

St. John's University

St. John's Scholar

Theses and Dissertations

2025

SLEEP'S IMPACT ON MUSIC PROCESSING IN THE DEVELOPING BRAIN

Rigel Leonard Baron

Follow this and additional works at: https://scholar.stjohns.edu/theses_dissertations



Part of the [Music Commons](#)

SLEEP'S IMPACT ON MUSIC PROCESSING IN THE DEVELOPING BRAIN

A thesis submitted in partial fulfillment
of the requirements for the degree of

MASTER OF SCIENCE

to the faculty of the

DEPARTMENT OF COMMUNICATION SCIENCES AND DISORDERS

of

ST. JOHN'S COLLEGE OF LIBERAL ARTS AND SCIENCES

at

ST. JOHN'S UNIVERSITY

New York

by

Rigel Leonard Baron

Date Submitted: April 26, 2025

Date Approved: April 29, 2025

Rigel Leonard Baron

Dr. Yan H. Yu

**© Copyright by Rigel Leonard Baron 2025
All Rights Reserved**

ABSTRACT

SLEEP'S IMPACT ON MUSIC PROCESSING IN THE DEVELOPING BRAIN

Rigel Leonard Baron

Sleep plays a crucial role in human development, particularly in early childhood. While its influence on brain function is well documented, the relationship between sleep and auditory processing in the developing brain remains underexplored. The present study aimed to investigate how sleep duration impacts cortical responses to music, focusing on children aged 5 to 10. Of the 14 children recruited, 11 (6 female, 5 male) were included in the final analysis following EEG quality checks. Participants underwent standardized assessments to confirm normal hearing, language ability, and intelligence. Using a six-feature passive listening oddball paradigm, EEG data was recorded via a 64-channel HydroCel Geodesic Sensor Net while children listened to the musical paradigm and engaged in quiet activities. Event-related potentials (ERPs) were analyzed across six music conditions: intensity, location, pitch, rhythm, slide, and timbre. Participants were divided into two groups based on average sleep duration (more or less than 10 hours per night). ERP data was evaluated using repeated measures ANOVA and cluster-based permutation testing. Results revealed that children who slept more exhibited more positive amplitude responses in all six conditions. Additionally, the study explored the role of mismatch negativity (MMN), a pre-attentive auditory response, and found that sleep-related differences in MMN were not significant, despite trends of MMN observed. Hemispheric analysis indicated a greater right hemisphere dominance in children with

less sleep, while also displaying a greater sleep-related sensitivity to amplitude attenuation in the left hemisphere. These findings contribute new insights into the impact of sleep on auditory processing in children, suggesting that sleep may modulate brain responses within hemispheres, with greater ERP implications for understanding sleep as a confounding variable in cognitive studies and clinical research.

DEDICATION

To Dad, I love you.

ACKNOWLEDGMENTS

A very special thank you to the one person this could not have been completed without, Dr. Yan H. Yu. Your mentorship has been invaluable. Your lab introduced me to a unique perspective of science I never imagined experiencing. Thank you for contributing your time, resources, and thought into my academic and intellectual development.

Completion of this thesis marks the end of the beginning in my journey through science, and I am grateful for your shaping of the path ahead.

TABLE OF CONTENTS

DEDICATION.....	ii
ACKNOWLEDGMENTS	iii
LIST OF TABLES	vi
LIST OF FIGURES	vii
INTRODUCTION	1
METHODS	12
Participants.....	12
Oddball Paradigm	13
EEG Acquisition & Experimental Procedure	14
Data Preprocessing.....	15
DATA ANALYSIS AND RESULTS.....	17
Event Related Potentials	17
Step 1: Three-way Repeated Measures ANOVA	18
Step 2: Two-way Repeated Measures ANOVA	19
Step 3: Time Window Permutation Testing.....	19
Mismatch Negativity.....	20
Peak Based Analysis	20
Cluster-Based Permutation Testing	22
Hemispheric Analysis	23
Paired <i>t</i> -Tests	23
Modulation Index.....	24
Source Analysis	25

DISCUSSION.....	26
Event Related Potentials	26
Mismatch Negativity.....	28
Hemispheric Effects.....	30
CONCLUSION.....	33
APPENDICES	35
REFERENCES	44

LIST OF TABLES

Table A1. Participant Sleep Data.....	35
Table A2. 200 ms ERP Permutation Statistics.....	39
Table A3. Hemispheric Activity Comparison	40

LIST OF FIGURES

Figure 1. Auditory Oddball Paradigm	14
Figure 2. Overall Mean MMN Amplitude.....	21
Figure 3. Mean MMN Amplitude by Condition.....	22
Figure 4. Difference Waveforms for Fz Electrode	23
Figure 5. Hemispheric Comparison of Brain Activity.....	25
Figure B1. 64-Channel HydroCel Geodesic Sensor Net	41
Figure B2. Source Localization – Sleep More Group.....	42
Figure B3. Source Localization – Sleep Less Group.....	43

INTRODUCTION

Evolution serves as the foundational mechanism through which all life on Earth has developed, from single-celled prokaryotes that emerged over 3.4 billion years ago, to the eventual rise of humans. Despite the complexity of this evolutionary timeline, it is difficult to argue that the process has been anything but remarkably efficient, culminating in species like *Homo sapiens* – biologically intricate and evolutionarily optimized. This transformation reflects both idiosyncrasies and shared traits with our evolutionary ancestors. Yet, even with extraordinary scientific advances, the purpose of many evolved characteristics remains elusive. One such mystery is the function of sleep.

Sleep is a near-universal biological phenomenon, observed in organisms as simple as fruit flies and jellyfish, all the way to humans (Nath et al., 2017; van Alphen et al., 2021). Its evolutionary persistence, despite rendering individuals inactive and vulnerable for extended periods, suggests it serves vital physiological functions. But how could a feature that places its host in a coma-like state for hours on end – time that could be spent hunting, foraging, or defending oneself – survive the rigors of natural selection? Sleep, for all its waste, consuming nearly 30 years of a human's life, must possess one, if not a plethora of critical functions. This apparently high cost of sleep, laid against an evolutionary backdrop, makes understanding its function particularly compelling.

While sleep affects the entire body, many of its most profound effects are concentrated in the brain. Electroencephalography (EEG), a noninvasive and relatively low-cost methodology, has become a cornerstone of sleep research (Al-Shama et al., 2024; Lambert & Peter-Derex, 2023). EEG measures brain activation by capturing electrical activity generated from neuronal firing via electrodes placed on the scalp.

While this technology is by no means new, dating back to 1924, the past several decades have marked a notable adaptation in the way researchers use EEG to conduct research and analyze datasets. The 1960's marked another era of EEG adaptation with a measurement known as an event-related potential (ERP), offering further insight in the cortical function of the brain at a specific moment in time. ERP research typically involves the presentation of a stimulus or event and subsequent measuring of the brain's electrical response. Unfortunately, given the extreme electrical and mechanistic complexity of the brain, especially when compared to a simple 64-electrode EEG, it is difficult to isolate the signal (ERP, or response to the stimulus) from the noise (the countless other processes the brain is engaged in at any given moment). To solve this, computational statistics will average the brain's response to a specific stimulus over the course of many trials, thus resulting in an ERP. This average, given a large enough sample size, will eliminate a substantial proportion of the noise, leaving the data with a pure ERP waveform, a temporal representation of the changing voltage in the brain.

The same way in which EEG research evolved to incorporate new techniques and measurements, ERP research has cultivated its own nuances. Each wave that is generated in response to a stimulus contains different components. These are typically divided into N and P, representing negative and positive voltages, respectively. The negative and positive waves are further subdivided by their respective time after stimulus presentation. The P1 (also referred to as P50 in auditory EEG) wave is graphically represented by a positive inflection, peaking between 40 and 75 milliseconds (ms) after the stimulus presentation. The N1 (also referred to as N100) is graphically represented by a negative deflection with a trough somewhere between 90 and 200 ms. Additional waves follow the

same format, with P2 and P3 waves among popularly studied components of ERP (Sur & Sinha, 2009). The P1, N1, and P2 waves are often studied together and referred to as the obligatory auditory response (or P1-N1-P2 complex), as these waveforms are consistently and automatically produced by the brain and observed through EEG in response to auditory stimuli (Näätänen & Picton, 1987).

In its totality, analysis of ERPs is one of the most foundational techniques EEG researchers use to study the human brain. Despite numerous variables that constrain the spatial accuracy of EEG and ERP data, these techniques still offer excellent temporal resolution and provide an objective methodology to garner a precise understanding of how the brain responds to any variety of circumstances. Because of this, ERPs have wide-ranging implications not only as a tool to better understand the brain, but also as a valuable diagnostic measure (Landa et al., 2014). Since their discovery, ERPs have attracted the attention of researchers across the world, leading to an abundance of research, yet due to their cross-discipline and near universal nature, it is difficult to imagine a future where ERP research reaches its terminus. One area of particular curiosity concerns the overlap between sleep research and ERP. Existing adult data has shown fairly conclusively that both sleep restriction and total sleep deprivation affect the brain's raw ERP responses to simple auditory stimuli. An early study conducted by Morris et al. (1992) found significant attenuation of the P300 component (P3b) along with an increase in P300 latency following periods of sleep deprivation in adults. Further adult sleep deprivation research supported the earlier finding, confirming a decrease in P300 amplitude coupled with a decrease in amplitude of N200. Interestingly, this same

study noted no change in the N100 component but saw an increase in amplitude of P200 (Lee et al., 2004).

Self-rated psychological scales used in parallel revealed a connection between sleepiness and the decrease in amplitude of P300 and N200, while showing the increase in P200 amplitude reflected characteristics of a negative mood, rather than sleepiness (Lee et al., 2004). Total sleep deprivation has also been shown to attenuate ERP amplitudes in response to visual stimuli (Corsi-Cabrera et al., 1999). These are important findings because they demonstrate that sleep modulates sensory and cognitive brain responses and suggests that researchers and clinicians alike may benefit from factoring sleep into their studies as a confounding variable.

Despite the breadth of research concerning sleep and ERPs in adults, there is a noticeable dearth of research investigating how sleep affects ERPs in children – an avenue certainly worth exploring. Investigating ERPs in children offers unique insights and perspectives that are largely unattainable in adult data. Childhood represents a critical stage of brain development for auditory processing, characterized by clear structural and neurophysiological distinctions from adults. These developmental differences suggest that children may process stimuli differently than adults during EEG studies (Boen et al., 2022; Manzi et al., 2011; Panizza et al., 2021). Exploring these divergences – or any unexpected similarities – can offer new insights into the functional maturation of the brain and enhance our understanding of cognitive development. Unfortunately, studying sleep in children is not nearly as simple as studying sleep in adults. Rightfully so, children should not undergo total sleep deprivation, nor extended sleep restriction in the pursuit of research. However, these conditions are typically the means by which the

independent variable of sleep is manipulated in adults. Due to this, few studies have examined ERP responses in children in relation to sleep. Among the limited literature, existing data suggest that even minor disruptions in sleep can significantly affect neurocognitive functioning, often reflected as reduced amplitude in ERP components – particularly the P300 (Molfese et al., 2013). To build upon these preliminary findings, the current study aims to evaluate whether sleep consistently attenuates ERP responses when tested across a diverse and robust set of auditory stimuli.

