

# The mental and physical effects of riding a motorcycle

## *Catalyst Agency LLC*

The brain's response to external stimuli is known to vary with internal state. While low-level processing of auditory stimuli has been shown to vary in low-intensity laboratory conditions, it is not clear how sensory modulation varies during the physical and attention-demanding task of riding a motorcycle. We here investigated how riding a motorcycle affects "pre-attentional" sensory processing using an auditory oddball task and the electroencephalography signals it evokes: the N1 and mismatch negativity. We additionally investigated how riding a motorcycle modulates the concentrations of hormones, including cortisol, DHEA-S, testosterone, and epinephrine. We report that the N1 amplitude—the brain's response to auditory tones—was significantly reduced during motorcycle riding relative to baseline recordings, and that the mismatch negativity (the change in N1 in response to unexpected tones) was greater during riding. We additionally report that riding significantly increased both the level of catecholamine and the ratio of DHEA-S to cortisol. Together, these results suggest that riding increases attention by strengthening focus and heightening the brain's passive monitoring of changes in the sensory environment while reducing the immediate stress response.

## Introduction

For years, anecdotal reports have suggested that riding a motorcycle may have beneficial effects on riders' brains and physiological states. However, the nature of these effects has not been characterized. Here, in an experimental setting, we tested several possibilities. First, we predicted that if riding enhances focus, then riders should be **less** sensitive to distractions while riding than in a non-riding condition. Second, if riding heightens sensory processing, then riders should be **more** sensitive to changes in their sensory environment while riding.

To address these hypotheses, we used electroencephalography (EEG) to measure brain responses to auditory tones presented passively during riding. Because auditory brain responses are well characterized (Risto Näätänen, Kujala, & Winkler, 2011; R. Näätänen, Paavilainen, Rinne, & Alho, 2007), auditory tones are a reliable tool for passively probing sensory and attention processing during riding. For instance, approximately 100 ms after a tone, EEG shows a distinct response (called N1) associated with activity of the brain's auditory cortex in processing that tone. This response can be enhanced in two ways. First, the response is stronger if the listener attends closely to the auditory tones, and is weaker if the tones are ignored. If the act of riding a motorcycle effectively focuses the rider's attention on the visual modality at the expense of the auditory modality, then we would expect a weaker response while riding (Alain & Arnott, 2000; Sussman, 2007). Second, the response is enhanced if the

brain detects a sudden change in the auditory environment (e.g., a sudden noise from a glass shattering in an already-noisy cafeteria) (Garrido, Kilner, Stephan, & Friston, 2009; R. Näätänen et al., 2007). This orienting response can be thought of as a heightened sensory state.

Combining these two modes of attenuation, we would expect motorcycle riders, relative to car drivers, to present a weakened overall N1 to auditory tones because riders must pay more attention to visual inputs than auditory ones; at the same time, regardless of attention, we would expect riders to display a stronger change in N1 upon the introduction of auditory irregularities, consistent with a heightened state of auditory processing. Finally, we tested whether these brain changes are associated with an increase in physiological arousal and decrease in the stress response, as reported anecdotally by riders.

## **Methods & Materials**

### **Participants**

77 participants (23 female, age  $42 \pm 14$  years) were recruited from Southern California using an IRB-approved recruitment flyer. Participants were required to answer screening questions. Inclusion criteria were as follows: between 18 and 70 years old, neurologically healthy, not taking antipsychotics, and comfortable riding a motorcycle (on a 5-point scale, we accepted participants who rated their comfort as a 3 or greater). We conducted no validation of participants' self reports. Following exclusion of data due to noise, artifacts, and acquisition errors, the final sample comprised 42 participants.

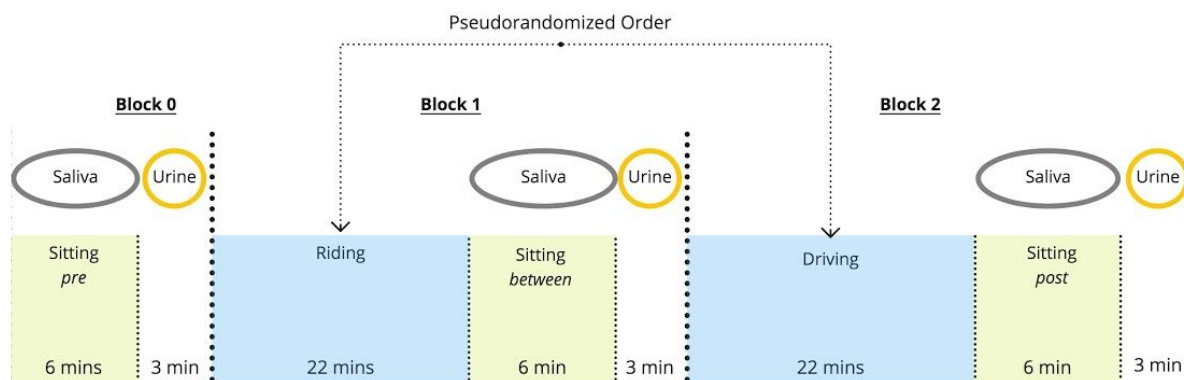
The protocol was approved by IntegReview Independent Review Board Services ( <https://integreview.com/> ), and all participants provided written informed consent.

