporal lobe [9]. When exposed to contextual nature sounds there were no statistically significant differences between groups of anxiety and neighborhood noise level. While when exposed to non-contextual nature noise, the node Cz was statistically significant within sex.

Previous research looked at specific types of noises such as honking events in traffic [9]. This analysis of the data looks at traffic noise as a whole and came to somewhat similar results

in which the temporal lobe showed significant differences in activation when exposed to traffic noise. This trend ends with construction noise and nature noise. Responses to Contextual construction noise did not align with previous literature as we did not observe significant activation in the frontal lobe. What differs is that non-contextual noise only shows significant activation in the occipital lobe. Due to non-contextual noise being the frequencies that compose construction noise we would expect similar activation in exposures. Along with this finding, previous studies have chosen to separate construction noises, but our soundtrack combined multiple types of construction noises. For example, we considered jackhammer, bulldozers, and vehicle beeping in one soundtrack when others looked at them individually.

When observing contextual nature noise exposure there were no statistically significant nodes, and this could be due to proposed effects of nature sound. Nature sound has been linked to improved recovery after psychological stressors [20]. This could explain why we observed no increased activation in any of the lobes of the brain in response to nature sounds. Non-contextual nature noise exposure seems to have a different effect due to significant activation in node Cz. While this activation was seen in the reference node, this could imply that activity in all the regions was higher or lower on average. This could be due to the underlying frequencies of nature sounds, or the recognition of nature as relaxing is removed once it is unrecognizable.

Strengths and Limitations

Our study has some marketed strengths. Our study was an experimental trial, this being a gold standard for reporting results. This study also accounts for a participant's everyday neighborhood noise exposure. Many studies do not account for the habituation of participants

that return home to higher levels of noise in their daily life. This study looks at the participants beyond just the experimental noise exposure and accounts for some of the effects of habituation.

Limitations of this study are a butter bandpass filtering lack of power, the restrictive exclusion criteria, and the EEG protocol itself. This study does not utilize butter bandpass filtering which is a recognized way of EEG analysis. This process usually cleans data to a specific frequency and allows for direct analysis of specific brain waves. If this study was performed butter bandpass filtering would clean results further and allow for better analysis. The study itself contained only 22 participants and due to the modifiers of the experiment significantly reduced the power of the study. While the covariates are necessary for analysis, it stratifies upon a small group of participants that could cause a small impact on readings to appear larger. Along with this, outliers are difficult to expose due to the small sample size and having large variation in the dataset without multiple comparison points. Such as having only one person with an anxiety score of 11, when having multiple could determine if a measurement was an outlier. To continue with the last point, due to the exclusion criteria removing those with psychiatric conditions it removed our chances of capturing a wider variety of GAD-7 scores. Studies which included these participants could see drastically different results. Lastly, the EEG soundtrack and protocol could use more periods of rest instead of continuous noise. The soundtrack would switch between one type of noise exposure into the next without a period of rest for the participant to relax again. This could cause some of the effects to blend together at the transition points. This could also explain how some exposures had no effect because of the continuous nature of the exposure. The last issue of the protocol is a lack of repeated measures. The data was generated from one set of measurements in which a participant only had one 26-minute session. A better study would use repeated measurements such as triplicates, to get an

average effect of noise exposure on each participant. To remedy these issues in the experiment of this type, noise exposure should occur for a set interval, and then a rest period should be provided. This would allow for multiple measurements, and a more accurate judgment of different types of noise exposures.

Future Work

Future studies should continue to look at both contextual and non-contextual noises. This study provides a basis in trying to understand the differences between the underlying frequencies of a noise compared to an identifiable version of the noise. While this study compared multiple different types of contextual and non-contextual noises, future studies should focus on one noise exposure to clarify all its different aspects. Studies should include measures for neighborhood noise level to adjust for those who have become habituated to certain noise levels and exposures. Lastly further questions and surveys should be developed to ascertain the common noise exposures that people in urban and suburban areas experience in their daily lives.

Conclusion

Noise exposure still needs to be studied further to come to more accurate and conclusive health and wellbeing effects. This is due to other aspects of noise exposure that can be studied such as contextual and non-contextual noise in which differences should be studied further and in more detail. More efforts should be used to evaluate how noisy environments that participants live in could affect their different reactions to noise exposures. Overall EEG is still an appropriate way to measure noise exposure non-auditory effects, and non-contextual noise could offer more insight into physiological aspects of noise exposure.