In addition to general ERP components, one specific measure that has gained considerable attention in sleep-related research is mismatch negativity (MMN). Given its pre-attentive nature, MMN provides a unique opportunity to assess how sleep may influence automatic auditory processing. MMN typically appears as a negative deflection between 150 and 250 ms, elicited using an oddball paradigm. This type of response is exogenous and pre-attentive, thus it can occur without the prerequisite of attention, and does not require overt behavioral response (Näätänen et al., 2015). Importantly however, MMN is a difference waveform and thus cannot be seen by a lone standard graph of an ERP. Mathematically it is represented with the waveform generated by a deviant stimulus minus its standard waveform analog and is typically visualized on its own separate graph. As MMN measures the difference between the brain's response to a standard versus a deviant stimulus, it is therefore commonly referred to as an index of automatic change-detection, however competing hypotheses attempt to tease apart its neural origins. The model-adjustment hypothesis, for example, suggests MMN is elicited as the result of the brain's top-down formation of a sensory predictive model around the standard stimulus. Consequently, if any following stimulus matches the preceding stimulus, the sound

undergoes repetition suppression, further reinforcing this top-down predictive model. However, if a deviant stimulus is introduced, the predictive model is no longer reinforced, and a more negative waveform is produced. An alternate explanation suggests that MMN's origin is much simpler. The adaptation hypothesis posits neurons of the primary auditory cortex adapt to repeated exposure of standard stimuli, attenuating and delaying the N1 ERP component. Once a deviant stimulus is introduced, it interrupts the neuronal adaptation, triggering the neurons within the primary auditory cortex to "reset", displaying a greater N1 response to the deviant stimulus. This break in the neuronal adaptation results in a deviant waveform that differs from the preceding response to a standard, and again, MMN would be observed (Fitzgerald & Todd, 2020; Garrido et al., 2009; Näätänen et al., 2007).

Due to the nature and mechanisms of auditory MMN, a better understanding developed through research is critical to potentially uncovering insight into how different facets of acoustics impact our brain. The field of MMN research is broad and young, and many subfields are still in their infancy. To date, studies have shown MMN is often a valuable ancillary diagnostic tool, serving as a biomarker and early indicator for numerous cognitive pathologies including schizophrenia, autism spectrum disorder, attention deficit/hyperactivity disorder, and neurodegenerative diseases (Donaldson et al., 2023; Ford et al., 2022; Lindín et al., 2013). MMN has even gone so far as to serve as an indicator of retained cognitive function in comatose patients, providing early notice of potential recovery (Zhou et al., 2021). While MMN has been utilized diagnostically for several decades, many of its important functions are still being uncovered, making comprehensive research into its facets even more necessary.

Among these sparsely studied facets, lies sleep and its role in MMN. The existing literature in this area is ambiguous at best, with some studies concluding that sleep deprivation, in one form or another, results in an attenuation of MMN (Donaldson et al., 2023; Ford et al., 2022; Lindín et al., 2013; Zhou et al., 2021). Meanwhile, other studies offer an antithetical conclusion that sleep deprivation has no significant impact on MMN (Bortolotto et al., 2011; Gosselin et al., 2006; Muller-Gass & Campbell, 2019; Salmi et al., 2005). This inconsistency highlights a critical gap in our understanding of sleep's role in the brain, particularly in children, and underscores the potential of MMN as a tool for assessing the relationship between sleep and brain development. Just as with sleep's influence on raw ERP components, understanding its role in the developing brain holds significant potential. Given that MMN reflects the brain's ability to form and maintain a sensory memory trace, investigating how sleep influences MMN offers important insight into how sleep may affect neural mechanisms of sensory memory, pattern learning, and change detection. Understanding this relationship could help clarify the broader role of sleep in supporting cognitive functions during critical periods of brain development.

Due to MMN's reliance on standard and deviant stimuli, the foremost methodology to elicit an auditory MMN response is through application of an oddball paradigm (Garrido et al., 2009). Oddball paradigms are typically sequences of tones where one stimulus, the standard, is played repeatedly, while a deviant stimulus is interjected into the sequence with the goal of producing tell-tale MMN activity. Existing data is most often derived from pure-tone oddball paradigms, in which the deviant stimulus only alters in pitch. Several sleep studies examine deviations in duration rather than frequency; however, few, if any, use a robust framework of deviant sounds. This is

the advantage of music. Music allows researchers to create oddball paradigms that deviate in a range of variables, not only pitch or duration. Beyond this, music has two additional advantages. One, especially in children, it is much easier to obtain greater quantities of data – most people, let alone young children, are not particularly keen on listening to repetitive tones for long durations, regardless of the task given to distract them. Music solves this, as children are more willing to listen to music for longer periods of time, allowing for the collection of greater volumes of data, without risking agitating the child; a risk that carries the potential for a decrease in EEG signal-to-noise ratio with an accompanying increase in undesirable EEG artifacts (DiStefano et al., 2019). Beyond their numerous advantages, musical paradigms offer the distinct benefit of functioning as a universal medium, facilitating the assessment of children across diverse linguistic backgrounds. Finally, music provides greater ecological validity than pure-tone or single-deviant paradigms, as it better represents the diversity and complexity of sounds experienced in everyday life. More broadly, music – and specifically, the oddball paradigm used in this study – incorporates a range of acoustic modifications while presenting stimuli in a structured musical arrangement, making it more reflective of natural sound processing, which may yield data that more closely represents typical brain activity (Kliuchko et al., 2016).

Music processing is primarily associated with the right hemisphere of the brain, which is more actively engaged when listening to music, though the left hemisphere also plays a role (Evers et al., 1999; Kimura, 1964). Despite existing knowledge, research remains inconclusive regarding the influence of sleep on music processing across hemispheres. In one study, researchers analyzed both music and language processing

using psychoacoustic behavioral tests: participants were asked to discriminate between monoaurally presented melodies for music processing, and to identify monoaurally presented words that were filtered to distort their acoustic features for word processing. In both tasks, participant accuracy served as the primary outcome measure. The study found that sleep deprivation impaired word processing presented to the left ear and music processing presented to the right ear. Due to the brain's contralateral connections between the ears and hemispheres, word processing – primarily a left-hemisphere function – was disrupted on the right side of the brain, while music processing, which is predominantly right-hemispheric, was more impaired on the left. This suggests an inverse relationship between the hemisphere experiencing most activation and the hemisphere that experiences the most attenuation caused by insufficient sleep. Simply put, if a task is left hemisphere dominant, the right hemisphere's activity would be dampened. This study also examined neurophysiological measures, noting a significant increase in P300 and MMN latency in an experimentally sleep deprived group, however it noted no significant amplitude attenuation in either condition. While these are insightful findings, the study only observed the relationship between hemispheric involvement by means of a participant's behavioral measures and did not directly compare neurophysiological data between the left and right hemispheres (Díaz-Leines et al., 2017). Additional studies complicate the previous findings by presenting conflicting data. These studies show that spatial working memory, which is primarily a right-brain function, was more diminished in the right hemisphere than in the left. As a result, brain activity became more symmetrical, as opposed to the asymmetry suggested in the Díaz-Leines data (Peng et al., 2020). This notion that the brain's dominant region experiences greater activity reduction

than its non-dominant region is also supported by behavioral data. A 2004 study found that the right hemisphere showed greater impairment when recalling and identifying emotional facial expressions – a task typically dominated by the right hemisphere in healthy individuals. (Pallesen et al., 2004). Overall, existing data remains inconclusive about how sleep affects activity levels in each hemisphere of the brain. One possible explanation for the conflicting results is a regional task-based hypothesis, which suggests that sleep's impact on hemispheric activity modulation is not governed by a single rule that can be reduced to the hemispheres involved. Instead, its effects vary depending on the specific task being performed and the corresponding brain regions or networks involved. Building on this idea, the current study aims to gather additional regional and hemispheric data while investigating the underlying mechanisms of hemispheric activity through the analysis of detailed neurophysiological music data in children.

Ultimately, despite prior electrophysiological research on sleep's role in the brain, significant gaps persist, particularly in our understanding of sleep's impact on the developing brain. The present study aims to address these gaps by utilizing electroencephalography and a unique musical paradigm to examine three distinct yet interconnected measures of neurophysiological function. First, it investigates sleep's effect on ERPs. While existing research in adults suggests that insufficient sleep generally attenuates ERP amplitudes, data on how this phenomenon affects children remains limited. Second, the study explores the role of sleep in modulating MMN, a measure with inconclusive findings in adults and no existing data in children. Lastly, this study examines how sleep influences neural activity in the left versus right hemispheres. Current findings in this area remain inconsistent, presenting multiple hypotheses

regarding how varying levels of sleep impacts potential differences in hemispheric modulation. Together, this study seeks to provide a novel perspective on sleep's role in the developing brain by integrating rarely studied electrophysiological data in children with a musical paradigm to observe changes in cortical activity. The present findings may provide valuable diagnostic insights for considering sleep as a confounding variable in clinical settings while also contributing to a broader understanding of sleep's impact on brain function.

METHODS

Participants

EEG data were collected from 14 children, aged 5 to 10 years, each of whom listened to a multifeature passive listening oddball paradigm once, yielding a total of 14 datasets. Data from three participants were excluded due to excessive noise in the EEG recordings following post-processing, resulting in a final sample of 11 participants for analysis.

Sleep data was collected for each participant covering the seven nights prior to and including the night before EEG testing. On the day of testing, participants were asked to estimate their nightly sleep duration by reporting estimated bedtimes and waketimes. Parental input was used in all cases to verify bedtime and waketime estimates. These reports were averaged across the seven nights to calculate each child's mean nightly sleep duration. Based on a median value of approximately 10 hours per night, participants were divided into two groups: those averaging more than 10 hours per night (Sleep More) and those averaging less (Sleep Less). These groups were used for all statistical comparisons (Table A1; Appendix A).

Additionally, children completed a qualitative assessment using the Pictorial Sleepiness Scale (PSS; Maldonado et al., 2004), a visual tool designed to assess subjective sleepiness in young children, similar in purpose to the Karolinska and Stanford Sleepiness Scales (Åkerstedt & Gillberg, 1990; Hoddes et al., 1973). Older children (closer to age 10) were generally able to report their sleep habits and PSS ratings independently, while younger participants often required parental assistance. Although

the qualitative sleepiness ratings were not included in the statistical analysis, they present a potential avenue for future exploration.

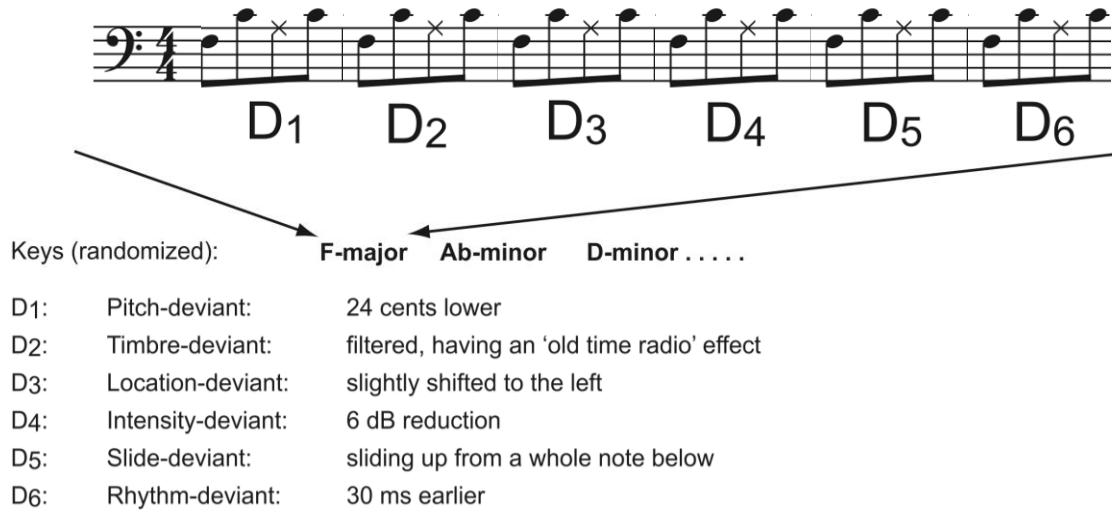
Of the 11 children whose data was included in the final analysis, six were female and five were male. Several additional metrics were gathered on the participating children to control for potential confounds. To ensure children fell within normal ranges for hearing, language, and intelligence, all children were given an intelligence test (TONI-4; Brown et al., 2010), an English language test (PPVT-5; Dunn et al., 2019), and hearing test at the time of their participation. Also, the parents/guardians were asked whether their child spoke additional languages. If the child was able to speak more than one language, they were given language tests to assess their proficiency in the additional languages. Parents/guardians completed a questionnaire regarding the child's musical background. The study was approved by the human subject research institutional review board at St. John's University, New York, and was conducted in compliance with the Declaration of Helsinki. Parental consent was obtained from the parent/legal guardian(s) for each participant.

