Neural Signatures of Adverse Childhood Experiences: The Role of Alpha and Theta Spectral Dynamics and Phase Coherence in Cognitive Processing

By

Adam Carlo Gerard Mariotti, BScH

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Approved: Dr. Christine Lackner
Supervisor

Approved: Dr. Derek Fisher
Committee Member

Approved: Dr. Ghada Radwan
Committee Member

Approved: Dr. Jason Ivanoff
Committee Member

Approved: Dr. David Westwood
External Examiner

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Abstract

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Abstract*:* Adverse childhood experiences (ACEs) can permanently alter neurological structure and function, increasing the risks for a multitude of health issues. Here, we used auditory Go/NoGo and selective attention paradigms to explore the effects of ACE exposure, type of ACE, and subjective rating of the impact of the ACE(s) on the Error-related Negativity, N100, and N200 event-related potentials (ERPs). We calculated Event-Related Spectral Perturbation (ERSP) and Intertrial Coherence (ITC) at frontal sites on both alpha and theta frequency bands for these ERPs. We found that ERSP and ITC across frequency bands were associated with ACEs, such that when ERSP and ITC decreased, ACE exposure increased across dimensions. Our findings show that spectral power and phase synchrony are disrupted on alpha and theta frequency bands as a likely product of childhood adversity. These disruptions during response inhibition tasks suggest deficits in cognitive control at different stages of the information processing stream.

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Chapter 1: Introduction

1.0 Adverse Childhood Experiences

Childhood and adolescence are environmentally sensitive periods of neurological, physical, and psychological development. Many early life experiences can alter developmental trajectories. Trauma and prolonged stress can permanently alter brain structure and function; thus, it is important to minimize a child's exposure to adverse stressors (Chapman et al., 2007). However, this is not always possible; indeed, children are often exposed to adverse childhood experiences (ACEs) that can have lasting consequences. ACEs encompass a broad range of acute and chronic stressors experienced between the ages of 0 to 17. Examples of ACEs include an extended hospital stay, the death of a loved one, sexual or physical abuse, growing up in poverty, unstable households, parental substance use, and parental mental health problems (Lackner et al., 2018). Early exposure to the stressors mentioned above can increase the risk for a variety of health complications later in life, such as heart disease, cancer, lung disease, depression, substance abuse, sexually transmitted infections, and obesity (Chapman et al., 2007; Felitti et al., 1998).

Critically, the negative effects of ACEs can occur at not only the individual, but also societal, level. For instance, the estimated annual costs of ACEs in Europe and North America are $581 billion, and $748 billion, respectively (Bellis et al., 2019). This financial cost primarily stems from a burden placed on healthcare systems, social services, and associated incarceration costs. Significantly, 75% of these costs are related to individuals who have experienced two or more ACEs. Bellis and colleagues (2019) propose that a 10% reduction in ACE prevalence could save roughly $105 billion annually in North America and Europe combined. These findings highlight the prevalence of ACEs in the general population and underscore the importance of treating ACEs as the public health epidemic that they are. The negative impact of early stress

exposure has been well-documented for many years (Fride & Winstock, 1987; Miller & Cohen, 2001; Tottenham & Sheridan, 2010), emphasizing the importance of early intervention and support for children exposed to ACEs.

Thus, we explored individual differences in neurophysiology by investigating if patterns of event-related spectral perturbation (ERSP) and intertrial coherence (ITC) are related to ACE exposure, category, and subjective experience of ACEs in a young adult sample.

It is essential to appropriately categorize and identify specific childhood experiences considered to be adverse, as all ACEs may not have equivalent effects across individuals (Vu et al., 2022). The original CDC-Kaiser ACE study classified ACEs into two overarching categories: Abuse, and Household Dysfunction/Challenges (Felitti et al., 1998). Under their model, specific questionnaire items fall into these categories. First, physical, psychological, and sexual abuse fall into the abuse category. Second, household dysfunction/challenges include living with family members that abuse illicit substances, experience mental illness, have suicidal thoughts and behaviours, divorce or parental separation, incarcerated family members, and intimate partner violence or domestic violence (Felitti et al., 1998).

Beyond these three domains, Oh et al. (2018) published a meta-analysis reviewing ACE classification and providing a clear framework for grouping questionnaire items with ACE categories. This further exploration resulted in two additional ACE domains: neglect, and “other adversities”. Neglect items relate to both childhood physical and emotional neglect. Other adversities encompass childhood adversity that do not neatly fit into the other three categories. For instance, other adversities include experiences such as witnessing community violence, bullying, separation from parents, stays in foster care, and serious medical procedures or illnesses (Oh et al., 2018). These additional categories enhance sensitivity for detecting

individual differences in electrophysiological function as they relate to ACEs. Beyond this, we further investigated individual differences by collecting data on the subjective impact of events within these four domains. We aligned our analyses with the framework provided in Oh et al. (2018) to investigate the relationships among ERSP, ITC, and ACE dimensions. The first step in our analyses is exploring how, and where, ACEs may exert their harmful effects.

1.1 Neurological Development and ACEs

Certain brain regions, such as the prefrontal cortex (PFC) and hippocampus, are particularly susceptible to the negative effects of ACEs due to the large number of cortisol receptors located therein (Kim & Kim, 2019). Neurochemical alterations to these regions may result in long-term consequences such as dysregulated cortisol systems, reduced academic performance, and increased vulnerability to mental health issues (Kim & Kim, 2019; McLean et al., 2024). Furthermore, hypothalamic-pituitary-adrenal (HPA) axis activity acts as a statistical mediator in the relationship between childhood adversity and future psychopathology (Koss & Gunnar, 2017).

These findings highlight the importance of understanding how chronic and acute stress related to ACEs can affect the neurochemistry and structure of the PFC and hippocampus. Here we focus on the PFC, as it plays a critical role in executive functions, such as decision making, problem solving, and planning (Miller & Cohen, 2001). Beyond this, the PFC also helps with regulating behaviour, emotions, and with navigating social situations (Fuster, 2001). The PFC is heavily interconnected with the limbic system, a network of neurons involved with emotion regulation and the innate fight or flight system (Barbas, 2000; Fuster, 2001). Through the integration of these systems, the PFC exercises top-down control over cognitive and emotional processes to ensure that selected responses are appropriate and adaptive for a given situation

(Miller & Cohen, 2001). The PFC matures through early adulthood, making it susceptible to the influence of stress-inducing life events for a prolonged period (Gogtay et al., 2004).

ACE exposure can significantly alter the development and function of the PFC, primarily through cortisol secretion via the HPA axis. Exposure to chronic stress during critical periods of brain development has been consistently shown to produce significant changes in the structure and function of the PFC (Hanson et al., 2010; Teicher et al., 2016). For example, ACEs have been linked to lower PFC grey matter volume, thus compromising the ability of this region to perform executive functions optimally (Hanson et al., 2010). Prolonged stress responses often coincide with dysregulation of the HPA axis. Changes observed in HPA axis function can involve structural damage that affects the ability of innate stress-response systems to return to baseline levels of cortisol secretion (McEwen, 2000; McLean et al., 2024). Such changes can impair the PFC's ability to modulate emotional and behavioural responses; thereby, increasing the risk of psychological disorders like anxiety, depression, and impulse control disorders (Evans & Kim, 2013).

There are several underlying mechanisms as to why ACEs affect the neurophysiology of the PFC. When chronic ACE exposure occurs, this leads to a heightened response in the amygdala, which is a central component of the brain's fear and stress response system (Tottenham & Sheridan, 2010). Increases in amygdala activity are associated with a a greater stress response, which in turn leads to heightened cortisol production. This hyperactivation of the amygdala may interfere with the connection of the amygdala to the PFC (reducing the PFC's ability to regulate emotional expression). In an animal model, this disruption in connectivity can lead to enhanced dendritic sensitivity in the amygdala, increasing its susceptibility to stress, as well as dendritic retraction in the PFC, which reduces its regulatory capabilities (Holmes &

Wellman, 2009). Prolonged activation of the HPA access damages PFC neurons and synapses, leading to compromised cognitive control (Lupien et al., 2009). Neuroimaging has shown disrupted white matter integrity in ACE-exposed individuals, which may affect the communication within the PFC as well as between the PFC and other structures via white matter tracts, such as the left uncinate fasciculus (Eluvathingal et al., 2006).