Figure 1. Different activities with decibel readings

Sound	Loudness (dBA)
Breathing	10
Whisper	30
Moderate Rainfall	50
Normal Conversation	60
Traffic	70
Police Siren	80
Jet Plane	120
Jackhammer	130

Figure 2: EEG headset node reference

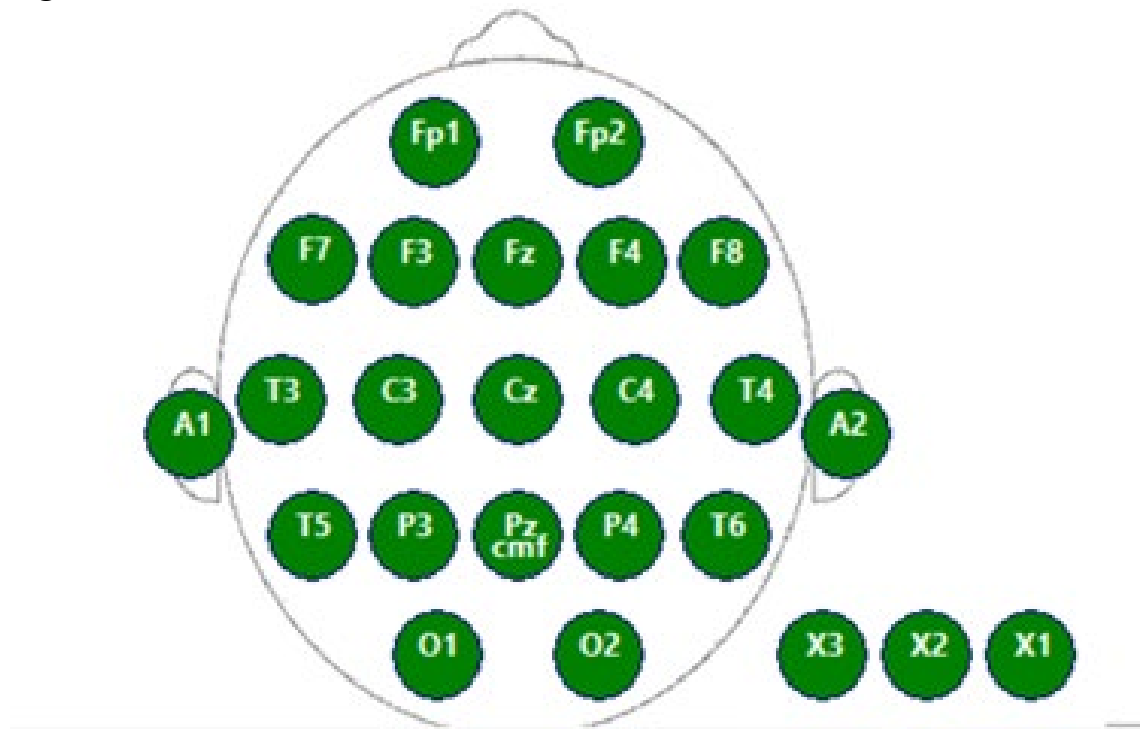


Figure 3: Timeline of soundtrack exposure from beginning of EEG study until the end.

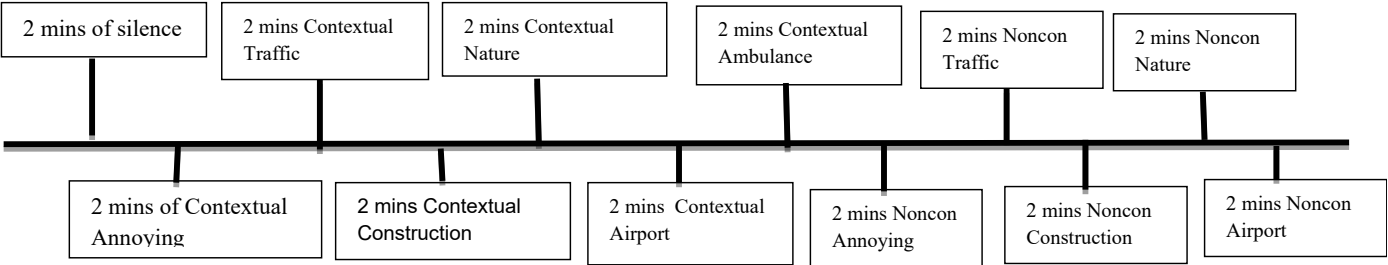


Figure 4: Hypothesized Directed Acyclic Graph of Noise Exposure and Brain Signaling