Oddball Paradigm

The present multifeature mismatch negativity oddball paradigm was adapted as a hybrid of the designs described by Vuust et al. (2011) and Kliuchko et al. (2016), while retaining the acoustic modifications originally employed in the Vuust et al. paradigm. The paradigm itself consists of a piano composition arranged in an Alberti bass sequence, based on a Western arpeggio chord progression, with notes arranged in groups of four, where the third note in every other sequence of four serves as a deviant. To introduce musical variability, the key was changed every sixth measure to one of 12 major or 12

minor keys, allowing each of the six total deviants to be presented once per measure and key. Keys were pseudorandomized such that no key was repeated until all 24 had been cycled through. To maintain consistency, keys were centered around C4, with bass notes ranging between F3 and E4. The presentation of deviants throughout the paradigm was also pseudo-randomized and deviants were altered from their standard counterparts in one of six acoustical modifications: pitch, timbre, location, intensity, slide, or rhythm (Figure 1). Each note, or stimulus, lasts 200 ms with 5 ms allotted for fading in and out at the beginning and end of the note with an interstimulus interval of 5 ms. The experiment paradigm was presented to each participant on a single day for 14 minutes.

Figure 1. Auditory Oddball Paradigm



Present auditory oddball paradigm comprising repeating four-note sequences, with the third note in alternate sets designated as a deviant stimulus (modified from Vuust et al., 2011).

EEG Acquisition & Experimental Procedure

Prior to EEG setup, head circumference was measured to determine appropriate net size. Each participant was then fitted with a 64-channel HydroCel Geodesic Sensor

Net (Electrical Geodesics, Inc., 2007), with Cz serving as the reference electrode. Six frontocentral sites (Appendix C; C3, C4, Cz, F3, F4, and Fz) were selected for MMN analysis (Hao Hsieh et al., 2018; Kliuchko et al., 2016; Shafer et al., 2010) (Figure B1; Appendix B). Electrode impedances were kept below 10 kΩ. EEG was recorded at a 1000 Hz sampling rate with an online bandpass filter of 0.1-100 Hz. Participants were then seated in a sound-attenuated, electromagnetically shielded booth, where electrode connectivity across all 64 channels was verified. Once setup was complete, the booth doors were closed, and participants began the auditory oddball paradigm. Each participant was exposed to 144 deviant trials for each of the six acoustic conditions – pitch, timbre, location, intensity, slide, and rhythm – resulting in a total of 864 deviant trials per participant. For data analysis, 432 standard trials were associated with each deviant category, based on a 3:1 standard-to-deviant ratio, yielding a total of 2,592 standard trials. During EEG recording, children were allowed to engage in quiet activities of their choice (e.g., drawing, tablet-based games) to remain calm and reduce movement artifacts. Seating arrangements were individualized: some children sat alone, others with a parent or researcher. Researchers remained in the booth for participants who required support due to restlessness or attentional difficulties, in order to minimize EEG noise.

Data Preprocessing

Initial preprocessing and segmentation were performed using NetStation software (Electrical Geodesics, Inc.). For offline processing, EEG data were band-pass filtered between 0.1 and 30 Hz using a finite impulse response (FIR) filter. The FIR filter has a linear phase response, ensuring accurate preservation of signal timing and waveform shape. The high pass and low pass filter were justified given the limited distortion to

individual data and the concentration of MMN energy in the 2 to 5 Hz frequency bands (Picton et al., 2000; Rousselet, 2012). Following this, data were exported to BESA (Brain Electrical Source Analyses) Research 7.1 (BESA GmbH, Germany) for artifact correction and averaging. Ocular artifacts (e.g., blinks) were automatically removed using BESA's standard averaging algorithm, followed by manual inspection to remove excessively noisy channels. During offline processing in BESA, zero-phase filters were applied: a high-pass filter at 0.3 Hz (12 dB/octave slope) and a low-pass filter at 20 Hz (24 dB/octave slope). EEG data were segmented into -200 ms to 1500 ms epochs and time-locked to the onset of the first stimulus in each four-note sequence. Noisy channels were interpolated with BESA spline interpolation using data from neighboring electrodes, with total interpolations kept under 15% to ensure data quality.

DATA ANALYSIS AND RESULTS

Event Related Potentials

Prior to the statistical tests, data was segmented. Given the high sampling rate of 1000 Hz, the data was time averaged into 20 ms bins to reduce data volume and number of comparisons. Since the musical paradigm was arranged in an Alberti bass pattern where four notes were played per trial, the data was then partitioned into four note epochs to fit this sequence. To do this the data was further truncated to include a range of 0 ms to 980 ms as 0 ms marked the onset of the first stimulus, 205 ms the second stimulus, 410 the third stimulus, and 615 the fourth stimulus so the range of 0 ms to 980 ms included a full view of the ERP responses to each stimulus without including extraneous pre-stimulus data. The data was further binned into categories to include amplitude (as measured in microvolts) for each participant, in each music condition (i.e.: intensity, rhythm, pitch, slide, timbre, and location), and at each of the six scalp sites used for data analysis (i.e.: C3, C4, Cz, F3, F4, Fz).

Following data rearrangement, repeated measures Analysis of Variance (ANOVA) was used to identify pattern deviance across the main effects of sleep group (Sleep More versus Sleep Less), electrode site, and music condition, as well as the interactions between sleep and site, sleep and condition, site and condition, and the three-way interaction among sleep, site, and condition, on ERP amplitude responses. This was done to determine whether sleep, electrode selection, or music condition independently or interactively influenced ERP amplitudes. Follow-up repeated measures two-way ANOVAs were conducted separately for each music condition for the purpose of evaluating the effects of sleep group, electrode site, and their interaction for each music

condition. This allowed for more targeted analysis of how sleep-related differences might vary depending on the type of musical deviance. To complement these analyses, cluster-based permutation testing was performed to assess sleep group differences in ERP amplitude across multiple time windows (0-200 ms, 200-400 ms, 400-600 ms, and 600-800 ms). This approach was used to control for multiple comparisons across time and space and to identify temporally and spatially connected clusters where sleep group differences were statistically robust.

Step 1: Three-way Repeated Measures ANOVA The first step in the data analysis procedure involved examining whether sleep quantity influenced EEG amplitudes across scalp sites and music conditions using a repeated measures factorial three-way ANOVA. In this test, sleep group was treated as a between-subjects factor, based on each participant's average nightly sleep duration during the week preceding testing. Participants who averaged more than 10 hours of sleep per night were placed into the "Sleep More" group, while those who averaged less than 10 hours per night were placed into the "Sleep Less" group. Electrode site (C3, C4, Cz, F3, F4, Fz) and music condition (pitch, timbre, location, intensity, slide, and rhythm) were treated as within-subjects factors, as each participant contributed data across all sites and conditions. The analysis was conducted with trial-level EEG amplitude data, averaged across all time points using RStudio utilizing the RVAideMemoire package. Results revealed a significant main effect of sleep group ($p < 0.001$), as well as significant interactions between sleep and site ($p < 0.001$), sleep and condition ($p < 0.001$), and a three-way interaction among sleep, site, and condition ($p < 0.001$). These findings suggest that the influence of sleep on EEG amplitudes varies depending on both the electrode location and the type of musical

deviance in a given epoch. Follow-up analyses were performed to further explore the nature of these interactions.

Step 2: Two-way Repeated Measures ANOVA The second step narrowed the comparisons down to a two-way ANOVA, again comparing the difference that may exist between the two sleep groups (participants who averaged more than 10 hours of sleep per night were placed into the "Sleep More" group, while those who averaged less than 10 hours per night were placed into the "Sleep Less" group), however this time comparing average amplitudes across time for sleep and site within each music condition. Due to the increased number of ANOVAs performed (one for each music condition) a False Discovery Rate (FDR) correction was applied to the p values to reduce the Family-Wise Error Rate (FWER) – the probability of making one or more Type I errors (false positives) – induced by multiple comparisons. FDR was selected over similar correction methodologies such as a Bonferroni correction due to the low number of comparisons and overly conservative nature of Bonferroni – often increasing Type II errors (false negatives). Following this, p values for sleep x site interactions in all music conditions showed significance ($p < 0.001$), while sleep's individual impact on all six music conditions also showed significance (timbre: $p < 0.0241$, intensity: $p < 0.001$, location: $p < 0.001$, rhythm: $p < 0.001$, slide: $p < 0.001$, pitch: $p < 0.0174$). The significance detected within this step provided a statistical justification for a third step within the model.

Step 3: Time Window Permutation Testing To analyze the effect sleep may play on ERPs in different time periods, data was segmented into 200 ms time bins, corresponding roughly to the onset of each stimulus (0-200 ms, 200-400 ms, 400-600 ms, and 600-800 ms). Subsequently, cluster-based permutation statistics were performed between sleep

groups (Sleep More versus Sleep Less) for all data sites and music conditions on all 200 ms time bins (250 permutations). Cluster permutation testing was employed in this context to control the FWER and address the issue of multiple comparisons. This approach offers several advantages over traditional correction methods such as FDR and Bonferroni, enhancing statistical power while accounting for the spatial and temporal dependencies inherent in the data (Maris & Oostenveld, 2007). This analysis revealed statistically significant clusters in all six music conditions. Intensity and location displayed significant clusters in the earlier time windows (0-400 ms) across a variety of sites but showed no consistently significant effect at a single set of sites across time windows. Pitch showed significant clusters in the earliest time bin (0-200 ms) at C3, Cz, and Fz. Rhythm showed significance across a range of time bins and sites. Slide and timbre showed the strongest significance with larger clusters across multiple sites and time bins, specifically within the 0-400 ms window (Table A2; Appendix A).