Developmental neuroscientists have demonstrated a multitude of structural and functional changes that occur as a product of ACEs via EEG, fMRI, voxel-based morphometry, and PET. For example, Tomoda et al. (2011) found an average of a 14.1% increase in grey matter volume (GMV) in the left superior temporal gyrus, with a moderate effect size, in children who experienced parental verbal aggression (PVA). Grey matter is composed of neuronal bodies, axon terminals, and dendrites that assist in communication with nearby neurons. Due to the density of neuronal bodies in grey matter, these regions are heavily implicated in information processing (Tomoda et al., 2011). While these findings of increased GMV ran contrary to the proposed hypotheses by Tomoda et al. (2011), the findings suggest the dynamic nature of exposure to early adversity on the developing brain and potential compensatory mechanisms in place to support development. In this case, the left superior temporal gyrus may adapt to chronic stress and continue to develop normally, despite exposure to PVA.

Conversely, other neuroimaging studies have shown that children who experienced abuse also exhibited reduced ventrolateral PFC (VLPFC), as well as right lateral orbitofrontal cortex (OFC) thickness; regions heavily implicated in emotional regulation (Gold et al., 2016). Gold et al. (2016) deviated from previous literature typically investigating cortical volume by using cortical thickness as their outcome. Reductions in PFC thickness have been associated with mental illness across an array of internalizing and externalizing disorders (Fairchild et al., 2015;

McLaughlin et al., 2014). As a result of the differential effects of childhood adversity on neurological structure and development, it is important to investigate how these differences may present functionally. In doing so, we can examine whether ACEs leave individualized neural signatures, representative of neurophysiology and characterized via neuroelectric activity, that correspond to dimensions of childhood adversity.

1.2 Electroencephalography and Event-Related Potentials

One method that can be used to study individual differences in cognitive function is electroencephalographic (EEG) recordings. From EEG recordings, we can derive event-related potentials (ERPs), ERSP, and ITC (Makeig et al., 2004). By leveraging these three EEG analysis techniques, we aim to identify neurological signatures that correspond to ACEs. Furthermore, these insights could inform the development of targeted interventions and therapies to mitigate the effects of ACEs on cognitive and emotional well-being. As ERPs are particularly useful in characterizing neural responses to specific events, we use ERPs generated by various attention tasks requiring the use of executive functions and therefore the recruitment of PFC regions to evaluate individual differences in ERSP, as well as neural synchrony.

ERPs are derived from an EEG recording and provide an exceptionally sensitive method of indexing instantaneous brain function via observed electrical activity on a millisecond-by-millisecond basis (Light & Makeig, 2015). This degree of temporal resolution far outperforms the sensitivity of commonly used neuroimaging techniques, such as positron emission tomography (PET) or functional magnetic resonance imaging (fMRI), when recording changes in information processing and cognition. For example, Luck (2005) discusses the utility of ERPs in delineating the timing of cognitive events, helping separate dimensions of cognitive processing. This is of particular relevance to the present study, as we aim to sequentially map the

information processing stream from early sensory processing to response-selection, ending with response evaluation – in this case, error-monitoring.

An ERP waveform can be generated by averaging activity evoked by stimuli including tones or flashes of light, recognition, decision making, and responses to specific events (Luck, 2005). This allows average electrical activity to be captured that is time-locked to the onset of a stimulus or specific participant response. Specifically, this is advantageous for allowing researchers to empirically quantify subtle changes in cognition that may assist in clarifying observed behaviour (Makeig et al., 2004). For the present study, we aim to investigate how frontally localized cortical regions response at different stages of the information processing stream, and how these responses are associated with ACEs.

ERP components can be divided into three main categories: 1) *exogenous* sensory components triggered by the presence of a stimulus. 2) *endogenous* components reflecting task-dependent neural processes. 3) *motor* components that accompany the evaluation and execution of motor responses (Luck, 2005). Notably, some ERPs do not require an overt response from the participant; however, there must be an exogenous event to time-lock to (Luck, 2005). This allows researchers the ability to separate how an individual evaluates a stimulus and how they respond to it. This distinction is important for the present study, as we analyzed both stimulus-locked (likely prior to anticipatory responses), and response-locked neural signals. We evaluated the response-locked error-related negativity (ERN) in an auditory Go/NoGo task. We evaluated the stimulus-locked NoGo N2 (N2), and the stimulus-locked N100 (N1) waveforms collected from the Go/NoGo and selective auditory attention task, respectively. Together, these waveforms allow us to represent instantaneous neuroelectric activity at different stages of the information processing stream at specific scalp sites. For the purpose of the present study, we focused on a

collection of ERPs known as medial-frontal negativities, encompassing the ERN, N1, and N2 waveforms (Frömer et al., 2016; van Noordt & Segalowitz, 2012).

1.3 Error-Related Negativity ERP

The ERN is a fronto-centrally distributed waveform that is generated in response to errors (Shalgi et al., 2009). The ERN typically reaches maximum amplitude 50–80 ms following an erroneous response. The ERN was first demonstrated by Gehring et al. (1993) as a neurological support of the human conflict monitoring system during cognitively demanding tasks, such as the Go/NoGo task used in the present study. While the ERN is a waveform representative of error-monitoring and is generated in response to errors, a waveform called the correct-response negativity (CRN) of equal latency, yet less negative amplitude, is generated following correct responses (Suchan et al., 2007). The peak amplitude of the ERN is typically observed at Fz and FCz electrodes and generated by the underlying anterior cingulate cortex (ACC), that plays a role in conflict monitoring (Dehaene et al., 1994). The negative ERN deflection is thought to represent the rapid neurological evaluation of actions and outcomes; specifically, as it related to the mismatch between intended and actual responses (Holroyd & Coles, 2002).

The ERN is sensitive to individual differences in personality traits like anxiety, attention-deficit/hyperactivity disorder (ADHD), and obsessive-compulsive disorder (Hajcak et al., 2003; Klymkiw et al., 2017; Moser et al., 2013). Interestingly, those with higher levels of anxiety typically demonstrate greater ERN amplitudes. These findings suggest that the ERN may serve as a marker of individual differences in mental health and performance monitoring (Hajcak et al., 2003). Further, a study published by Lackner et al. (2018) found that those who experienced a greater number of ACEs demonstrated larger ERN-CRN differences. These findings suggest that

differences in ERN amplitude may correspond to specific neurological signals that are affected by ACEs.

1.4 NoGo N2 ERP

The NoGo N2 waveform plays a role in cognitive control and conflict monitoring and typically has a peak amplitude between 180–300 ms post stimulus presentation (Luck, 2014). The N2 is generated by tasks that are cognitively demanding and require individuals to inhibit responses to rare visual or auditory stimuli (Sur & Sinha, 2009). Trials requiring inhibition generate a NoGo N2. Previous literature has shown that the N2 wave can be observed in paradigms like the Go/NoGo task, where participants must withhold a response to a rare stimulus (Donkers & van Boxtel, 2004; Folstein & Van Petten, 2008; Klymkiw et al., 2017). Response inhibition activates neural circuitry responsible for differentiating and generating an N2 waveform; thus, providing empirical evidence for the underlying cognitive processes.

The NoGo N2 wave has been localized to the ACC and the PFC, both of which are heavily involved in cognitive control and error processing (Nieuwenhuis et al., 2003; van Noordt & Segalowitz, 2012; Yeung et al., 2004). The ACC plays an important role in conflict monitoring and detecting errors, while the PFC, especially the medial prefrontal cortex (mPFC), is involved in exercising cognitive control and adjusting behaviour based on feedback (van Noordt & Segalowitz, 2012). For instance, Nieuwenhuis et al. (2003) explored the role of the ACC in conflict monitoring and error detection in a series of fMRI studies. Specifically, a Stroop task was used to demonstrate increased activation in the ACC in response to the presentation of incongruent stimuli, which require a greater degree of cognitive control. Similarly, van Noordt and Segalowitz (2012) investigated the role of the PFC in exercising cognitive control and adjusting behaviour based on feedback by using a Go/NoGo task. They found that increases in

PFC activation were positively associated with better task performance during the response-inhibition Go/NoGo task. Lastly, Yeung et al. (2004) used EEG to explore the role of the PFC in conflict monitoring and error detection, and how this relates to task performance. They found that the PFC, the site of N2 generation, was predictive of task performance, such that as the PFC became more engaged, task performance increased.