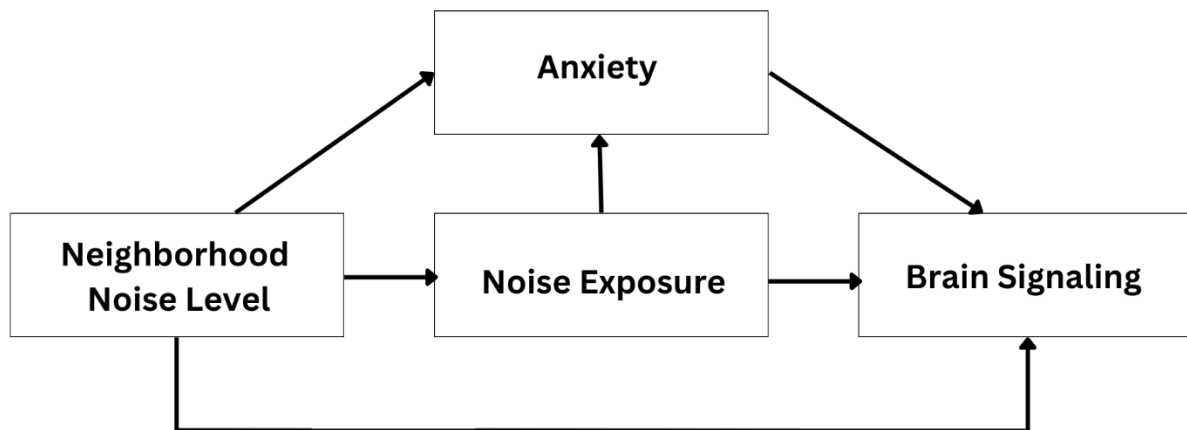


Figure 5: Data Cleaning and Analysis Process

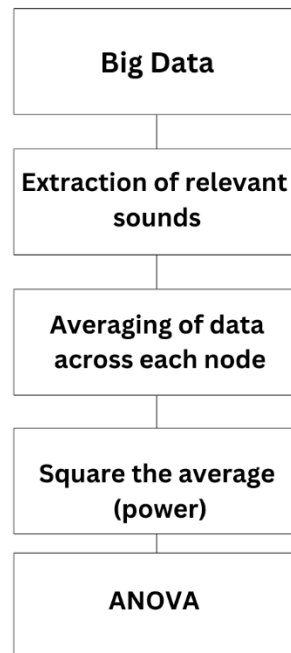


Table 1. Characteristics of Study Population	
Sex	n(%)
Women	20 (90.9%)
Men	2 (9.1%)
Race	
Asian	1 (4.5%)
Black	3 (13.6%)
Hispanic	3 (13.6%)
Mixed	4 (18.2%)
White	11 (50.0%)
Neighborhood Noise Level	
Below 55 dBA	10 (45.5%)
55 dBA	8 (36.4%)
65 dBA	4 (18.2%)
GAD-7 score	
0-4 (no/low anxiety)	17 (77.3%)
5-9 (mild anxiety)	4 (18.2%)
10-15 (moderate anxiety)	1 (4.5%)

Table 2 Tukey Analysis of Group Differences Adjusted for Neighborhood Noise Level			
Noise Exposure	Nodes	Comparison Groups	Difference Value
Traffic Contextual	T5	Anxiety moderate-no/low	90.23*
		Anxiety Moderate-mild	90.00*
	X2	Anxiety moderate- mild	$9.81 \times 10^{-15}*$
		Anxiety moderate-mild	$9.55 \times 10^{-15}*$
Construction Non-contextual	O2	Sex Female-Male	-12.48*
Nature Non-contextual	Cz	Sex Female-Male -1.01	-1.01*
P-value 0.05 * For statistically significant results only			

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