Mismatch Negativity

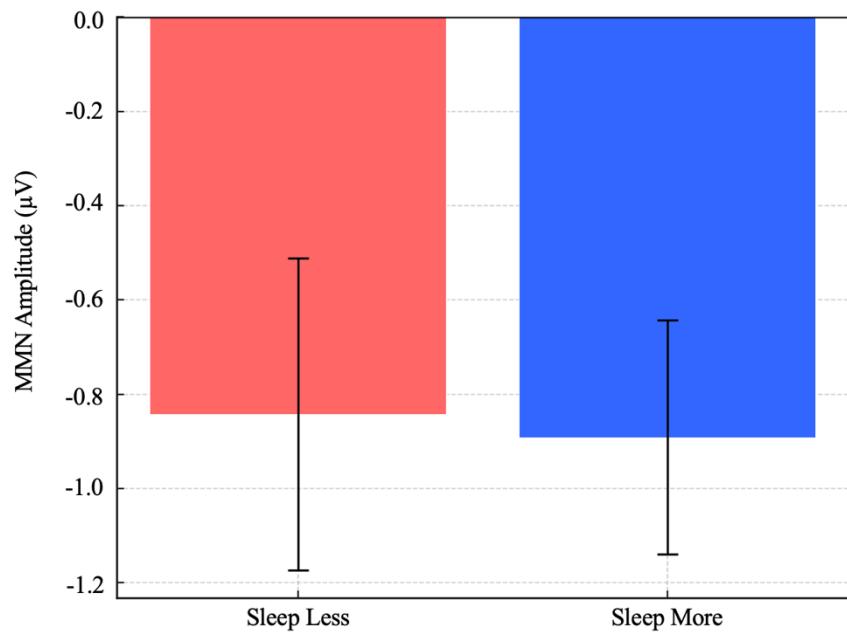
Peak Based Analysis A multi-tiered statistical framework was utilized to analyze MMN between sleep groups to determine if overall significance existed between MMN in the Sleep Less and Sleep More groups, with follow-on cluster-based permutation t-tests to determine how specifically MMN was represented at its canonical site, Fz. MMN was defined as the difference between the third stimulus (deviant) and second stimulus (standard) and calculated using the formula:

$$MMN = Amplitude_{deviant} (400-600\ ms) - Amplitude_{standard} (200-400\ ms)$$

Peak analysis was performed mirroring the methodology from Vuust et al. (2011), using the mean MMN amplitude within a 40 ms window centered on the grand-average MMN

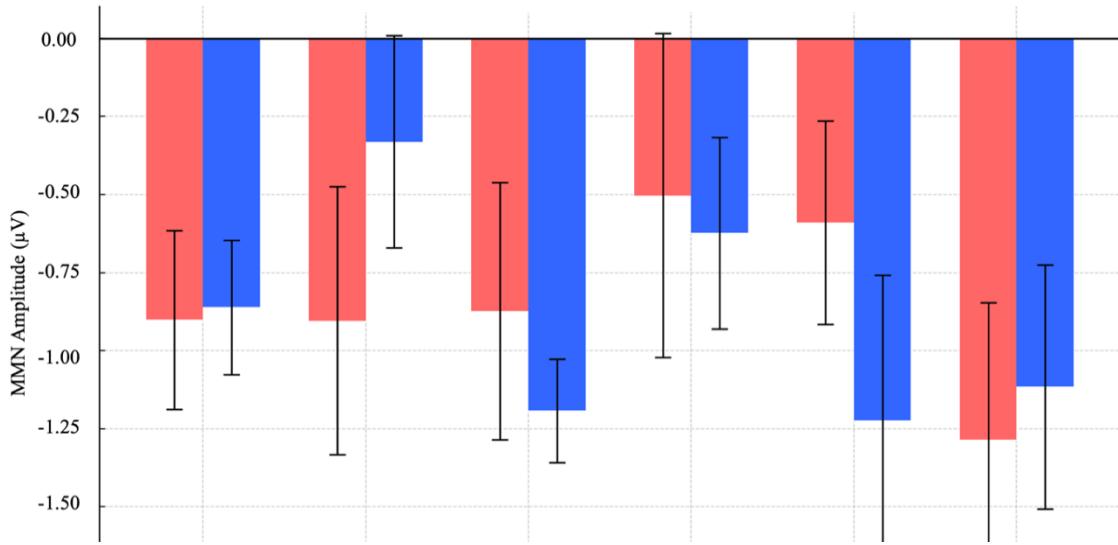
peak latency. No significant group differences were observed across the overall average ($p = 0.91$, Figure 2) or within individual music conditions (all $p > 0.25$, Figure 3).

Figure 2. Overall Mean MMN Amplitude



MMN amplitude values averaged across all sites and all conditions for both sleep groups. MMN calculated by averaging the 40 ms window surrounding the peak MMN value Error bars represent the variability within each group.

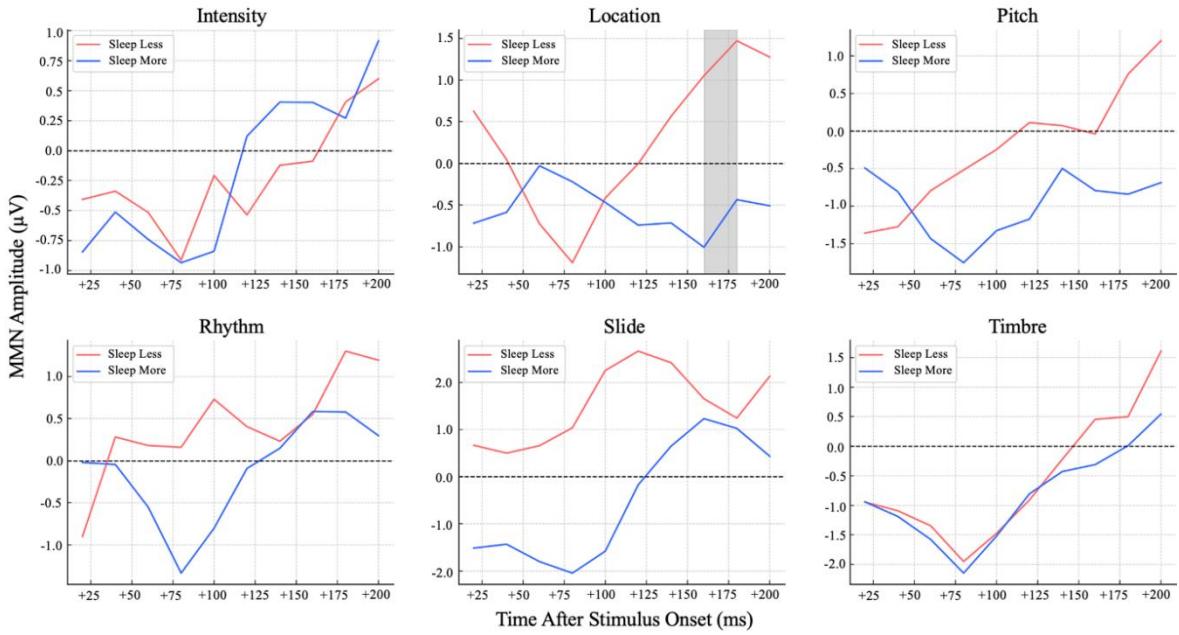
Figure 3. Mean MMN Amplitude by Condition



MMN amplitude values for each music condition, calculated by averaging the 40 ms window surrounding the peak MMN value. The analysis includes data from all sites, averaged across the six music conditions, and compared between the two sleep groups. Error bars represent the variability within each group.

Cluster-Based Permutation Testing To complement the peak-based analysis, additional cluster-based permutation t-tests were used to determine whether there were any specific windows of time where sleep shows significance within the greater 200 ms bin of MMN. This analysis was performed only at the Fz site for all six music conditions. A cluster-forming threshold of $p < 0.05$ was used, and 250 permutations were run to estimate null distributions. This analysis remained mostly consistent with peak analysis, showing only one window of significance between groups in the location condition 160-180 ms post stimulus onset with a statistically stronger (more negative) amplitude revealed in the Sleep More group. Additionally, visually in slide, rhythm, pitch, and timbre the Sleep More group had stronger amplitudes, however this distinction did not survive the significance threshold posed by the cluster-based permutation testing (Figure 4).

Figure 4. Difference Waveforms for Fz Electrode



Difference waveforms obtained by subtracting the standard stimulus from the deviant stimulus at electrode Fz for each music condition, comparing both sleep groups. The grey area in the location graph highlights the region of significance identified during the permutation statistical analysis.

Hemispheric Analysis

Paired t-Tests Finally, to assess hemispheric differences and the potential influence of sleep on these differences, analyses of raw EEG amplitude responses averaged across time and averaged across all music conditions were restricted to lateral electrodes only, excluding midline sites (Cz and Fz) from the original set of six (C3, C4, Cz, F3, F4, Fz). The resulting lateral electrodes were used for hemispheric analysis, with C3 and F3 representing the left hemisphere, and C4 and F4 representing the right. These homologous electrode pairs (C3, F3; C4, F4) were then compared using paired t-tests. Analyses were separated based on the participant's sleep group to determine how sleep quantity may impact hemispheric asymmetries. The resulting analysis showed a statistically significant right hemisphere dominance at frontal sites ($t = 5.04, p = 0.007$) for the sleep less group. Although additional comparisons did not reach statistical

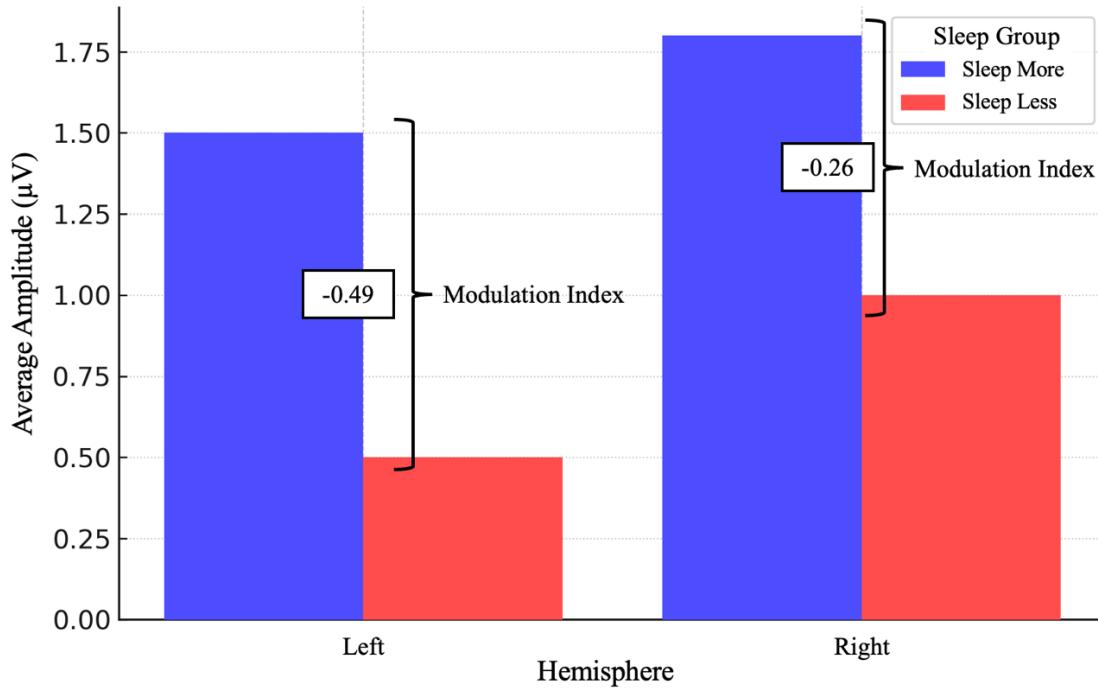
significance, the t-statistics suggest a trend toward right-hemisphere dominance at frontal sites and left-hemisphere dominance at central sites (Table A3; Appendix A).

Modulation Index To quantify the effect of sleep within each hemisphere, a normalized metric – the Modulation Index (MI) – was computed using raw EEG amplitude responses averaged across time and averaged across all music conditions at the lateral electrode pairs (C3, F3; C4, F4). The MI reflects the relative amplitude difference between the Sleep Less and Sleep More groups, allowing comparison of how strongly sleep modulates activity in the left versus right hemisphere. MI is calculated through the formula:

$$MI = \frac{Sleep\ Less - Sleep\ More}{Sleep\ Less + Sleep\ More}$$

Here, Sleep Less refers to the average amplitude across all times and music conditions for all participants within that group, while Sleep More represents the corresponding average for its group. The grand average MI across both hemispheres was -0.37, indicating that participants in the Sleep More group exhibited higher amplitudes across all conditions and lateral electrode sites –consistent with findings from the repeated measures ANOVA. Hemisphere-specific analyses revealed an MI of -0.49 in the left hemisphere and -0.26 in the right, suggesting that sleep may exert a stronger modulatory influence on neural activity in the left hemisphere (Figure 5). A permutation test (10,000 iterations) was performed on the indices, revealing a significant difference between the MI of the left and right hemispheres ($p < 0.001$).