Furthermore, research has shown that the N2 component is not only a marker of conflict detection but also of the PFC's preparatory mechanisms for inhibitory control (Kok, 1986; Falkenstein et al., 1999). During response inhibition tasks, high-conflict trials typically show a more negative N2 amplitude following stimulus presentation (Falkenstein et al., 1999; Nieuwenhuis et al., 2003; van Veen & Carter, 2002; Yeung et al., 2004). The increase in amplitude is thought to reflect the heightened effort used by the PFC to monitor and resolve conflicts, which in this case, refers to the inhibition of incorrect responses prior to overt response selection.

1.5 N100 ERP

The N100 (N1) is a stimulus-locked ERP, generated in response to the presentation of a stimulus, that peaks at approximately 100 ms (Näätänen & Picton, 1987). Similar to the ERN and N2, the N1 waveform is characterized by a large negative deflection observed at fronto-central sites. This waveform is generated in the primary auditory cortex and varies in amplitude depending on stimulus type, intensity, and the attentional state of the individual (Hillyard et al., 1973; Näätänen & Picton, 1987; Vaughan & Ritter, 1970). For example, Hillyard et al. (1973) found that the negative component of the N1 auditory evoked potential was significantly larger for attended compared to unattended tones. These findings provide additional support the use of

the selective attention task in the present study, as seen in Lackner et al. (2013). By including the N100 waveform, we can evaluate early sensory processes that occur prior to response selection.

Selective attention refers to the ability to filter out irrelevant information while simultaneously allocating mental resources to important information (Desimone & Duncan, 1995), supporting rational and goal-directed actions. The N1 provides a metric of individual differences in a selective attention capacity (Light & Makeig, 2015). We expect larger amplitudes of the N1 waveform as individuals focus on the relevant stimuli (i.e., target; Hillyard et al., 1973), as well as greater synchronization of this negative response. Further, more negative N1 waveforms have been demonstrated in conditions such as schizophrenia and ADHD; thus, alluding to a potential use of the N1 waveform as a biomarker for conditions related to childhood adversity (Barry et al., 2003; Koshiyama et al., 2021; Rosburg et al., 2008). Beyond comparing these differences exclusively through the evaluation of ERP amplitudes, we can also evaluate subtle changes in cognitive control and error detection using EEG-derived frequency bands.

1.6 Neurological Frequency Bands

EEG captures a range of frequencies of neural activity. In ascending order, delta (0.5-4 Hz), theta (4-7 Hz), alpha (8-12 Hz), beta (12-30 Hz), and gamma (>30 Hz) frequencies are present in various quantities as a function of cognitive demand. Each plays a unique role in cognitive activities such as attention, memory, learning, and emotional regulation (Clayton et al., 2015). During sleep, slow waveforms increase relative to wakefulness (Bernardi et al., 2019; Steriade, 2006). Higher frequency waveforms such as beta and gamma are typically observed while performing the most cognitively demanding tasks, with irregular patterns of alpha activity infiltrating the signal as attention lapses (Klimesch, 2012). Together, these waves reflect problem solving and the integration of information among neural structures in the PFC; thus,

demonstrating a role in the synchronization of key neural networks. Collectively, these frequency bands provide insights into cognition, as well as typical and atypical neurological function (Clayton et al., 2015).

Here, we focus on the role of alpha (8-12 Hz), and theta (4-7 Hz) in the information processing stream. Alpha waves generally occur when an individual is awake but not exerting an exceptional amount of cognitive effort (Ippolito et al., 2022). These waves are thought to reflect internal mental processes during information processing , attention, and working memory tasks (Ferat et al., 2022; Ippolito et al., 2022). Alpha waves are most prominent in the occipital lobe but can also be readily observed in frontal and parietal regions. Further, increased alpha activity is associated with reductions in stress, and inversely associated with alertness, both of which uniquely contribute to the effective regulation of emotions and management of task demands (Eidelman-Rothman et al., 2016).

Interestingly, increases in alpha activity may predict errors in response inhibition tasks. Mazaheri et al. (2009) found that in the time period leading up to stimulus presentation in a Go/NoGo task, participants exhibited significant increases in posterior and left central alpha activity prior to making an error (i.e., this pre-stimulus brain state led to an increased likelihood of committing an error). Due to the relationship of alpha activity and idleness, increases in alpha during a cognitively demanding task likely represent lapses in attention, leading to errors. Following the errors, participants demonstrated significant decreases in alpha activity; thus, indicating a re-orientation and increase in attention to the task (Mazaheri et al., 2009).

Beyond error prediction, altered alpha wave activity can be observed in a variety of psychiatric and psychological conditions. These alterations in alpha activity may occur organically but can also be the product of trauma experienced that effectively leads to long-term

alterations in neurological function (Eidelman-Rothman et al., 2016). For instance, individuals with early ACE exposure have been shown to have disruptions in alpha wave activity in the PFC consistent with those observed in anxiety and depressive disorders (Knyazev, 2007). In individuals with anxiety disorders, PFC alpha activity is typically reduced (Roxburgh et al., 2023). Notably, these disruptions in alpha activity can induce states of hypervigilance and increased levels of anxiety.

As a result of this decreased alpha activity, increased beta activity is commonly observed; thus, leading to a state of hyperarousal and heightened stress (Knyasev, 2007). This imbalance reflects the current understanding of anxiety disorders and highlights the role of alpha waves in maintaining a state of calm and relaxation. Similarly, individuals with depression have been shown to exhibit asymmetrical alpha activity, such that alpha power is reduced in the left frontal lobe compared the right (Zhou et al., 2023). Some theories suggest that this reflects an imbalance in the neurophysiological underpinnings of the approach-avoidance system (Uusberg et al., 2014). This imbalance further enhances depressive symptomology as it leads to reductions in motivation and increases in negative affect (Sutton & Davidson, 1997).

In addition to alpha, theta waves can also provide insight into neurocognitive processes. Theta activity represents slower waveforms that range from 4-7 Hz and are commonly associated with spatial exploration, inhibitory control, and memory retrieval (Eisma et al., 2021). Hippocampal theta oscillations play a role in encoding events into long-term memory (Creery et al., 2022). Further, asymmetrically distributed or excessive theta activity is often observed in ADHD, depression, learning disabilities, head injuries, brain lesions, and in individuals with impulse control difficulties (Moran & Hong, 2011; Zhang et al., 2022). For example, reduced frontal theta activity has been demonstrated during emotion regulation tasks in individuals with

borderline personality disorder, ADHD, and substance-use disorder (Haaf et al., 2024; McLoughlin et al., 2022). Focusing on frontally distributed theta activity, studies have found a connection between theta activity and cognitive control (Cavanagh & Frank, 2014; Eisma et al., 2021). Eisma et al. (2021) found that stimuli requiring a greater degree of cognitive control (i.e., target conditions) evoked higher theta power compared to nontarget conditions.

Theta oscillations are particularly involved in tasks that involve working memory, executive control, managing short-term memory load, and are sensitive to exogenously triggered stimuli associated with errors (Mussel et al., 2016). Accordingly, theta waves have been proposed to serve as a proxy for decision points, while simultaneously representing functional frontal brain network states. Interestingly, patterns of theta oscillations can predict levels of epistemic motivation (Mussel et al., 2016). This suggests that theta band activity is present during task-related decision making, an integral component of the present study.