### **Stimuli and Experimental Design**

Participants performed the oddball task during both riding and non-riding conditions. The oddball task is a simple paradigm in which auditory tones are presented to both ears at regular intervals (on average, 1.5 seconds apart). The task is passive, meaning that participants do not respond to each stimulus. The tones occur at both high and low frequencies, and critically, the ratio of the frequencies is uneven (70% vs. 30%). This design defines the less-frequent tones as the "oddball" stimuli, nested within "standard" stimuli. This design is validated to elicit an N1 responses to all stimuli, corresponding to the auditory brain response, as well as a mismatch negativity (MMN) response to the oddball tones. The MMN is a difference wave; it reflects a change in the N1 during the oddball stimuli relative to the standard stimuli. This design thus tests the auditory brain response to the auditory tones, and pre-attentive sensory processing to the oddball stimuli. The oddball task was presented during riding and non-riding conditions. After 6 minutes of the oddball task, participants had 1 minute without tones.

All participants rode their own motorcycle and drove a provided car (Lexus NX200) on a set route under normal driving conditions. The order of these two blocks was pseudo-randomized to eliminate temporal effects. Before and after the driving and riding blocks, participants sat in a chair, immobile, and provided saliva samples. Participants provided a urine sample after all three immobile conditions (**Figure 1**). This experimental design allowed our analysis to draw inferences about the effects of riding/driving during the activity itself as well as afterward, all while controlling for baseline levels of activity. By design, there were no intersubject variables.

The experiment was conducted at two separate locations: Angeles Crest Highway outside Los Angeles, and Mesa Grande at Lake Henshaw. Both routes took approximately 22 minutes, round trip, to complete.



**Figure 1: Experimental Design.** Block durations reflect planned time, not actual time.

## Equipment

Brain activity and heart rate data were recorded using a 64-channel EEG cap and 2-channel active electrocardiogram (EKG) from the eego sports package by ANT Neuro (<https://www.ant-neuro.com/>).

Hormone concentration was determined by liquid chromatography with tandem mass spectrometry (LC-MS/MS) from saliva and dried urine samples. Hormone concentrations from urine samples were normalized for kidney output by corresponding creatinine (Cr) levels.

## EEG Preprocessing & Analysis

All processing of the EEG data was performed in Matlab (v.R2018b, Mathworks Inc.) using EEGLAB software (v.14.1.2) (Delorme & Makeig, 2004). Power was computed using EEGLAB spectopo. All data was processed according to the following five steps.

(1) EEG data was down-sampled to 250 Hz and trimmed to remove any pre- or post-recording signals, dominated by task-unrelated noise.

(2) A high-pass filter (.75 Hz) was applied to remove artifacts due to slow drifts.

(3) The Artifact Subspace Reconstruction (ASR) algorithm (Chang, Hsu, Pion-Tonachini, & Jung, 2018) was applied to first identify and then remove bad channels, as well as to remove extreme artifacts in the data. Our parameters dictated that channels should be removed if they (i) contained more than 15 minutes of flat line data or (ii) failed to meet a correlational threshold (i.e., a correlation  $> 0.7$  with other channels for a majority of the data). The ASR algorithm is a PCA-like algorithm that constructs a subspace representation of artifact-free data to create a reference. The algorithm uses this reference to identify windows along the time series that depart from this subspace statistically, indicating the presence of artifact; these windows are then reconstructed based on the clean data. The key parameter in this approach is the definition of artifact, which we based on the number of standard deviations by which a window deviated from the reference data. Based on a formal assessment (Chang et al., 2018), we adopted a threshold of 100 standard deviations, which provides a very conservative approach that identifies only the most extreme artifacts. Critically, because this algorithm operates within a moving window (1–2 seconds in width) along the time series, it is capable of identifying non-stationary, extreme artifacts that are not easily removed by any other traditional approach. Thus, we used the ASR algorithm to eliminate large transient artifacts (like motion, which we expect from children) while preserving the data. The technique was developed for real-time, brain-computer interface applications, making it well-suited to our experiment. The cleaned data were re-referenced to the average reference.