Figure 5. Hemispheric Comparison of Brain Activity



This figure represents the average amplitude recorded across all scalp sites and music conditions. This figure compares mean amplitudes between both sleep groups in the left and right hemispheres. The indicated regions display what the calculation of modulation index represents: the relative differences between sleep conditions in the left and right hemispheres.

Source Analysis To explore hemispheric trends in brain activity, source localization was performed using BESA's integrated source analysis software. The analysis was intended to visualize general patterns of hemispheric activation, rather than to provide statistical evidence for lateralization related to music processing or sleep. Two grand average files were created – one for the Sleep Less group and one for the Sleep More group – incorporating data from all music conditions across all 64 scalp sites. To streamline processing, the time window was limited to 0-800 ms, which captured the most prominent neural responses. The resulting data was analyzed using Classical LORETA (Low Resolution Electromagnetic Tomography) Analysis Recursively Applied (CLARA) (Figures B2 & B3; Appendix B).

DISCUSSION

The present study aimed to elucidate the role of sleep in the developing brain by examining electrophysiological responses to a music-based paradigm in children. Specifically, it investigated (1) sleep's general effect on ERP amplitude, (2) its influence on MMN responses, and (3) the hemispheric implications of sleep quantity on brain function. A statistical approach was employed to address each of these components, offering both a framework for interpreting the current findings and a roadmap for future inquiry. While the study adds valuable child-specific data to an area of research largely centered on adults, the results must be interpreted with caution due to limitations such as small sample size and the observational nature of the study's approach to sleep. To fully unpack the significance and implications of the findings, each core domain is discussed individually, followed by a synthesis of how these elements converge to inform our broader understanding of sleep and auditory processing in the developing brain.

Event Related Potentials

The present findings align with prior research demonstrating that sleep loss attenuates ERP responses. This effect has been well-documented in adults and, to a very limited extent, in children (Molfese et al., 2013; Morris et al., 1992). The current study extends these findings by confirming that sleep significantly impacts ERP amplitudes in children, supporting the notion that sleep modulates auditory processing. Importantly, this attenuation was observed across all six musical deviant conditions, indicating that the effect of sleep is robust and not limited to a specific type of auditory manipulation. The relatively weaker sleep-related effects observed for location deviants may be partially explained by the method of stimulus delivery. In the present study, location changes were

presented through external speakers rather than isolated earphones. This setup may naturally blur the differences heard between the ears due to overlapping acoustic information. This may have reduced the salience of spatial differences, making neural responses to location deviants weaker overall and less sensitive to sleep-related modulation. The relatively weaker sleep-related difference in pitch and intensity deviants may be explained by the basic sensory processing mechanisms engaged during audition of these deviants, features that may be comparatively resilient to moderate sleep disruption. Pitch discrimination, for example, largely engages the early auditory cortex with limited reliance on higher-order integration. Intensity detection, being a low-level sensory function is also typically robust against attentional fluctuations or cognitive fatigue (Näätänen & Picton, 1987). In contrast, auditory features requiring more complex temporal or spectral integration - such as timbre and slide - may be more cortically taxing, thus exhibiting stronger susceptibility to the effects of reduced sleep (Tervaniemi et al., 1994). While the primary focus of the raw ERP analysis was not on individual deviant types, the broad impact across conditions suggests that reduced sleep generally dampens cortical responsiveness to auditory inputs. Permutation-based statistical analysis further validated these effects, revealing significant clusters predominantly in early time windows – prior to the deviant note – regardless of condition. This pattern is expected as notes played prior to the deviant would be unaffected by the upcoming deviant condition. The relative absence of significance in the 400-600 ms window, when the deviant notes typically occurred, supports the MMN results and suggests that sleep-related differences may manifest as more salient in general auditory processing. Given that the deviant musical feature occurs at approximately 400 ms, significant clusters detected in the 0-200

ms and 200-400 ms windows likely represent baseline auditory processing differences rather than direct effects of the deviant feature. Thus, despite the logical explanation for why acoustical modifications such as timbre and slide may be more affected by sleep group, early significant effects should be interpreted cautiously, as they more likely reflect sleep-related modulation of general auditory responsiveness rather than acoustic feature deviance detection. Although specific ERP components were not isolated in this waveform analysis, the observed patterns reinforce trends seen in adult data and contribute novel insight to the limited literature on sleep and auditory ERPs in children. Notably, the large number of trials per condition strengthens the reliability of these findings within subjects, however the study's small sample size may have led to unwanted between subject variance and leaves room for future research to shore up such gaps.

Mismatch Negativity

Existing literature presents conflicting findings regarding the influence of sleep manipulation on MMN, with some studies supporting a measurable effect (Donaldson et al., 2023; Ford et al., 2022; Lindín et al., 2013; Zhou et al., 2021) and others finding none (Bortoletto et al., 2011; Gosselin et al., 2006; Muller-Gass & Campbell, 2019; Salmi et al., 2005). Against this backdrop, the present study examined whether sleep quantity affects MMN responses to various deviant musical conditions in children. The use of a musical paradigm and a younger population represents a novel contribution to the MMN literature. However, statistical analysis did not reveal a significant relationship between sleep quantity and MMN amplitude across conditions. While permutation testing identified a single significant cluster – a 20 ms region within one of six musical

conditions – this isolated result does not provide strong evidence to reject the null hypothesis. Thus, the data do not offer conclusive support for the hypothesis that sleep quantity significant impacts MMN responses in children.

Despite the lack of statistical significance, visual inspection of waveforms suggests consistent differences in MMN responses between children who slept more and those who slept less in nearly all conditions. These apparent trends raise the possibility that real effects were present but obscured by methodological constraints. Notably, the study did not include an experimental manipulation of sleep, relying instead on naturalistic group assignment. Additionally, the sample size may have been insufficient to detect subtle group differences, especially given the known variability in EEG responses among children (Callaway & Halliday, 1973). These limitations highlight the potential for underpowered results and underscore the importance of replication using larger samples and controlled sleep conditions. Such approaches could reduce individual variability and enhance sensitivity to detect true group effects.

Taken together, these findings suggest caution in drawing definitive conclusions about the relationship between sleep quantity and MMN responses in children. Nevertheless, the resulting conclusion is that MMN is relatively unaffected by sleep deprivation. This aligns with MMN's role as a pre-attentive auditory mechanism – one that operates automatically and independently of higher-order cognitive processes such as attention and perception, which are themselves more susceptible to the effects of sleep loss (Chua et al., 2017; Liberalesso et al., 2012). If confirmed, this would imply that MMN may be a stable neurophysiological marker for children, even in the context of mild sleep deprivation. Despite this, until a concrete conclusion is reached regarding

sleep's role on MMN, it may behoove researchers and clinicians to consider lack of sleep as a potential confounding variable in MMN research, particularly in pediatric populations.

Hemispheric Effects

The hemispheric results of music processing in the present study, support existing findings suggesting a general, though subtle, lateralization toward the right hemisphere during music processing (Evers et al., 1999; Kimura, 1964). Interestingly, when examining average EEG amplitudes across time and music conditions between homologous electrode pairs (left hemisphere: C3, F3; right hemisphere: C4, F4) within each sleep group, a statistically significant right-hemispheric dominance emerged at frontal sites in the Sleep Less group – indicating higher EEG amplitudes in this region. No other sleep-related hemispheric asymmetries were statistically significant. At first glance, this may appear to contradict the broader ERP findings showing attenuated amplitudes in participants with less sleep. However, follow-up analysis using a Modulation Index (MI) provided clarity: MI results revealed a greater modulatory effect of sleep in the left hemisphere, suggesting that the left hemisphere is more sensitive to sleep-related attenuation. This discrepancy suggests that the right hemisphere's increased activity in the sleep less group may reflect a relative preservation of function, rather than an increase in resource allocation. In contrast, the left hemisphere showed a greater amplitude reduction, which may be driving the overall decrease in ERP amplitude observed in the sleep less group. This hemispheric asymmetry may be explained by the brain's reliance on the right hemisphere during music processing, potentially making it more resilient to the effects of sleep loss. In this task-specific context, the right

hemisphere may be preserved to support key auditory processes, while the left hemisphere – less central to this task – may experience the greater impact of less sleep. This interpretation is consistent with the stable MMN responses observed in the sleep less group, suggesting that sleep loss does not necessarily impair essential auditory discrimination functions. Based on this modulatory hypothesis and MMN’s relative resilience to sleep related deficits, future research could investigate whether functions or regions adjacent to MMN in space or time are impaired by sleep loss – potentially revealing what, if any, neural processes are “sacrificed” to preserve MMN’s integrity. Overall, in relation to the hemispheric effects of sleep on music processing in the developing brain, the observed asymmetry may reflect a reallocation of neurophysiological resources to support task-relevant regions, or an intrinsic vulnerability of the left hemisphere to sleep loss in this context.

Understanding how these findings fit within the broader literature is critical. The results of this hemispheric analysis generally align with prior research, and where they do diverge, they highlight variability in how sleep affects neural processing across different tasks and populations. The broader ERP waveforms analyzed here suggest a consistent pattern in the context of music processing, a greater contralateral attenuation under conditions of reduced sleep. That is, when the brain is processing music – a task with right hemispheric bias – the less involved hemisphere, in this case the left hemisphere, experiences attenuation (Díaz-Leines et al., 2017). This raises important questions as to why other cognitive tasks might show the opposite effect, an ipsilateral attenuation under similar sleep conditions (Peng et al., 2020). One possibility is that sleep interacts differently with specific cortical regions depending on the functional demands of the task.

Future research should move beyond broad hemispheric comparisons to examine regional dynamics in greater detail. Just as whole-scalp ERP averages can obscure critical differences, whole-hemisphere analyses may fail to capture the fine-grained effects of sleep on neural function. A region-specific approach may be key to advancing our understanding of how sleep loss alters the brain's ability to process complex stimuli such as music.

CONCLUSION

Overall, the present study contributes to the growing body of literature emphasizing the importance of sleep in auditory processing, extending existing adult research into younger populations (Colrain & Campbell, 2007). While the long-term developmental consequences of reduced sleep remain uncertain, these findings reinforce the critical role of sleep in supporting healthy neurophysiological function. The observed broad attenuation of ERP amplitudes across scalp sites and musical conditions in children who slept less suggests a general reduction in neural responsiveness. Although this attenuation is not inherently pathological, prior research has associated similar patterns of reduced ERP amplitude with various clinical conditions, including substance use, ADHD, antisocial behavior, and depression (Hosch et al., 2023; Patrick et al., 2006; Peisch et al., 2021; Whalen et al., 2020). While the current findings do not imply a causal link between sleep patterns and psychopathology, they highlight shared neurophysiological features that warrant further investigation.

These findings should be interpreted within the context of several limitations. Although the musical paradigm enabled many trials per participant, the overall participant sample size remains a limiting factor for generalizability. Moreover, the observational design of the study – while ecologically valid – may have blunted the effects of sleeping less when compared to controlled sleep manipulation studies. Nevertheless, this naturalistic approach offers unique insights into how typical sleep patterns impact cortical function in everyday contexts. Future studies aiming to explore sleep-related effects on neural processing should consider combining observational and experimental methods to balance ecological validity with causal inference. As noted in

the present study, increasing participant sample sizes is especially important in such designs.

In summary, the current research underscores the importance of considering sleep as a potential confounding variable in child EEG studies. Even while MMN did not exhibit clear sleep-related differences, overall ERP amplitudes appear sensitive to natural variations in sleep quantity. Furthermore, the study introduces new perspectives on hemispheric effects in music processing and suggests opening the door to more regionally specific investigations into how sleep influences task-dependent neural responses. Ultimately, this study represents but a single drop in the expanding river of sleep and ERP research – yet it contributes to the current that is steadily flowing into a vast ocean of potential knowledge, deepening our understanding of sleep’s role in brain function, development, and the neurophysiological mechanisms underlying human cognition.