As of 2019, 184 EEG studies have documented differences in alpha and theta activity across various psychiatric disorders (Newson & Thiagarajan, 2019). While effect sizes were not frequently reported, those that were documented ranged from small to moderate. These disorders include depression, ADHD, autism, addiction, bipolar disorder, anxiety, panic disorder, post-traumatic stress disorder, obsessive-compulsive disorder, and schizophrenia (Painter & Scannapieco, 2013). When considering the heightened risk an individual is at of experiencing these psychiatric illnesses due to ACE exposure, it is imperative to investigate these differences in neural activation to detect biomarkers that can assist in the early identification and treatment of these disorders by clinicians. Specifically, we aim to expand these results and determine if there are certain patterns of alpha and theta activation associated with ACEs. ERSP can be used

to evaluate changes in spectral power as a function of individual differences and task conditions (Rossi et al., 2014).

1.7 Event-Related Spectral Perturbation

ERSP can be used alongside ERP analysis to evaluate the spectral activity associated with target waveforms, thereby providing further insight into these neurocognitive processes (Rossi et al., 2014). Notably, ERSP represents complex neuroelectric responses in an interpretable format by mapping event-related synchronization (ERS) and event-related desynchronization (ERD) at particular scalp or source sites. ERS represents an increase in spectral power at a target scalp site or source, suggesting heightened functional engagement of these structures. Conversely, ERD represents a decrease in power representing the suppression of neural activity in a particular region at a particular frequency (Pfurtscheller & Lopes da Silva, 1999).

Cumulatively, investigating these differences in ERS and ERD on alpha and theta can detect subtle changes in cognition and activation of underlying neural networks, thus, highlighting individual differences in ERSP and how they may relate to ACEs. Yet, few neuroimaging studies have examined how particular patterns of neural activation, measured via alpha and theta ERSP relate to ACEs. However, some existing literature has demonstrated event-related reductions in spectral power in those with PTSD, compared to those without (DeLaRosa et al., 2019). Those who experienced PTSD showed significant ERD, represented by more negative ERSP, on the alpha frequency band when viewing threatening images compared to those without PTSD. Interestingly, the group without PTSD showed increases in theta ERSP following stimulus presentation, while the PTSD group did not, suggesting deficits in cognitive engagement on both alpha and theta frequency bands. These results are relevant to the current

study when considering the heightened risk of a PTSD diagnosis as a function of ACEs (Painter & Scannapieco, 2013). When evaluating changes in neuroelectric activity in the time-frequency domain, it is important to also consider the synchronization of target waveforms following stimulus onset to accurately quantify differences in neural activation.

1.8 Intertrial Coherence

ITC quantifies how consistently the phase of a neurological (electrical or magnetic) response to a stimulus, or a response, aligns over a series of trials (van Diepen & Mazaheri, 2018). ITC ranges between 0 and 1. An ITC value of 0 indicates an absence of phase synchrony, where an ITC value of 1 indicates perfect phase synchrony (Makeig et al., 2004). Further, lower ITC scores indicate that a neurological response is not consistent across repeated trials.

Past research has found that ITC is disrupted as a function of mental and physical health conditions. For example, individuals with schizophrenia have reduced alpha and theta ITC (Sauer et al., 2023). Specifically, patients with schizophrenia have a significantly lower alpha ITC in cingulate and temporal regions. The cingulate cortex is an important mediator of emotion regulation, sensing the environment, and action (Koshiyama et al., 2020). Desynchronization in this region can lead to behavioural issues commonly observed in schizophrenia, and autonomic nervous system (ANS) dysfunction, including changes in heart rate, blood pressure, and digestion (Rolls, 2019). ANS hyperactivity (Sicilaiano et al., 2022), and ANS hypoactivity (Obradović & Boyce, 2012) has been associated with childhood trauma. These findings demonstrate the role of the PFC in emotion regulation and cognitive control. Taken together, the associations among alpha and theta oscillations and numerous psychological conditions emphasize the importance of investigating how ACEs can affect these neurophysiological functions.

1.9 The Present Study

In the present study, we focus on fronto-centrally distributed alpha and theta due to the localization of neural generators of the N1, N2, and ERN waveforms in this region. Thus, by calculating alpha and theta ERSP and ITC for fronto-central sites we investigate neural mechanisms underlying cognitive processes associated with ACEs. To do this, we measure alpha and theta ERSP and ITC during the timing of the ERN, N2, and N1 to uncover neural signatures associated with dimensions of ACEs.

The objectives of this project are two-fold. First, we aimed to investigate the relationships among alpha and theta oscillatory activity and ACEs. Due to the role of alpha waves in internal mental processes, information processing, and working memory, we believe that fronto-central alpha synchrony may be disrupted with increased ACEs, resulting in distinct neurological signals. Further, due to the presence of asymmetrically distributed or excessive theta in common psychiatric conditions, we believe that those with a high number and severity of ACEs may display similar neural oscillatory patterns, thus, resulting in a more consistent negative inhibitory response – measured via ITC and ERSP during the ERN and N2. Overall, we expect that there may be specific oscillatory patterns associated with the subcategories of ACEs, namely, abuse, household dysfunction, and other adversities. Within each domain, we expect that the degree of neurological synchronization (measured via ITC) will decrease as the severity of ACEs increases.

Secondly, we aim to contribute to the growing body of neurodevelopmental literature exploring the effects of early life experiences on neurological function into adulthood. Past literature has primarily explored the effects of psychiatric disorders on ERSP and ITC. We aim to bridge the gap in the literature by exploring how ACEs can affect ITC and ERSP and result in

specific neural signatures representative of these ACEs. First, we aimed to examine alpha and theta power (ERSP) and neurological phase synchronization (ITC) of the ERN and N2 waveforms using an auditory Go/NoGo task. Next, we intended to explore the alpha and theta ERSP and ITC during the timing of the N1 waveform generated via an auditory selective attention task. From the resulting ERSP and ITC values across tasks and conditions, we constructed a series of multiple regression models to predict dimensions of ACEs and determine if certain patterns of neural activation emerge as a result of specific ACE exposure. Based on these proposed analyses, we developed three primary hypotheses outlined below.

1.10 Hypotheses

1. Prior literature has demonstrated reductions in alpha and theta ERSP as a product of conditions related to ACEs (Eidelman-Rothman et al., 2016; McLoughlin et al., 2022; Moran & Hong, 2011; Sutton & Davidson, 1997). Thus, we hypothesize that alpha and theta spectral perturbation during the response-locked ERN, and stimulus-locked N1 and N2 waveforms will negatively predict overall ACE dimensions, such that decreases in alpha and theta ERSP will predict a greater number of ACEs, abuses, household dysfunction, other adversities, and greater subjective impact of ACEs.
2. Existing literature has shown that ITC is disrupted as a result of mental and physical health conditions and can subsequently impact cognitive control and emotion regulation (Rolls, 2019; Sauer et al., 2023; Sicilaiano et al., 2022). Accordingly, we hypothesize that alpha and theta phase consistency during the response-locked ERN, and stimulus-locked N1 and N2 waveforms will negatively predict all ACE categories; such that decreases in alpha and theta ITC will predict a greater number of ACEs, abuses, household dysfunction, other adversities, and greater subjective impact of ACEs.

3. Collectively, we hypothesize that patterns of ERSP and ITC across frequency bands and ERPs will produce neural signatures predictive of exposure to our five ACE dimensions.

Chapter 2: Auditory Go/NoGo Paradigm

Method

2.0 Participants

One-hundred sixty-nine individuals completed the intake questionnaire and signed the consent form for a two-part study at Mount Saint Vincent University (MSVU), Canada (Age range 18–37 years). Of these 169, sixty-four completed the ACEs questionnaire and participated in the second part of this study, the EEG testing session. Two of these individuals were excluded from the final sample due to poor EEG recordings, resulting in a final sample of 62 (M_{age} = 24.4 years; 53 women). A power analysis revealed a required sample size of forty-nine individuals to achieve a medium effect size at the .05 significance level, with a power of .80 given our experimental design. Participants were recruited from the university and community population to increase the generalizability of our sample (Table 1). Our recruitment was primarily done through the Saint Mary's University (SMU) and MSVU SONA system, an online experiment management platform. University student participants may have been eligible to receive bonus points toward psychology courses at their professor's discretion. Additional recruitment was done through word of mouth, advertisements on Kijiji and social media platforms, as well as posters placed on SMU and MSVU campus. Participants were required to be between the ages of 18 to ~35-years-old (to promote accuracy in memory of childhood experiences), have normal or corrected-to-normal vision, normal hearing, and be fluent in English at the time of participation.