(4) Following the removal of gross, transient artifacts, we used independent component analysis (Extended Infomax Algorithm) (Lee, Girolami, & Sejnowski, 1999; Makeig, Jung, Bell, Ghahremani, & Sejnowski, 1996) to identify remaining artifacts that were more stationary in nature: (i) eye blinks and lateral movements, (ii) pulsation, and (iii) any remaining channel deviations.

(5) Finally, from the cleaned data, we extracted features of interest. The N1 was calculated by extracting 1-second epochs from -100 ms to 900 ms following each auditory stimulus. These epochs were averaged for oddball auditory tones, and separately for standard auditory tones. Mean baseline (-100–0 ms) voltage was subtracted from each average event-related potential (ERP), and the peak amplitude of the N1 was extracted to index the auditory response of each individual for each type of auditory tone (oddball/standard). The mismatch negativity (MMN) was calculated as the peak in the difference of these waveforms.

(6) We analyzed N1 responses with a general linear model that included two factors—state (riding or driving) and stimulus (oddball or standard)—to test for main effects of riding on

auditory responses. Additional paired sample t-tests were used to evaluate the effect of riding on the MMN response.

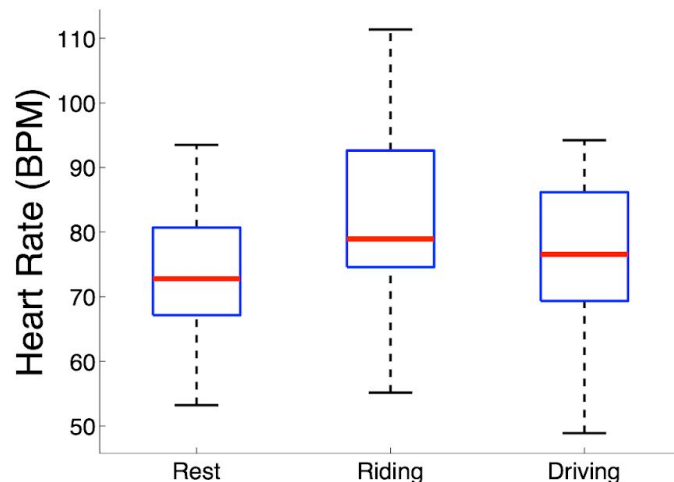
## Statistics

All tests of statistical significance employed paired, parametric t-tests or F-test, unless otherwise noted. Missing samples were excluded rather than imputed, and thus, degrees of freedom may vary slightly between tests even within the same modality. Effect size and significance were computed by mean.

## Results

### Heart Rate

After data exclusion due to electrode detachment, excessive noise, and acquisition errors, we retained useable EKG data from 38 participants. Heart rate in the riding condition was significantly different from rest ( $\mu = 8.7$  BPM,  $t(37) = 4.7$ ,  $p < 10^{-4}$ ) and driving ( $\mu = 5.0$  BPM,  $t(37) = 3.2$ ,  $p < 0.01$ ), corresponding to increases of 11% and 7%, respectively (**Figure 2**).



**Figure 2: Heart Rate.** All data are paired. Each red line indicates the median, blue marks the interquartile range, and black denotes the most extreme non-outlier. Heart rate, shown here in beats per minute (BPM), was significantly different during riding than both resting and driving.