APPENDICES

Table A1. Participant Sleep Data

Sleep Metric	Test Day	TD-1	TD-2	TD-3	TD-4	TD-5	TD-6	Average
*Quality of Sleep (PSS)	1	1	1	2	2	3	1	1.57
*Sleep Times	07:00 PM - 09:00 AM	09:00 PM - 07:00 AM						
*Duration of Sleep (hours)	10	10	10	10	10	10	10	10.0
Quality of Sleep (PSS)	2	2	3	3	4	3	3	2.86
Sleep Times	11:00 PM - 09:30 AM	11:00 PM - 09:30 AM	10:30 PM - 06:50 AM					
Duration of Sleep (hours)	10.5	10.5	8.33	8.33	8.33	8.33	8.33	8.95
Quality of Sleep (PSS)	1	1	1	1	2	2	1	1.29
Sleep Times	08:00 PM - 06:30 AM	08:00 PM - 07:00 AM						
Duration of Sleep (hours)	10.5	11	11	11	11	11	11	10.93
Quality of Sleep (PSS)	3	1	1	2	1	2	1	1.57
Sleep Times	09:30 PM - 10:30 AM	09:30 PM - 08:00 AM	09:30 PM - 07:00 AM					
Duration of Sleep (hours)	13	10.5	9.5	9.5	9.5	9.5	9.5	10.14

Quality of Sleep (PSS)	1	5	1	1	1	1	1	1.57
Sleep Times	01:00 AM - 09:00 AM	10:00 PM - 08:00 AM	11:00 PM - 07:00 AM					
Duration of Sleep (hours)	8	10	8	8	8	8	8	8.29
Quality of Sleep (PSS)	1	1	1	1	1	1	1	1.0
Sleep Times	09:00 PM - 08:00 AM	09:00 PM - 08:00 AM	09:00 PM - 07:30 AM					
Duration of Sleep (hours)	11	11	10.5	10.5	10.5	10.5	10.5	10.64
Quality of Sleep (PSS)	3	1	1	5	5	1	1	2.43
Sleep Times	09:25 PM - 07:35 AM	09:25 PM - 07:35 AM	09:00 PM - 07:00 AM					
Duration of Sleep (hours)	10.166	10.166	10	10	10	10	10	10.05
Quality of Sleep (PSS)	1	1	2	4	1	1	1	1.57
Sleep Times	6:00 PM - 7:00 AM	9:00 PM - 7:00 AM	09:30 PM - 07:30 PM					
Duration of Sleep (hours)	13	10	10	10	10	10	10	10.43
Quality of Sleep (PSS)	2	1	1	1	1	1	1	1.14
Sleep Times	09:30 PM -	9:30 PM -	9:30 PM -	9:30 PM -	9:30 PM -	9:30 PM -	9:30 PM -	

	08:00 AM	7:00 AM	7:00 AM	7:00 AM	7:00 AM	7:00 AM	7:00 AM	
Duration of Sleep (hours)	10.5	9.5	9.5	9.5	9.5	9.5	9.5	9.64
Quality of Sleep (PSS)	2	2	3	2	3	2	2	2.29
Sleep Times	10:00 PM - 07:00 AM	09:00 PM - 07:00 AM						
Duration of Sleep (hours)	9	10	10	10	10	10	10	9.86
Quality of Sleep (PSS)	1	2	4	5	5	4	4	3.57
Sleep Times	09:30 PM - 07:05 AM	10:00 PM - 07:50 AM	09:30 PM - 07:05 AM					
Duration of Sleep (hours)	10.42	9.83	10.42	10.42	10.42	10.42	10.42	10.34
Quality of Sleep (PSS)	3	5	2	2	2	1	1	2.29
Sleep Times	01:10 AM - 09:00 AM	09:00 PM - 07:15 AM	09:00 PM - 07:15 AM	09:30 PM - 07:15 AM	09:30 PM - 07:15 AM	09:30 PM - 07:15 AM	10:00 PM - 09:30 AM	
Duration of Sleep (hours)	7.83	9.75	9.75	9.75	9.75	9.75	11.5	9.73
Quality of Sleep (PSS)*	3	1	2	1	2	2	5	2.29
Sleep Times*	10:30 PM - 7:45 AM	09:30 PM - 06:45 AM	09:30 PM - 06:45 AM	09:30 PM - 06:45 AM	10:30 PM - 08:30 AM	09:30 PM - 06:45 AM	10:45 PM - 10:00 AM	
Duration of Sleep (hours)*	9.25	9.25	9.25	9.25	10	9.25	11.25	9.64

Quality of Sleep (PSS)*	1	3	5	3	4	5	5	3.71
Sleep Times*	11:05 PM - 08:10 AM	10:40 PM - 08:00 AM	09:10 PM - 08:30 AM	09:10 PM - 07:15 AM	09:10 PM - 07:15 AM	09:10 PM - 07:15 AM	09:10 PM - 07:15 AM	
Duration of Sleep (hours)*	9.08	9.33	11.33	10.08	10.08	10.08	10.08	10.01

Summary of participant sleep data. Participants marked with an asterisk were excluded from analyses due to excessive data noise.

Table A2. 200 ms ERP Permutation Statistics

Condition	Site	Time Range (ms)	Cluster Mass	p value
intensity	Cz	200-400	12.031	<0.004
intensity	F3	0-200	13.306	<0.004
intensity	F4	200-400	15.247	<0.004
location	Fz	400-600	6.779	<0.004
location	Cz	200-400	9.596	<0.004
location	C4	0-200	4.710	<0.004
pitch	Fz	0-200	9.801	<0.004
pitch	C3	0-200	8.327	<0.004
pitch	Cz	0-200	9.468	<0.004
rhythm	Fz	0-200	13.438	<0.004
rhythm	C3	400-600	5.848	<0.004
rhythm	Cz	200-400	9.728	<0.004
rhythm	F3	200-400	9.950	<0.004
slide	Fz	0-200	19.949	<0.004
slide	Cz	0-200	17.293	<0.004
slide	C4	0-200	17.375	<0.004
timbre	Fz	0-200	21.314	<0.004
timbre	C3	200-400	11.548	<0.004
timbre	Cz	0-200	21.094	<0.004
timbre	F4	0-200	8.608	<0.004

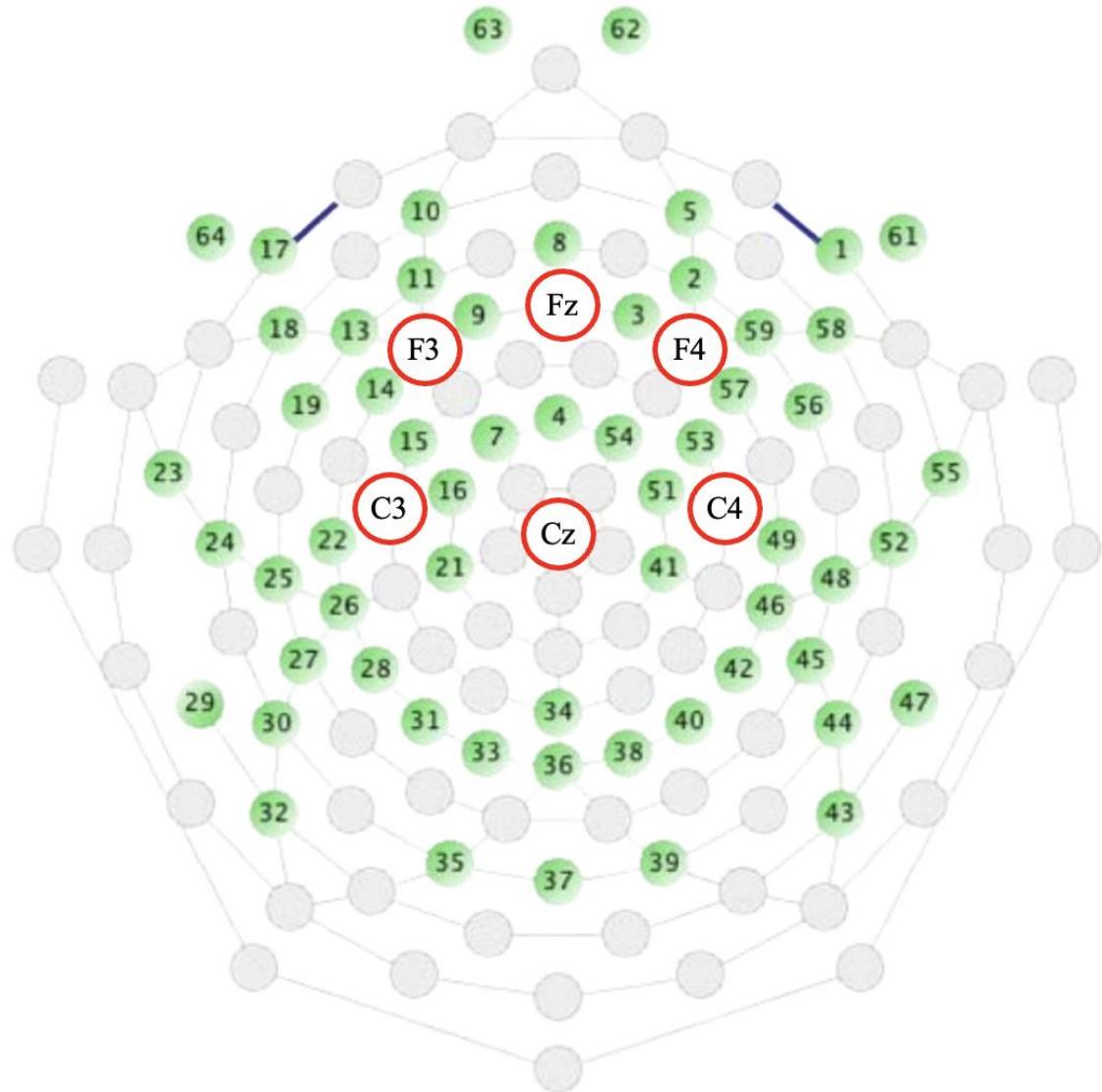
Results of cluster permutation testing for all music conditions at all scalp sites across different time windows (0-200 ms, 200-400 ms, 400-600 ms, and 600-800 ms) comparing sleep groups (Sleep More vs. Sleep Less).