Table 1.

Participant Demographic and Adverse Childhood Experience Exposure

	N	Minimum	Maximum	Mean	Std. Deviation
Age	59	18	37	24.39	5.66
Number of ACEs	62	2	19	9.87	3.88
Abuse	62	0	5	1.84	1.41
Household Dysfunction	62	0	5	1.44	1.21
Other	62	2	11	6.60	2.49
Subjective ACE Rating	62	14	169	69.97	35.84

Note. Maximum values for each of these dimensions are as follows: Number of ACEs = 32, Abuse = 5, Household Dysfunction = 5, Other = 20, Subjective ACE Rating = 320.

2.1 Measures

Adverse Childhood Experiences Questionnaire

ACEs were assessed using an adapted version of the *Interpersonal Trauma and Animal-Related Experiences in Female and Male Military Veterans* questionnaire (Baker et al., 1998; Appendix F). Our version of the questionnaire contained 32 items with three subscales of abuse, household dysfunction, and other adversities, consistent with the framework provided by Oh et al. (2018). No items fell into the neglect category. For each subscale, the first item queried whether the participant experienced the ACE or not; follow-up questions for those who responded yes were then asked to assess the subjective experience of the ACE. Questions investigating the subjective nature of the ACE include, how much stress did this cause you (from one to ten), how often do you currently think about this (once per week to never), when did it occur (approximate age), and how long did the initial stress or trauma of this event last (minutes to years). In the present study, we used the rating from one to ten of how much stress the event caused as a measure of subjective stress associated with a particular ACE. We summed these

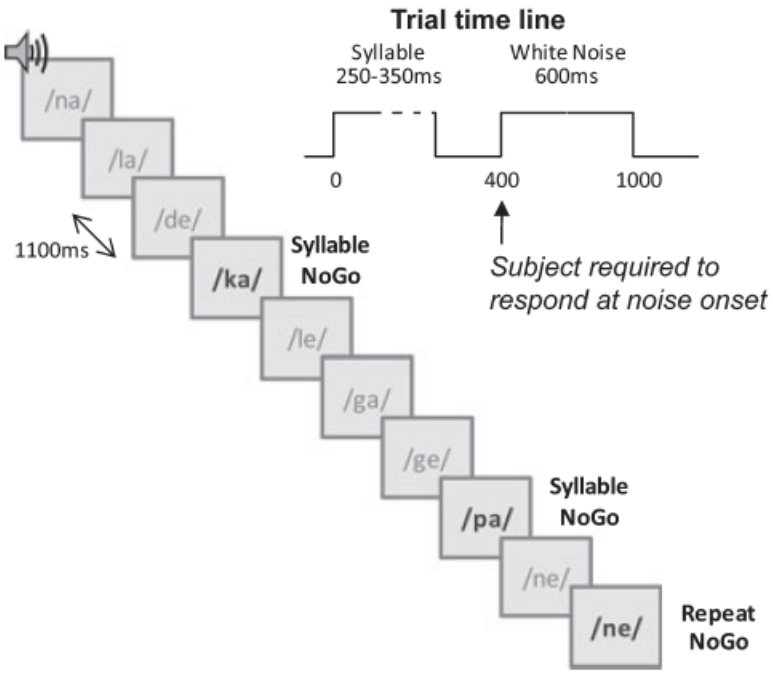
scores across items to derive a total score representing the cumulative subjective stress across ACEs for each individual. Cronbach's alpha was calculated at .903 for this measure.

Go/NoGo Paradigm

The Go/NoGo paradigm is modeled after Shalgi et al. (2009). Participants were seated approximately 90cm from the computer screen and asked to press the 'green button' on a Cedrus response box for every spoken consonant-vowel syllable that they heard (Go trials: KE, PE, NA, NE, LA, LE, GA, GE, DA, DE), but to withhold a response when they heard 'KA' or 'PA' (NoGo trials). Consonant-vowel syllables were presented binaurally through Etymotic 3C in-ear headphones to reduce the potential of electrical interference from standard earphones (Etymotic Research Inc.). White noise was presented for 600 ms between consonant-vowel syllables to prevent the potential blending of syllables. NoGo trials were presented approximately 18% of the time, with Go trials representing 82% of trials (Figure 1; Shalgi et al., 2009). Thus, approximately 108 NoGo, and 492 Go stimuli were presented in randomized order over six total blocks consisting of 100 syllables per block. This serves to create a Go response tendency, and thereby increase the number of errors made on NoGo trials. Response time and accuracy were recorded for correct Go responses (green button presses to the syllables listed above), only response time and accuracy were recorded for error NoGo responses (correct responses are the absence of a response).

Figure 1.

Visualization of Auditory Go/NoGo Paradigm



Note. Our paradigm was identical to that used in Shalgi et al. 2009.

2.2 Data Collection and Analysis

Statistical analyses for behavioural data generated in E-Prime were carried out using the default statistical analysis program in E-Prime, E-DataAid. We used E-DataAid to merge behavioural data files and calculate condition-specific averages for the Go/NoGo task. Beyond this, behavioural data were further analyzed using SPSS (IBM Corp).

Electrophysiological Recordings

Electrophysiological recordings were taken onsite using EEG equipment at the Electrophysiology Lab at MSVU (ELM). ERPs were extracted from EEG activity recorded from an electrode cap with Ag^{+}/Ag^{+}-Cl^{-} ring electrodes at 64 sites according to the 10–20 system of

electrode placement, with bilateral mastoid electrodes built into the electrode cap. Electrodes were also placed on the mid-forehead and nose to serve as ground and reference, respectively. All electrode impedances were below 5kΩ at the outset of recordings. Electrical activity was recorded with an amplifier bandpass of 0.1 to 1000 Hz, digitized at 500 Hz, which effectively applies a low pass filter of 250Hz, and stored on a hard drive for later offline analysis.

EEG Data Pre-Processing

EEG data were processed and analyzed using an open-access plugin for MATLAB called EEGLAB (Delorme & Makeig, 2004). Pre-processing of EEG data were done using the EEG Integrated Platform Lossless pipeline (EEG-IP-L), as described in Desjardins et al. (2021). The EEG-IP-L was used to identify independent components, channels, and signal activity that contained artifacts using a series of set criteria functions (Heffer et al., 2023). This was achieved using an independent components analysis (ICA). ICA allows for the separation of neurological, muscular, and cardiac electrical activity recorded by the encephalogram, and thus removes sources of noise from the data (Delorme & Makeig, 2004). After re-referencing to average and filtering the data from 1-30 Hz, we applied the infomax algorithm (Bell & Sejnowski, 1995) using EEGLAB (Delorme & Makeig, 2004). The infomax algorithm is used to separate mixed signals into statistically independent components. Here, the infomax algorithm further separated sources of neuroelectric activity from all other sources of electric activity, while retaining the maximum amount of neurological signal in our recordings (Bell & Sejnowski, 1995).

Upon decomposition of the electrical signal, the ICLabel plugin was applied to determine percent composition of each component as a product of eye blinks, neural activity, cardiac, eye movement, line noise, and other sources, as described in Pion-Tonachini et al. (2019). Independent components composed primarily of neuroelectric activity were retained). A

secondary manual inspection of signal composition was performed if channels failed to meet the set threshold of 65% composition of neurological activity. Beyond simply meeting the 65% threshold, quality assessments were performed based on component topographical maps, continuous activation, and power spectrum profiles (Desjardins et al., 2021). Cleaned EEG data were then projected back to the scalp for analysis. Spherical-spline interpolation was used to recreate any channels previously removed for data quality problems.