---

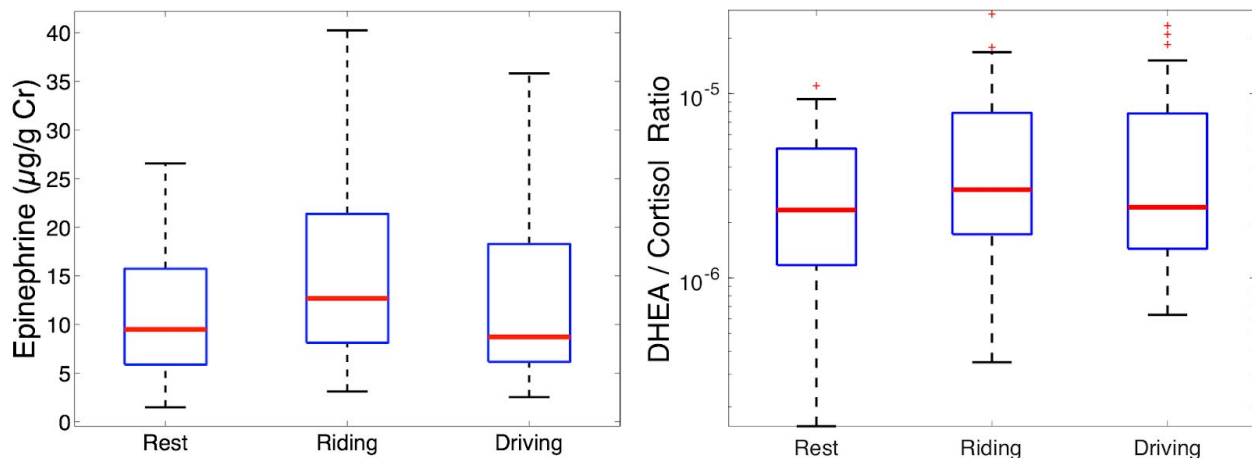
### Hormones

After data exclusion due to sample dilution and acquisition errors, we analyzed the urine data from 43 participants and salivary data from 46 participants.

Riding significantly increased levels of the catecholamine, epinephrine, by 27% relative to baseline ( $\mu = 15.0 \mu\text{g/g Cr}$  vs.  $\mu = 11.6 \mu\text{g/g Cr}$ ,  $t(40) = 5.6$ ,  $p < 0.05$ , **Figure 3A**). Driving did not increase epinephrine relative to baseline ( $\mu = 12.4$ ,  $t(42) = 1.2$ ,  $p = 0.23$ ).

Riding significantly decreased levels of cortisol by 28% relative to sulfated dehydroepiandrosterone (DHEA-S) ( $\mu = 0.177 \log$ ,  $t(42) = 4.5$ ,  $p < 10^{-4}$ , **Figure 3B**). This ratio is an indicator of stress (Buford & Willoughby, 2008; Morgan et al., 2004; Ritsner et al., 2004; Warnock et al., 2010), and thus its decrease can be interpreted as a reduction in stress. We did not collect longitudinal data to confirm or refute the transient or long-lasting nature of this hormonal shift.

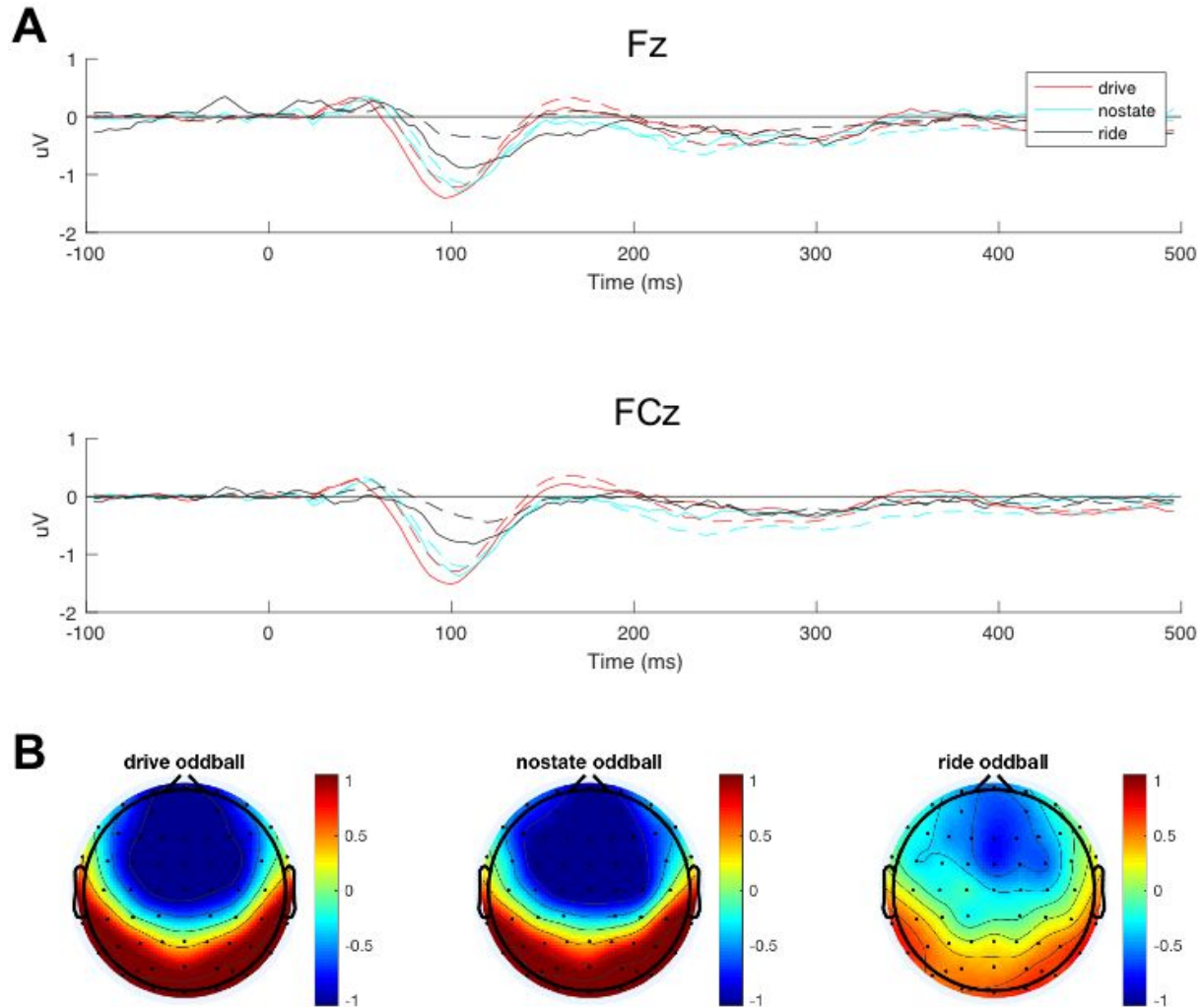
We observed no significant change in salivary testosterone levels.