Table A3. Hemispheric Activity Comparison

Sleep Group	Electrode Pair	t value	p value
less	Frontal (F4 vs F3)	5.042	0.007
less	Central (C4 vs C3)	-0.552	0.610
more	Frontal (F4 vs F3)	0.726	0.500
more	Central (C4 vs C3)	-0.494	0.642

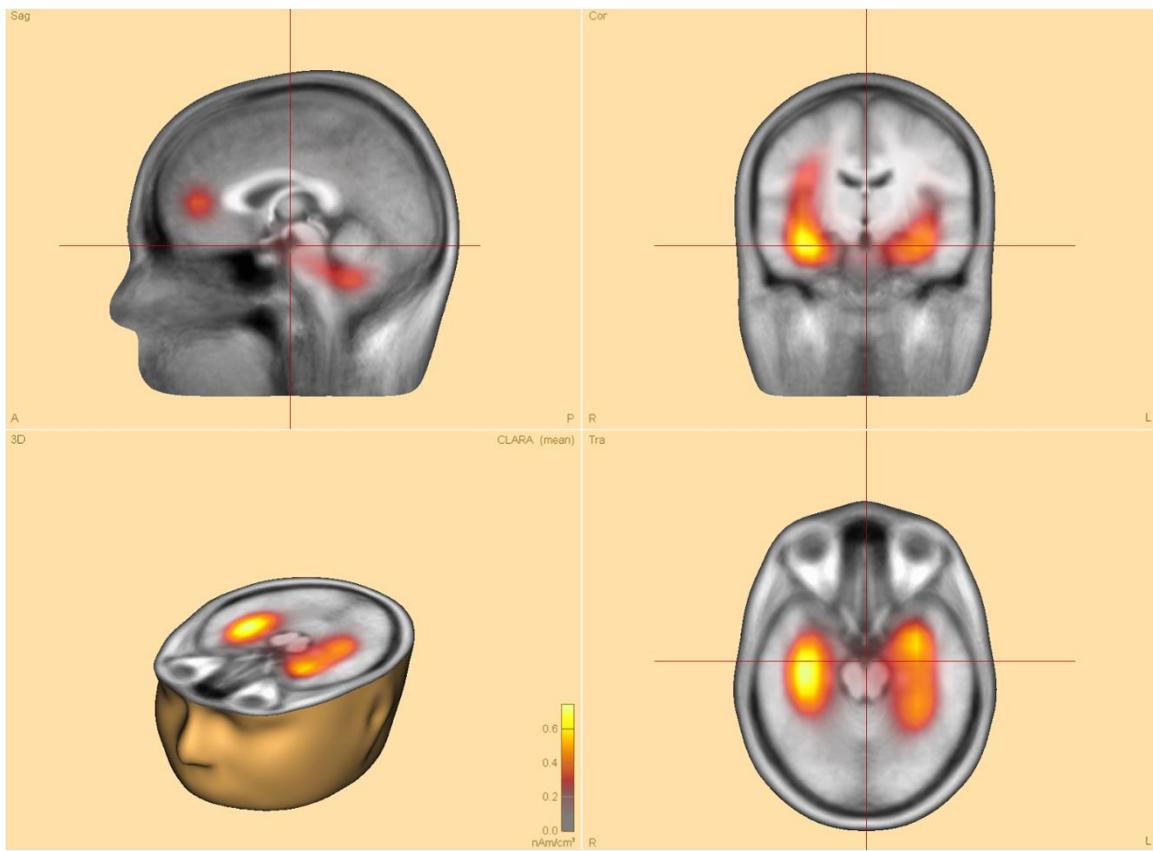
Results of t-tests comparing activation between homologous electrode pairs across hemispheres (left: F3 & C3; right: F4 & C4) within each sleep group. Positive t-values reflect greater activity in the right hemisphere.

Figure B1. 64-Channel HydroCel Geodesic Sensor Net



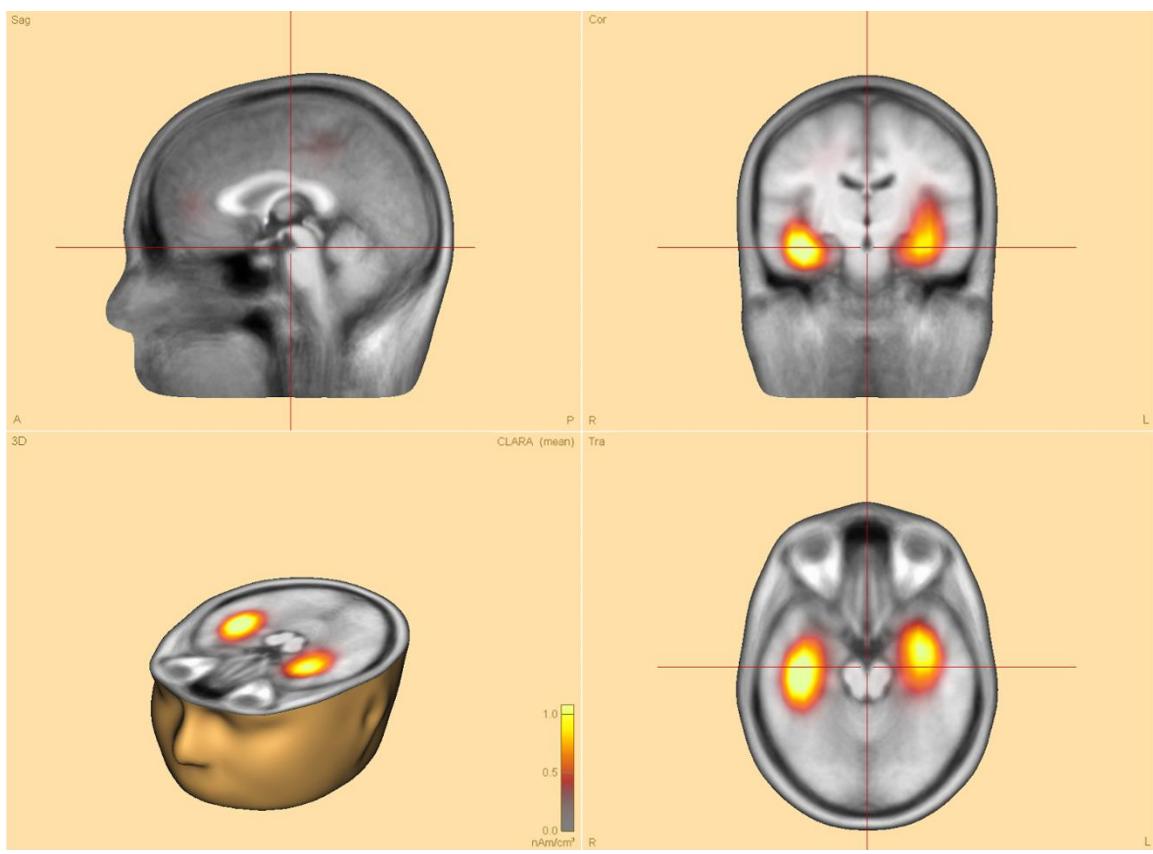
Electrode layout for the EEG sensor net used in the present study (Electrical Geodesics, Inc., 2007). Red circles indicate the six primary frontocentral sites (C3, C4, Cz, F3, F4, Fz) selected for statistical analysis.

Figure B2. Source Localization – Sleep More Group



Sagittal, coronal, 3D, and transverse MRI views (top left to bottom right) illustrating source-localized brain activity in the Sleep More group.

Figure B3. Source Localization – Sleep Less Group



Sagittal, coronal, 3D, and transverse MRI views (top left to bottom right) illustrating source-localized brain activity in the Sleep Less group.

REFERENCES

Åkerstedt, T., & Gillberg, M. (1990). Subjective and Objective Sleepiness in the Active Individual. *International Journal of Neuroscience*, 52(1–2), Article 1–2.
<https://doi.org/10.3109/00207459008994241>

Al-Shama, R. F. M., Uleman, J. F., Pereira, M., Claassen, J. A. H. R., & Dresler, M. (2024). Cerebral blood flow in sleep: A systematic review and meta-analysis. *Sleep Medicine Reviews*, 77, 101977. <https://doi.org/10.1016/j.smrv.2024.101977>

Boen, R., Quintana, D. S., Ladouceur, C. D., & Tamnes, C. K. (2022). Age-related differences in the error-related negativity and error positivity in children and adolescents are moderated by sample and methodological characteristics: A meta-analysis. *Psychophysiology*, 59(6), e14003. <https://doi.org/10.1111/psyp.14003>

Bortoletto, M., Tona, G. D. M., Scozzari, S., Sarasso, S., & Stegagno, L. (2011). Effects of sleep deprivation on auditory change detection: A N1-Mismatch Negativity study. *International Journal of Psychophysiology*, 81(3), 312–316.
<https://doi.org/10.1016/j.ijpsycho.2011.07.017>

Brown, L., Sherbenou, R. J., & Johnsen, S. K. (2010). *Test of Nonverbal Intelligence, Fourth Edition (TONI-4)* (Fourth). Pearson.

Callaway, E., & Halliday, R. A. (1973). Evoked potential variability: Effects of age, amplitude and methods of measurement. *Electroencephalography and Clinical Neurophysiology*, 34(2), 125–133. [https://doi.org/10.1016/0013-4694\(73\)90039-4](https://doi.org/10.1016/0013-4694(73)90039-4)

Chua, E. C.-P., Fang, E., & Gooley, J. J. (2017). Effects of total sleep deprivation on divided attention performance. *PLOS ONE*, 12(11), e0187098.
<https://doi.org/10.1371/journal.pone.0187098>

Colrain, I. M., & Campbell, K. B. (2007). The use of evoked potentials in sleep research. *Sleep Medicine Reviews*, 11(4), 277–293.
<https://doi.org/10.1016/j.smrv.2007.05.001>

Corsi-Cabrera, M., Arce, C., Río-Portilla, I. Y. D., Pérez-Garci, E., & Guevara, M. A. (1999). Amplitude Reduction in Visual Event-related Potentials as a Function of Sleep Deprivation. *Sleep*. <https://doi.org/10.1093/sleep/22.2.181>

Díaz-Leines, S., Gama-Moreno, O., Poblano, A., & Flores-Avalos, B. (2017a). Psychoacoustics and Neurophysiological Alterations after 30-36 Hours of Sleep Deprivation. *Neuropsychobiology*, 75(2), 89–95.
<https://doi.org/10.1159/000481350>

Díaz-Leines, S., Gama-Moreno, O., Poblano, A., & Flores-Avalos, B. (2017b). Psychoacoustics and Neurophysiological Alterations after 30-36 Hours of Sleep Deprivation. *Neuropsychobiology*, 75(2), Article 2.
<https://doi.org/10.1159/000481350>

DiStefano, C., Dickinson, A., Baker, E., & Jeste, S. S. (2019). EEG Data Collection in Children with ASD: The Role of State in Data Quality and Spectral Power. *Research in Autism Spectrum Disorders*, 57, 132–144.
<https://doi.org/10.1016/j.rasd.2018.10.001>

Donaldson, K. R., Jonas, K., Foti, D., Larsen, E. M., Mohanty, A., & Kotov, R. (2023). Mismatch negativity and clinical trajectories in psychotic disorders: Five-year stability and predictive utility. *Psychological Medicine*, 53(12), 5818–5828.
<https://doi.org/10.1017/S0033291722003075>

Dunn, L. M., Dunn, D. M., & Arrasmith, D. L. (2019). *Peabody Picture Vocabulary Test, Fifth Edition (PPVT-5)* (Fifth). Pearson.