EEG Post-Processing Analyses

Following the pre-processing stages of data analysis, we began post-processing analyses by segmenting the resulting data into time-locked epochs from -300 to 800 ms. We used EEGLAB to create ERP overlays and topographies to assess the specific times and locations of maximal ERP activation. We identified Fz as the site of maximal amplitude for the ERN and N2 waveforms and Cz as the site of maximal amplitude for the N1 waveform. The most negative voltages for the Go/NoGo paradigm were -19.02 to 119.12 ms for the ERN, and 209.21 to 299.30 ms for the N2. Similarly, we determined the time-locked most negative deflection for the N1 waveform during the auditory selective attention task to be 69.07 to 131.13 ms.

From there, we applied EEGLAB scripts to extract frequency data from two hundred evenly spaced frequency time points beginning at 3Hz and concluding at 30Hz, with an approximate spacing of .13Hz (Delorme & Makeig, 2004). We completed a further decomposition of these data to isolate alpha (4–7 Hz), and theta (8–12 Hz) activity. Additionally, we applied similar scripts to extract four hundred time points beginning at -715.72 ms and concluding at 715.72 ms (approximately four milliseconds between data points). Upon clearly defining our time-frequency parameters, we applied the “newtimef” function to calculate average ERSP and ITC across the Fz and Cz channels (Delorme & Makeig, 2004). These calculations

were executed by EEGLAB and relied on a fast Fourier Transform (lowest frequency), and wavelet decomposition (highest frequency) with the default EEGLAB settings (cycles were set at [3,0.5]).

We performed these calculations for response-locked Go/NoGo data, as well as stimulus-locked data for the Go/NoGo and selective attention tasks. Thus, the final calculations resulted in four hundred ERSP and ITC values evenly spaced from -715.72 ms to 715.72 ms on the alpha and theta frequency bands. Lastly, we independently averaged ERSP and ITC activity corresponding to our target time-frequency parameters for the ERN, N2, and N1 waveforms during the timing of their peak negativity (described above). This final step provided us with an integer representing average ERSP and ITC activation for each ERP on the alpha and theta frequency bands. These values were used to evaluate if certain patterns of alpha and theta can differentially predict dimensions of ACE exposure.

Results

2.3 Behavioural Data

To ensure that the Go/NoGo task functioned as intended, we first conducted behavioural analyses to confirm differences among conditions. Mean accuracy and response times for Go and NoGo trials are reported in Table 2. Response times or accuracy that fell more than two and a half standard deviations from the mean were excluded from the final behavioural analyses.

Table 2.

Average Accuracy and Reaction Times Across Conditions of the Go/NoGo Paradigm

	Accuracy (SD)	RT (SD)
Go	0.92 (.09)	334.32 (98.68)
NoGo	0.77 (.17)	316.72 (124.64)

Note. Response Time and Standard Deviation (in parentheses) are reported in milliseconds. RT for NoGo trials represents error responses.

We then conducted two paired samples *t*-tests to investigate differences in response time and accuracy as a function of Go and NoGo trials. A significant difference in accuracy was observed between Go and NoGo trials ($t = 6.86$; $p < .01$), with Go accuracy being significantly higher than NoGo accuracy. Differences in reaction times between correct Go and incorrect NoGo trials were significant ($t = 2.23$, $p = .03$), with NoGo errors being slightly faster than Go reaction times.

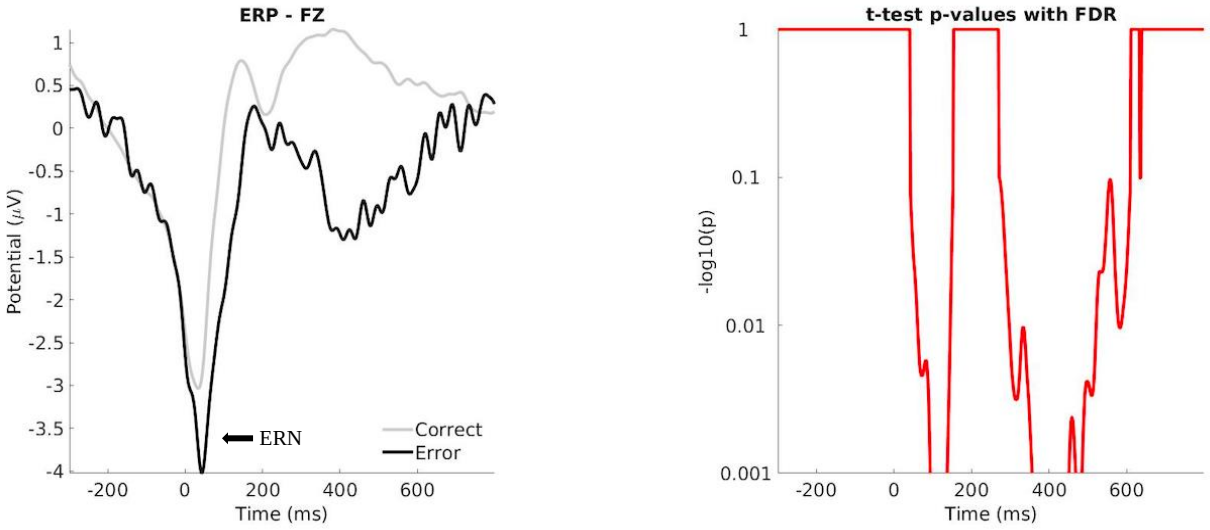
2.4 ERP Analysis

Using robust bootstrapped statistics with False Discovery Rate (FDR) correction, we determined if there were amplitude differences between conditions as a function of participant response, in essence, confirming if we could replicate traditional ERN – CRN differences (Figure 2). By applying the FDR filter, no more than 0.05% false positives with a 0.05 *p*-value

threshold were retained (Wilcox, 2012). See Delorme & Makeig (2004) for full details on robust estimation and hypothesis testing in EEGLAB.

Figure 2.

Response-locked Event-Related Potential Overlay with FDR Significance Values at Fz

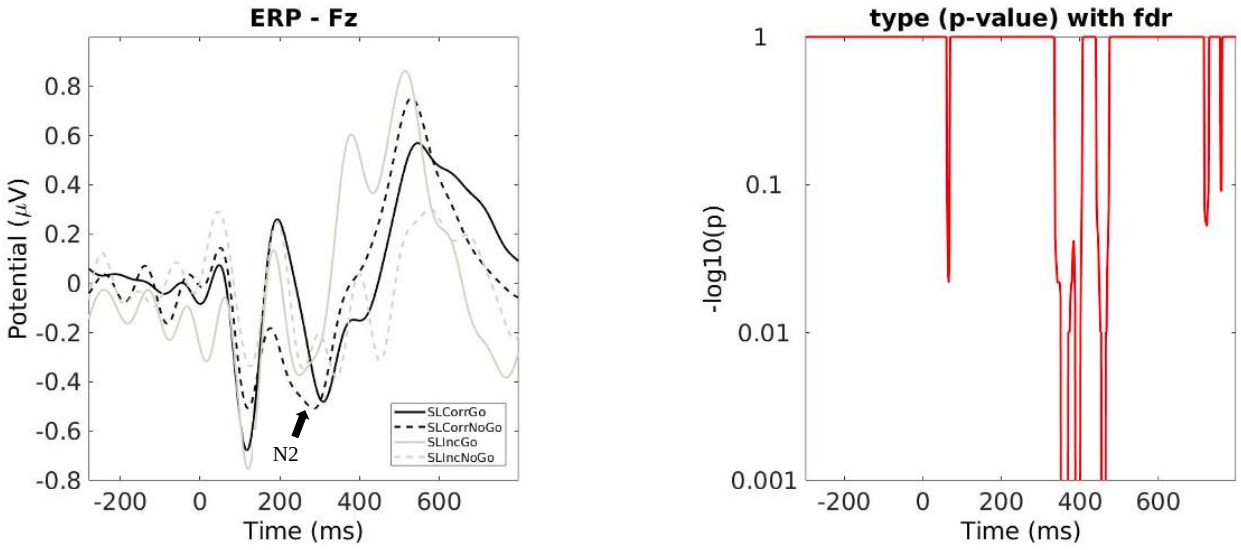


As seen in Figure 2, there are significant differences (p <.01) in ERP amplitude between correct and error responses from 40 to 170 ms and 290 to 560 ms post-response, the first of which represents the ERN. Here, the peak negative amplitude of the ERN can be observed at the Fz electrode site, 44 ms following the participant response.