**Figure 3: Hormone Testing.** All data are paired. Each red line indicates the median, blue marks the interquartile range, and black denotes the most extreme non-outlier. **A.** Changes in epinephrine urine concentration as a function of activity condition. Riding is significantly different than rest. **B.** Changes in the DHEA-S to cortisol concentration ratio as a function of activity condition. By mean, riding is significantly different than rest.

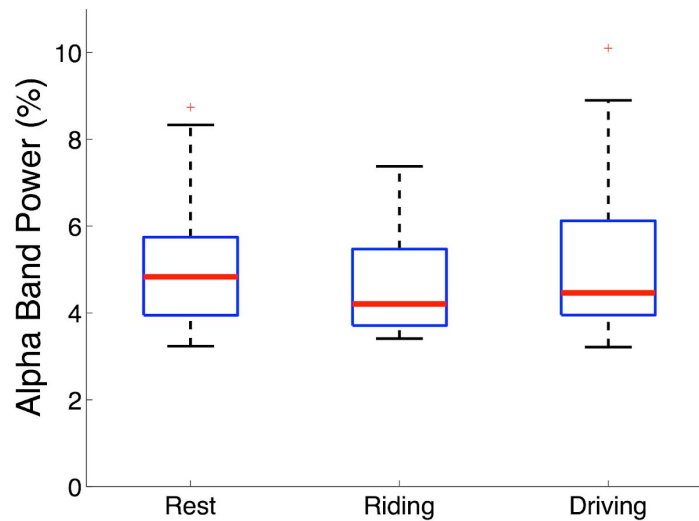
## EEG

The N1 (i.e., the brain's response to auditory tones) was significantly reduced during riding relative to baseline recordings ( $\mu = -1.32 \text{ uV}$  vs.  $-.62 \text{ uV}$ ,  $F(1,40) = 18.5$ ,  $p < .0001$ , **Figure 4A**). The whole-brain response 100 ms after the onset of the tone in each of the three main conditions is visualized in **Figure 4B**. This response was also significantly weaker during riding than during driving ( $\mu = -1.32 \text{ uV}$  vs.  $-.61 \text{ uV}$ ,  $F(1,40) = 22.48$ ,  $p < .001$ ). Consistent with these effects, the N1 during driving did not differ from baseline ( $F(1,39) = 1.39$ ,  $p = .25$ ). In addition, we found that the MMN (i.e., the change in N1 in response to unexpected tones) was greater during riding ( $\mu = -.50 \text{ uV}$  vs.  $-.09 \text{ uV}$ ,  $t(40) = 2.3$ ,  $p = .027$ ).



**Figure 4: Electroencephalogram ERP Results.** **A.** Event-related potentials in response to standard (dashed) and oddball (solid) tones in driving, riding, and immobile (nostate) conditions. **B.** The whole-brain response 100 ms after the onset of the tone in each of the three main conditions, showing that the brain is significantly less responsive to tones while riding.