Evers, S., Dannert, J., Rödding, D., Rötter, G., & Ringelstein, E.-B. (1999). The cerebral haemodynamics of music perception. *Brain*, 122(1), 75–85.
<https://doi.org/10.1093/brain/122.1.75>

Fitzgerald, K., & Todd, J. (2020). Making Sense of Mismatch Negativity. *Frontiers in Psychiatry*, 11, 468. <https://doi.org/10.3389/fpsyg.2020.00468>

Ford, T. C., Hugrass, L. E., & Jack, B. N. (2022). The Relationship Between Affective Visual Mismatch Negativity and Interpersonal Difficulties Across Autism and Schizotypal Traits. *Frontiers in Human Neuroscience*, 16, 846961.
<https://doi.org/10.3389/fnhum.2022.846961>

Garrido, M. I., Kilner, J. M., Stephan, K. E., & Friston, K. J. (2009). The mismatch negativity: A review of underlying mechanisms. *Clinical Neurophysiology*, 120(3), 453–463. <https://doi.org/10.1016/j.clinph.2008.11.029>

Gosselin, N., Mathieu, A., Mazza, S., Petit, D., Malo, J., & Montplaisir, J. (2006). Attentional deficits in patients with obstructive sleep apnea syndrome: An event-related potential study. *Clinical Neurophysiology*, 117(10), 2228–2235.
<https://doi.org/10.1016/j.clinph.2006.07.130>

Gumenyukw, V., Berne, C., Roth, T., Korzyukov, O., Jefferson, C., Kick, A., Spear, L., Tepley, N., & Drake, C. L. (2010). Shift Work Sleep Disorder is Associated with an Attenuated Brain Response of Sensory Memory and an Increased Brain Response to Novelty: An ERP study. *Sleep*, 33(5), 703–713.
<https://doi.org/10.1093/sleep/33.5.703>

Hao Hsieh, T., Yi Wu, K., & Fu Liang, S. (2018). Identification of Schizophrenic Patients and Healthy Controls Based on Musical Perception Using AEP Analysis. *Neuropsychiatry, 08*(04). <https://doi.org/10.4172/Neuropsychiatry.1000473>

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R., & Dement, W. C. (1973). Quantification of Sleepiness: A New Approach. *Psychophysiology, 10*(4), Article 4. <https://doi.org/10.1111/j.1469-8986.1973.tb00801.x>

Hosch, A., Harris, J. L., Swanson, B., & Petersen, I. T. (2023). The P3 ERP in Relation to General Versus Specific Psychopathology in Early Childhood. *Research on Child and Adolescent Psychopathology, 51*(10), 1439–1451.
<https://doi.org/10.1007/s10802-023-01061-0>

Kimura, D. (1964). Left-right Differences in the Perception of Melodies. *Quarterly Journal of Experimental Psychology, 16*(4), 355–358.
<https://doi.org/10.1080/17470216408416391>

Kliuchko, M., Heinonen-Guzejev, M., Vuust, P., Tervaniemi, M., & Brattico, E. (2016). A window into the brain mechanisms associated with noise sensitivity. *Scientific Reports, 6*(1), 39236. <https://doi.org/10.1038/srep39236>

Lambert, I., & Peter-Derex, L. (2023). Spotlight on Sleep Stage Classification Based on EEG. *Nature and Science of Sleep, 15*, 479–490.
<https://doi.org/10.2147/NSS.S401270>

Landa, L., Krpoun, Z., Kolarova, M., & Kasparek, T. (2014). Event-related Potentials and Their Applications. *Activitas Nervosa Superior, 56*(1–2), 17–23.
<https://doi.org/10.1007/BF03379603>

Lee, H.-J., Kim, L., Kim, Y.-K., Suh, K.-Y., Han, J., Park, M.-K., Park, K.-W., & Lee, D.-H. (2004). Auditory Event-Related Potentials and Psychological Changes during Sleep Deprivation. *Neuropsychobiology*, 50(1), 1–5.
<https://doi.org/10.1159/000077933>

Liberalesso, P. B. N., D'Andrea, K. F. K., Cordeiro, M. L., Zeigelboim, B. S., Marques, J. M., & Jurkiewicz, A. L. (2012). Effects of sleep deprivation on central auditory processing. *BMC Neuroscience*, 13(1), 83. <https://doi.org/10.1186/1471-2202-13-83>

Lindín, M., Correa, K., Zurrón, M., & Díaz, F. (2013). Mismatch negativity (MMN) amplitude as a biomarker of sensory memory deficit in amnestic mild cognitive impairment. *Frontiers in Aging Neuroscience*, 5.
<https://doi.org/10.3389/fnagi.2013.00079>

Maldonado, C. C., Bentley, A. J., & Mitchell, D. (2004). A Pictorial Sleepiness Scale Based on Cartoon Faces. *Sleep*, 27(3), 541–548.
<https://doi.org/10.1093/sleep/27.3.541>

Manzi, A., Nessler, D., Czernochowski, D., & Friedman, D. (2011). The development of anticipatory cognitive control processes in task-switching: An ERP study in children, adolescents, and young adults. *Psychophysiology*, 48(9), 1258–1275.
<https://doi.org/10.1111/j.1469-8986.2011.01192.x>

Maris, E., & Oostenveld, R. (2007). Nonparametric statistical testing of EEG- and MEG-data. *Journal of Neuroscience Methods*, 164(1), 177–190.
<https://doi.org/10.1016/j.jneumeth.2007.03.024>

Molfese, D. L., Ivanenko, A., Key, A. F., Roman, A., Molfese, V. J., O'Brien, L. M., Gozal, D., Kota, S., & Hudac, C. M. (2013). A One-Hour Sleep Restriction Impacts Brain Processing in Young Children Across Tasks: Evidence From Event-Related Potentials. *Developmental Neuropsychology*, 38(5), 317–336. <https://doi.org/10.1080/87565641.2013.799169>

Morris, A. M., So, Y., Lee, K. A., Lash, A. A., & Becker, C. E. (1992). The P300 event-related potential. The effects of sleep deprivation. *Journal of Occupational Medicine.: Official Publication of the Industrial Medical Association*, 34(12), 1143–1152.

Muller-Gass, A., & Campbell, K. (2019). Sleep deprivation moderates neural processes associated with passive auditory capture. *Brain and Cognition*, 132, 89–97. <https://doi.org/10.1016/j.bandc.2019.03.004>

Näätänen, R., Paavilainen, P., Rinne, T., & Alho, K. (2007). The mismatch negativity (MMN) in basic research of central auditory processing: A review. *Clinical Neurophysiology*, 118(12), 2544–2590. <https://doi.org/10.1016/j.clinph.2007.04.026>

Näätänen, R., & Picton, T. (1987). The N1 Wave of the Human Electric and Magnetic Response to Sound: A Review and an Analysis of the Component Structure. *Psychophysiology*, 24(4), 375–425. <https://doi.org/10.1111/j.1469-8986.1987.tb00311.x>

Näätänen, R., Shiga, T., Asano, S., & Yabe, H. (2015). Mismatch negativity (MMN) deficiency: A break-through biomarker in predicting psychosis onset.

International Journal of Psychophysiology, 95(3), 338–344.

<https://doi.org/10.1016/j.ijpsycho.2014.12.012>

Nath, R. D., Bedbrook, C. N., Abrams, M. J., Basinger, T., Bois, J. S., Prober, D. A., Sternberg, P. W., Gradinaru, V., & Goentoro, L. (2017). The Jellyfish Cassiopea Exhibits a Sleep-like State. *Current Biology*, 27(19), 2984-2990.e3.

<https://doi.org/10.1016/j.cub.2017.08.014>

Pallesen, S., Johnsen, B. H., Hansen, A., Eid, J., Thayer, J. F., Olsen, T., & Hugdahl, K. (2004). Sleep Deprivation and Hemispheric Asymmetry for Facial Recognition Reaction Time and Accuracy. *Perceptual and Motor Skills*, 98(3_suppl), 1305–1314. <https://doi.org/10.2466/pms.98.3c.1305-1314>

Panizza, D., Onea, E., & Mani, N. (2021). Early ERP Evidence for Children's and Adult's Sensitivity to Scalar Implicatures Triggered by Existential Quantifiers (Some). *Frontiers in Psychology*, 12, 657408.

<https://doi.org/10.3389/fpsyg.2021.657408>

Patrick, C. J., Bernat, E. M., Malone, S. M., Iacono, W. G., Krueger, R. F., & McGue, M. (2006). P300 amplitude as an indicator of externalizing in adolescent males. *Psychophysiology*, 43(1), 84–92. <https://doi.org/10.1111/j.1469-8986.2006.00376.x>

Peisch, V., Rutter, T., Wilkinson, C. L., & Arnett, A. B. (2021). Sensory processing and P300 event-related potential correlates of stimulant response in children with attention-deficit/hyperactivity disorder: A critical review. *Clinical Neurophysiology*, 132(4), 953–966. <https://doi.org/10.1016/j.clinph.2021.01.015>

Peng, Z., Dai, C., Cai, X., Zeng, L., Li, J., Xie, S., Wang, H., Yang, T., Shao, Y., & Wang, Y. (2020a). Total Sleep Deprivation Impairs Lateralization of Spatial Working Memory in Young Men. *Frontiers in Neuroscience*, 14, 562035.
<https://doi.org/10.3389/fnins.2020.562035>

Peng, Z., Dai, C., Cai, X., Zeng, L., Li, J., Xie, S., Wang, H., Yang, T., Shao, Y., & Wang, Y. (2020b). Total Sleep Deprivation Impairs Lateralization of Spatial Working Memory in Young Men. *Frontiers in Neuroscience*, 14, 562035.
<https://doi.org/10.3389/fnins.2020.562035>

Picton, T. W., Alain, C., Otten, L., Ritter, W., & Achim, A. (2000). Mismatch Negativity: Different Water in the Same River. *Audiology and Neurotology*, 5(3–4), 111–139.
<https://doi.org/10.1159/000013875>

Raz, A., Deouell, L. Y., & Bentin, S. (2001). Is pre-attentive processing compromised by prolonged wakefulness? Effects of total sleep deprivation on the mismatch negativity. *Psychophysiology*, 38(5), 787–795.

Rousselet, G. A. (2012). Does Filtering Preclude Us from Studying ERP Time-Courses? *Frontiers in Psychology*, 3. <https://doi.org/10.3389/fpsyg.2012.00131>

Sallinen, M., & Lyytinen, H. (1997). Mismatch negativity during objective and subjective sleepiness. *Psychophysiology*, 34(6), 694–702. <https://doi.org/10.1111/j.1469-8986.1997.tb02144.x>

Salmi, J., Huotilainen, M., Pakarinen, S., Siren, T., Alho, K., & Aronen, E. T. (2005). Does sleep quality affect involuntary attention switching system? *Neuroscience Letters*, 390(3), 150–155. <https://doi.org/10.1016/j.neulet.2005.08.016>

Shafer, V. L., Yu, Y. H., & Datta, H. (2010). Maturation of Speech Discrimination in 4- to 7-Yr-Old Children as Indexed by Event-Related Potential Mismatch Responses. *Ear & Hearing, 31*(6), 735–745.
<https://doi.org/10.1097/AUD.0b013e3181e5d1a7>

Sur, S., & Sinha, V. K. (2009). Event-related potential: An overview. *Industrial Psychiatry Journal, 18*(1), 70–73. <https://doi.org/10.4103/0972-6748.57865>

Tervaniemi, M., Saarinen, J., Paavilainen, P., Danilova, N., & Näätänen, R. (1994). Temporal integration of auditory information in sensory memory as reflected by the mismatch negativity. *Biological Psychology, 38*(2–3), 157–167.
[https://doi.org/10.1016/0301-0511\(94\)90036-1](https://doi.org/10.1016/0301-0511(94)90036-1)

van Alphen, B., Semenza, E. R., Yap, M., van Swinderen, B., & Allada, R. (2021). A deep sleep stage in *Drosophila* with a functional role in waste clearance. *Science Advances, 7*(4), eabc2999. <https://doi.org/10.1126/sciadv.abc2999>

Vuust, P., Brattico, E., Glerean, E., Seppänen, M., Pakarinen, S., Tervaniemi, M., & Näätänen, R. (2011). New fast mismatch negativity paradigm for determining the neural prerequisites for musical ability. *Cortex, 47*(9), 1091–1098.
<https://doi.org/10.1016/j.cortex.2011.04.026>

Whalen, D. J., Gilbert, K. E., Kelly, D., Hajcak, G., Kappenan, E. S., Luby, J. L., & Barch, D. M. (2020). Preschool-Onset Major Depressive Disorder is Characterized by Electrocortical Deficits in Processing Pleasant Emotional Pictures. *Journal of Abnormal Child Psychology, 48*(1), 91–108.
<https://doi.org/10.1007/s10802-019-00585-8>

Zhou, L., Wang, J., Wu, Y., Liu, Z.-Y., Yu, Y., Liu, J.-F., & Chen, X. (2021). Clinical significance of mismatch negativity in predicting the awakening of comatose patients after severe brain injury. *Journal of Neurophysiology*, 126(1), 140–147.
<https://doi.org/10.1152/jn.00658.2020>

Vita

Name

Rigel Leonard Baron

Baccalaureate Degree

*Bachelor of Arts,
University of New Mexico,
Albuquerque, New Mexico
Major: Psychology &
Criminology*

Date Graduated

December, 2020