We conducted a similar analysis with the stimulus-locked ERP data to evaluate condition differences in N2 amplitude (see Figure 3). As seen in Figure 3, there are significant differences (FDR corrected, p <.05) in ERP amplitude among stimulus-locked conditions at 55ms and intermittently from 365 to 440 ms post-stimulus. These results warranted further analyses of condition differences in N2 amplitude. Here, the peak negative amplitude of the N2 can be observed at the Fz electrode site, 255 ms following the participant response.

Figure 3.

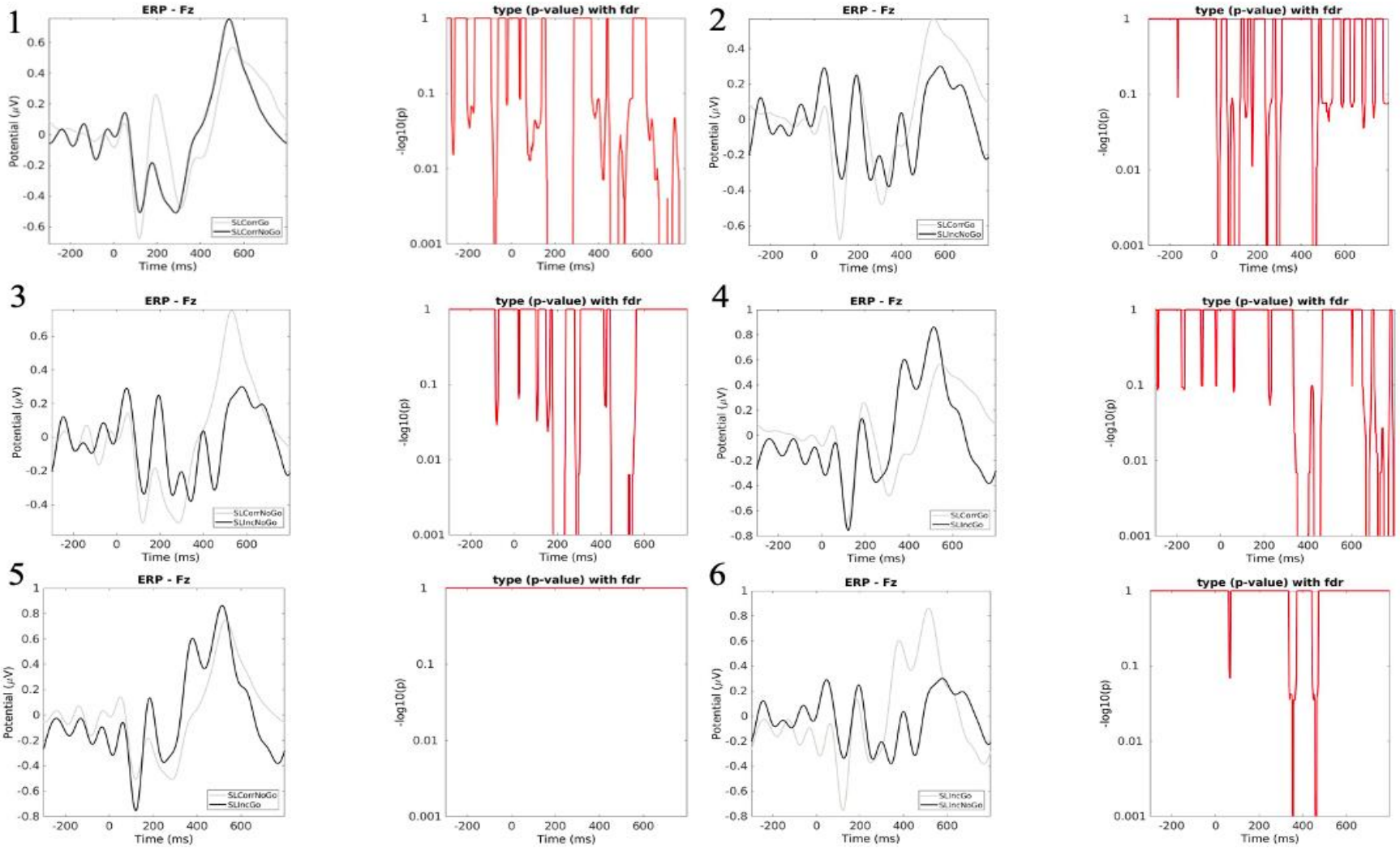
Stimulus-locked Event-Related Potential Overlay with FDR Significance Values at Fz



We then conducted a series of *t*-tests comparing all permutations of conditions. Our series of *t*-tests revealed significant differences in N2 amplitude between correct Go and correct NoGo responses (p <.01; contrast 1), such that correct NoGo responses had a more negative N2 amplitude. Significant differences were observed in N2 amplitude between correct Go and incorrect NoGo responses (p <.01; contrast 2), with correct Go N2 amplitude being more negative. There were significant differences in N2 amplitude between correct NoGo and incorrect NoGo responses (p <.01; contrast 3), with correct NoGo responses producing a more negative N2. No significant differences in N2 amplitude emerged for correct Go vs. incorrect Go, correct NoGo vs. incorrect Go, and incorrect Go vs. incorrect NoGo trials (p's >.05; contrasts 4-6). All results can be seen in Figure 4 below.

Figure 4.

Stimulus-locked Event-Related Potential Condition Comparisons with FDR Significance Values at Fz



Note. N2 peak amplitude can be observed from 200–300m

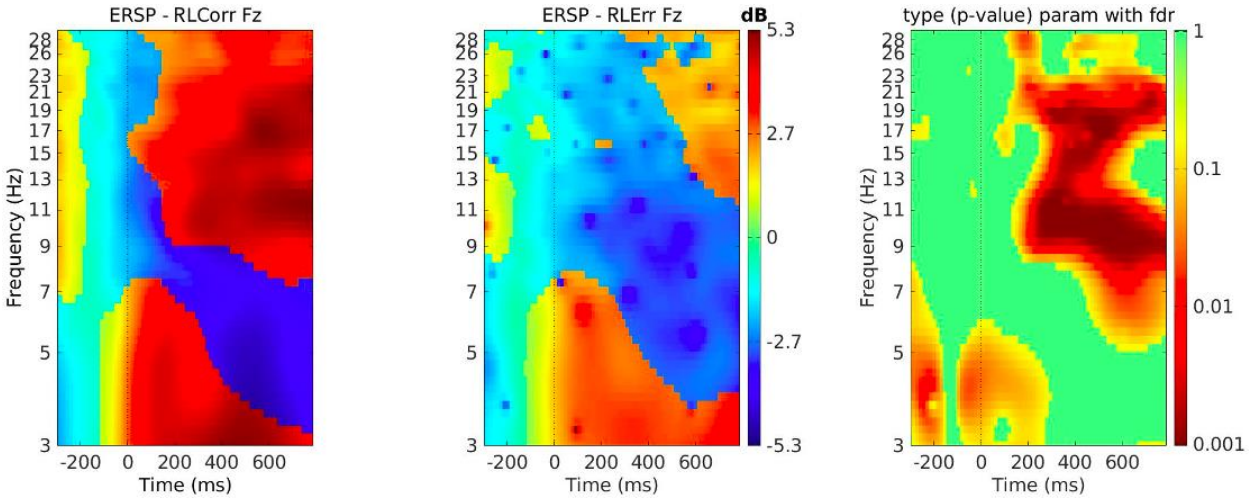
Once it was confirmed that there were behavioural and neurological differences between conditions, we extended our analyses into the time-frequency domain. To do so, we conducted both ERSP and ITC analyses with response and stimulus-locked data. We calculated ERSP and ITC values from 1–30 Hz, but our focus was alpha and theta during maximal ERP timings. Existing literature suggests that the maximum negative amplitude of the ERN can be seen between 40 and 120 ms after an error response (Crivelli & Balconi, 2017), and the most-negative peak of the N2 can typically be observed between 180 to 300 ms after a NoGo stimulus presentation (Luck, 2014). By evaluating individual peaks in our overlays and using data visualization tools in EEGLAB, we determined the ERN to fall between -19.12 to 121.22 ms and the N2 to fall from 209.21 to 299.30 ms and used this as a frame of reference for calculating response and stimulus-locked ERSP and ITC data, respectively.