A spectral analysis of frequency content during periods of silence revealed changes by condition. Specifically, the spectral power percentage in the alpha frequency band (8 - 12Hz) during riding was significantly less than either baseline ( $\mu = 0.32\%$ ,  $t(39)=4.0$ ,  $p<10^{-3}$ , **Figure 5**) or driving ( $\mu = 0.51\%$ ,  $t(39)=5.7$ ,  $p<10^{-5}$ )



**Figure 5: Electroencephalogram Spectral Results.** All data are paired. Each red line indicates the median, blue marks the interquartile range, and black denotes the most extreme non-outlier. Alpha band power, as a percentage of total spectral power, is greater in riding than either rest or driving.

## Discussion

In the sensory processing of auditory stimuli, the N1 has well-characterized involvement and known modulability with varying degrees of attention. In our experiment, N1 amplitude in response to both standard and oddball tones was lower in the riding condition than driving and resting conditions, suggesting that riding reduced susceptibility to distraction. Relatedly, we found that the MMN was also greater during riding than baseline, consistent with the prediction that riding heightens sensory processing. This interpretation of sensory-focus enhancement aligns with research showing an increased MMN response in meditators (Biedermann et al., 2016; Luo, Wei, & Weekes, 1999; Singh & Telles, 2015; Srinivasan & Baijal, 2007), as well as deviant MMN amplitude and/or latency in cases of cognitive dysfunction (Ford & Mathalon, 2012; Huttunen-Scott, Kaartinen, Tolvanen, & Lyytinen, 2008; Risto Näätänen, Sussman, Salisbury, & Shafer, 2014; Shelley et al., 1991; Umbricht & Krijes, 2005). Together, these results suggest that riding increases attention via two mechanisms: strengthening focus and heightening the brain's passive monitoring of changes in the sensory environment.

Furthermore, the decrease in whole-brain alpha band power during periods of silence, while riding relative to baseline and driving, is similar to the effects of caffeine. The magnitude and widespread nature of this change in brain activity while riding suggests an increase in alertness similar to a cup of coffee (Angelakis, Lubar, Stathopoulou, & Kounios, 2004; Barry et al., 2005; Dimpfel, Schober, & Spüler, 1993; Reeves, Struve, Patrick, & Bullen, 1995).



The hormonal data support a comparison of motorcycling with light exercise. Specifically, the observed increase in catecholamines and heart rate suggest that riding, but not driving, increases arousal of the sympathetic nervous system. The magnitude of the increase in these quantities, relative to rest and despite reduced catecholamine production while sitting (Christensen & Brandsborg, 1973; Von Euler & Hellner, 1952), is commensurate with light exercise (Hill et al., 2008; Zouhal, Jacob, Delamarche, & Gratas-Delamarche, 2008). Additionally, while we have no findings to report about long-term changes in hormonal biomarkers of stress as a function of riding a motorcycle, future longitudinal studies may choose to investigate this potential prophylactic link, as elevated glucocorticoid levels have been shown to contribute to neuronal death (Dinkel, MacPherson, & Sapolsky, 2003; Kerr, Campbell, Applegate, Brodish, & Landfield, 1991; Krystal, 1993; Sorrells, Munhoz, Manley, Yen, & Sapolsky, 2014; Uno, Tarara, Else, Suleman, & Sapolsky, 1989).

## References

- Alain, C., & Arnott, S. R. (2000). Selectively attending to auditory objects. *Frontiers in Bioscience: A Journal and Virtual Library*, 5, D202–D212. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/10702369>
- Angelakis, E., Lubar, J. F., Stathopoulou, S., & Kounios, J. (2004). Peak alpha frequency: an electroencephalographic measure of cognitive preparedness. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, 115(4), 887–897. <https://doi.org/10.1016/j.clinph.2003.11.034>
- Barry, R. J., Rushby, J. A., Wallace, M. J., Clarke, A. R., Johnstone, S. J., & Zlojutro, I. (2005). Caffeine effects on resting-state arousal. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, 116(11), 2693–2700. <https://doi.org/10.1016/j.clinph.2005.08.008>
- Biedermann, B., de Lissa, P., Mahajan, Y., Polito, V., Badcock, N., Connors, M. H., ... McArthur, G. (2016). Meditation and auditory attention: An ERP study of meditators and non-meditators. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, 109, 63–70.

<https://doi.org/10.1016/j.ijpsycho.2016.09.016>

Buford, T. W., & Willoughby, D. S. (2008). Impact of DHEA (S) and cortisol on immune function in aging: a brief review. *Applied Physiology, Nutrition, and Metabolism = Physiologie Appliquee, Nutrition et Metabolisme*, 33(3), 429–433. Retrieved from <http://www.nrcresearchpress.com/doi/abs/10.1139/h08-013>

Chang, C.-Y., Hsu, S.-H., Pion-Tonachini, L., & Jung, T.-P. (2018). Evaluation of Artifact Subspace Reconstruction for Automatic EEG Artifact Removal. *Conference Proceedings: ... Annual International Conference of the IEEE Engineering in Medicine and Biology Society. IEEE Engineering in Medicine and Biology Society. Conference, 2018*, 1242–1245. <https://doi.org/10.1109/EMBC.2018.8512547>

Christensen, N. J., & Brandsborg, O. (1973). The Relationship between Plasma Catecholamine Concentration and Pulse Rate during Exercise and Standing. *European Journal of Clinical Investigation*, 3(4), 299–306. <https://doi.org/10.1111/j.1365-2362.1973.tb00355.x>

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, 134(1), 9–21. <https://doi.org/10.1016/j.jneumeth.2003.10.009>

Dimpfel, W., Schober, F., & Spüler, M. (1993). The influence of caffeine on human EEG under resting condition and during mental loads. *The Clinical Investigator*, 71(3), 197–207. <https://doi.org/10.1007/BF00180102>

Dinkel, K., MacPherson, A., & Sapolsky, R. M. (2003). Novel glucocorticoid effects on acute inflammation in the CNS. *Journal of Neurochemistry*, 84(4), 705–716. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/12562515>

Ford, J. M., & Mathalon, D. H. (2012). Anticipating the future: automatic prediction failures in schizophrenia. *International Journal of Psychophysiology: Official Journal of the*

*International Organization of Psychophysiology*, 83(2), 232–239.

<https://doi.org/10.1016/j.ijpsycho.2011.09.004>

Garrido, M. I., Kilner, J. M., Stephan, K. E., & Friston, K. J. (2009). The mismatch negativity: a review of underlying mechanisms. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, 120(3), 453–463.

<https://doi.org/10.1016/j.clinph.2008.11.029>

Hill, E. E., Zack, E., Battaglini, C., Viru, M., Viru, A., & Hackney, A. C. (2008). Exercise and circulating cortisol levels: the intensity threshold effect. *Journal of Endocrinological Investigation*, 31(7), 587–591. <https://doi.org/10.1007/BF03345606>

Huttunen-Scott, T., Kaartinen, J., Tolvanen, A., & Lyytinen, H. (2008). Mismatch negativity (MMN) elicited by duration deviations in children with reading disorder, attention deficit or both. *International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology*, 69(1), 69–77.

<https://doi.org/10.1016/j.ijpsycho.2008.03.002>

Kerr, D. S., Campbell, L. W., Applegate, M. D., Brodish, A., & Landfield, P. W. (1991). Chronic stress-induced acceleration of electrophysiologic and morphometric biomarkers of hippocampal aging. *The Journal of Neuroscience: The Official Journal of the Society for Neuroscience*, 11(5), 1316–1324. Retrieved from

<https://www.ncbi.nlm.nih.gov/pubmed/2027050>

Krystal, J. H. (1993). Stress, the Aging Brain, and the Mechanisms of Neuron Death. *The Yale Journal of Biology and Medicine*, 66(2), 109. Retrieved from

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2588844/>

Lee, T. W., Girolami, M., & Sejnowski, T. J. (1999). Independent component analysis using an extended infomax algorithm for mixed subgaussian and supergaussian sources. *Neural*

*Computation*, 11(2), 417–441. Retrieved from

<https://www.ncbi.nlm.nih.gov/pubmed/9950738>

Luo, Y., Wei, J., & Weekes, B. (1999). Effects of musical meditation training on auditory mismatch negativity and P300 in normal children. *Chinese Medical Sciences Journal = Chung-Kuo I Hsueh K'o Hsueh Tsa Chih / Chinese Academy of Medical Sciences*, 14(2), 75–79. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/12901613>

Makeig, S., Jung, T.-P., Bell, A. J., Ghahremani, D., & Sejnowski, T. J. (1996). Blind separation of event-related brain response components. *PsycEXTRA Dataset*.

<https://doi.org/10.1037/e526132012-219>

Morgan, C. A., Southwick, S., Hazlett, G., Rasmusson, A., Hoyt, G., Zimolo, Z., & Charney, D. (2004). Relationships Among Plasma Dehydroepiandrosterone Sulfate and Cortisol Levels, Symptoms of Dissociation, and Objective Performance in Humans Exposed to Acute Stress. *Archives of General Psychiatry*, 61(8), 819–825. Retrieved from

<https://jamanetwork.com/journals/jamapsychiatry/fullarticle/482042>

Näätänen, R., Kujala, T., & Winkler, I. (2011). Auditory processing that leads to conscious perception: a unique window to central auditory processing opened by the mismatch negativity and related responses. *Psychophysiology*, 48(1), 4–22.

<https://doi.org/10.1111/j.1469-8986.2010.01114.x>

Näätänen, R., Paavilainen, P., Rinne, T., & Alho, K. (2007). The mismatch negativity (MMN) in basic research of central auditory processing: a review. *Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology*, 118(12), 2544–2590.

<https://doi.org/10.1016/j.clinph.2007.04.026>

Näätänen, R., Sussman, E. S., Salisbury, D., & Shafer, V. L. (2014). Mismatch negativity (MMN) as an index of cognitive dysfunction. *Brain Topography*, 27(4), 451–466.

<https://doi.org/10.1007/s10548-014-0374-6>

Reeves, R. R., Struve, F. A., Patrick, G., & Bullen, J. A. (1995). Topographic quantitative EEG measures of alpha and theta power changes during caffeine withdrawal: preliminary findings from normal subjects. *Clinical EEG*, *26*(3), 154–162. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/7554302>

Ritsner, M., Maayan, R., Gibel, A., Strous, R. D., Modai, I., & Weizman, A. (2004). Elevation of the cortisol/dehydroepiandrosterone ratio in schizophrenia patients. *European Neuropsychopharmacology: The Journal of the European College of Neuropsychopharmacology*, *14*(4), 267–273. <https://doi.org/10.1016/j.euroneuro.2003.09.003>

Shelley, A. M., Ward, P. B., Catts, S. V., Michie, P. T., Andrews, S., & McConaghy, N. (1991). Mismatch negativity: An index of a preattentive processing deficit in schizophrenia. *Biological Psychiatry*, *30*(10), 1059–1062. [https://doi.org/10.1016/0006-3223\(91\)90126-7](https://doi.org/10.1016/0006-3223(91)90126-7)

Singh, N., & Telles, S. (2015). Neurophysiological Effects of Meditation Based on Evoked and Event Related Potential Recordings. *BioMed Research International*, *2015*, 406261. <https://doi.org/10.1155/2015/406261>

Sorrells, S. F., Munhoz, C. D., Manley, N. C., Yen, S., & Sapolsky, R. M. (2014). Glucocorticoids increase excitotoxic injury and inflammation in the hippocampus of adult male rats. *Neuroendocrinology*, *100*(2-3), 129–140. <https://doi.org/10.1159/000367849>

Srinivasan, N., & Baijal, S. (2007). Concentrative meditation enhances preattentive processing: a mismatch negativity study. *Neuroreport*, *18*(16), 1709–1712. <https://doi.org/10.1097/WNR.0b013e3282f0d2d8>

Sussman, E. S. (2007). A new view on the MMN and attention debate: the role of context in processing auditory events. *Journal of Psychophysiology*, *21*(3-4), 164–175. Retrieved from

<https://econtent.hogrefe.com/doi/abs/10.1027/0269-8803.21.34.164>

Umbricht, D., & Krijes, S. (2005). Mismatch negativity in schizophrenia: a meta-analysis.

*Schizophrenia Research*, 76(1), 1–23. <https://doi.org/10.1016/j.schres.2004.12.002>

Uno, H., Tarara, R., Else, J. G., Suleman, M. A., & Sapolsky, R. M. (1989). Hippocampal

damage associated with prolonged and fatal stress in primates. *The Journal of*

*Neuroscience: The Official Journal of the Society for Neuroscience*, 9(5), 1705–1711.

Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/2723746>

Von Euler, U. S., & Hellner, S. (1952). Excretion of noradrenaline and adrenaline in muscular

work. *Acta Physiologica Scandinavica*, 26(2-3), 183–191.

<https://doi.org/10.1111/j.1748-1716.1952.tb00900.x>

Warnock, F., McElwee, K., Seo, R. J., Mclsaac, S., Seim, D., Ramirez-Aponte, T., ... Young, A.

H. (2010). Measuring cortisol and DHEA in fingernails: a pilot study. *Neuropsychiatric*

*Disease and Treatment*, 6, 1–7. Retrieved from

<https://www.ncbi.nlm.nih.gov/pubmed/20169040>

Zouhal, H., Jacob, C., Delamarche, P., & Gratas-Delamarche, A. (2008). Catecholamines and

the effects of exercise, training and gender. *Sports Medicine*, 38(5), 401–423.

<https://doi.org/10.2165/00007256-200838050-00004>