2.5 Response-Locked ERSP

To assess ERSP differences between correct and error responses, response-locked ERSP activity was extracted at Fz (site of maximal ERN amplitude). From these, we conducted a robust bootstrap FDR corrected *t*-test to evaluate condition differences (see Figure 5). We observed significant ERSP differences in theta from -30 to 250 ms following the participant response, with greater theta ERSP for corrects relative to errors. We observed significant alpha ERSP differences from 220 to 800 ms, with greater ERSPs for corrects compared to errors.

Figure 5.

ERSP at Fz as a Function of Condition



Linear Regressions.

Prior to conducting our regression analyses, we performed bidirectional collinearity diagnostics to ensure our regression models met necessary collinearity conditions. We used the Variation Inflation Factor (VIF) to evaluate collinearity. VIF is used to evaluate the amount of collinearity in regression analyses, indicating if there is a strong correlation between independent variables (Marcoulides & Raykov, 2019). Highly correlated predictor variables can adversely affect regression results and make it challenging to investigate true effects. VIF scores below one suggest no correlation, values between one and five suggest a moderate correlation, those between five and ten indicate a moderate to high correlation, and those greater than ten indicate a high correlation and significant multicollinearity that must be corrected (Marcoulides & Raykov, 2019).

We found that the number of ACEs, and subjective rating of ACEs were highly colinear (see Appendix A; Table A1). Further, subjective ACE rating and the number of ACEs were highly correlated with one another (Appendix A; Table A2). As subjective rating of ACEs is an

integral component of the present study, we wanted to ensure the inclusion of this dimension in our regression models and elected to observe associations among neural oscillatory activity and our five ACE dimensions. Prior to conducting this series of regressions, we also investigated the collinearity of ERSP and ITC predictor variables, ensuring variables did not exceed VIF thresholds (Appendix A; Table A3).

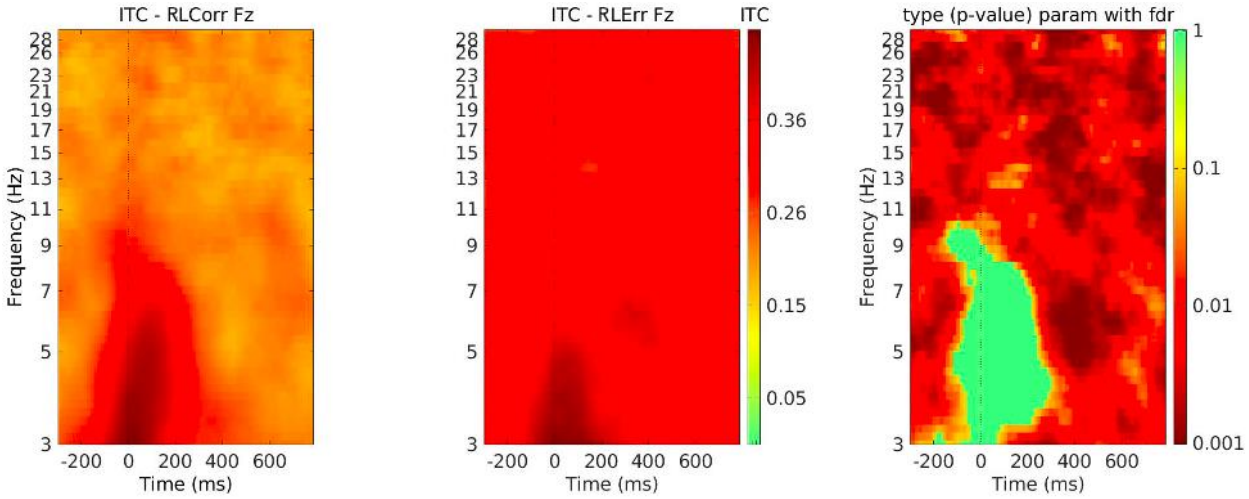
To evaluate individual differences and investigate if these patterns of ERSP could predict exposure to ACEs, we conducted two multiple linear regression analyses – one using alpha ERN ERSP, and the other using theta ERN ERSP to predict number of ACEs, subjective rating of ACEs, abuse, household dysfunction, and other adversities. Independent variables included alpha ERN ERSP, and theta ERN ERSP for correct and errors responses. None of these analyses emerged as significant across both frequency bands (see Appendix B for results).

2.6 Response-Locked ITC

A response-locked ITC analysis was performed to assess alpha and theta phase synchronization across trials. This revealed a significant difference in alpha ITC during the ERN at Fz. Interestingly, low-wave theta ITC from 0 to 200 ms following participant response did not show significant differences between conditions (Figure 6).

Figure 6.

ITC at Fz as a Function of Condition



Linear Regressions.

To investigate if patterns of ITC could predict exposure to ACEs, we conducted two multiple linear regression analyses – one using alpha ERN ITC, and the other using theta ERN ITC to predict number of ACEs, subjective rating of ACEs, abuse, household dysfunction, and other adversities. Independent variables included alpha ERN ITC, and theta ERN ITC for correct and errors responses. None of these analyses emerged as significant across both frequency bands (see Appendix A).

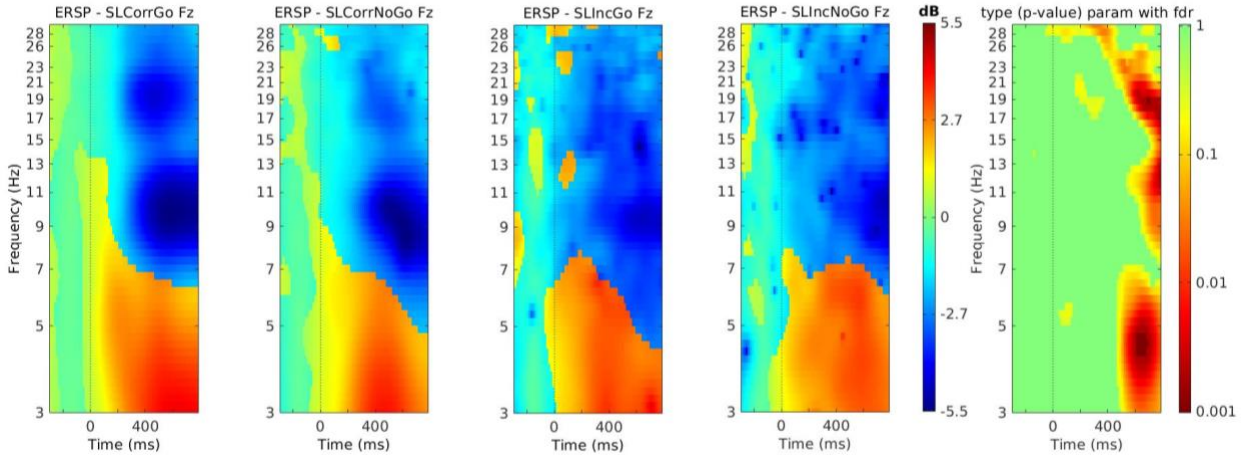
2.7 Stimulus-Locked ERSP

To further explore the relationships among ERSP and dimensions of ACEs, we extracted stimulus-locked ERSP activity across Go/NoGo task conditions. Specifically, we compared the ERSP of correct Go, correct NoGo, incorrect Go, and incorrect NoGo responses at Fz, the site of maximal amplitude of the stimulus-locked N2 waveform (see Figure 7). No significant differences across conditions emerged in the 300 ms following stimulus presentation. However, slightly greater levels of alpha ERSP can be observed from 180 to 270 ms when comparing

incorrect Go and incorrect NoGo responses. Across the four conditions, we observed significant ERSP differences in alpha and theta activity from 550 to 800 ms. These preliminary findings warranted further follow up described below.

Figure 7.

Comparative Analysis of ERSP at Fz as a Function of Condition



Note: The right-most panel shows the results of the omnibus 4-level 1-way ANOVA F-test

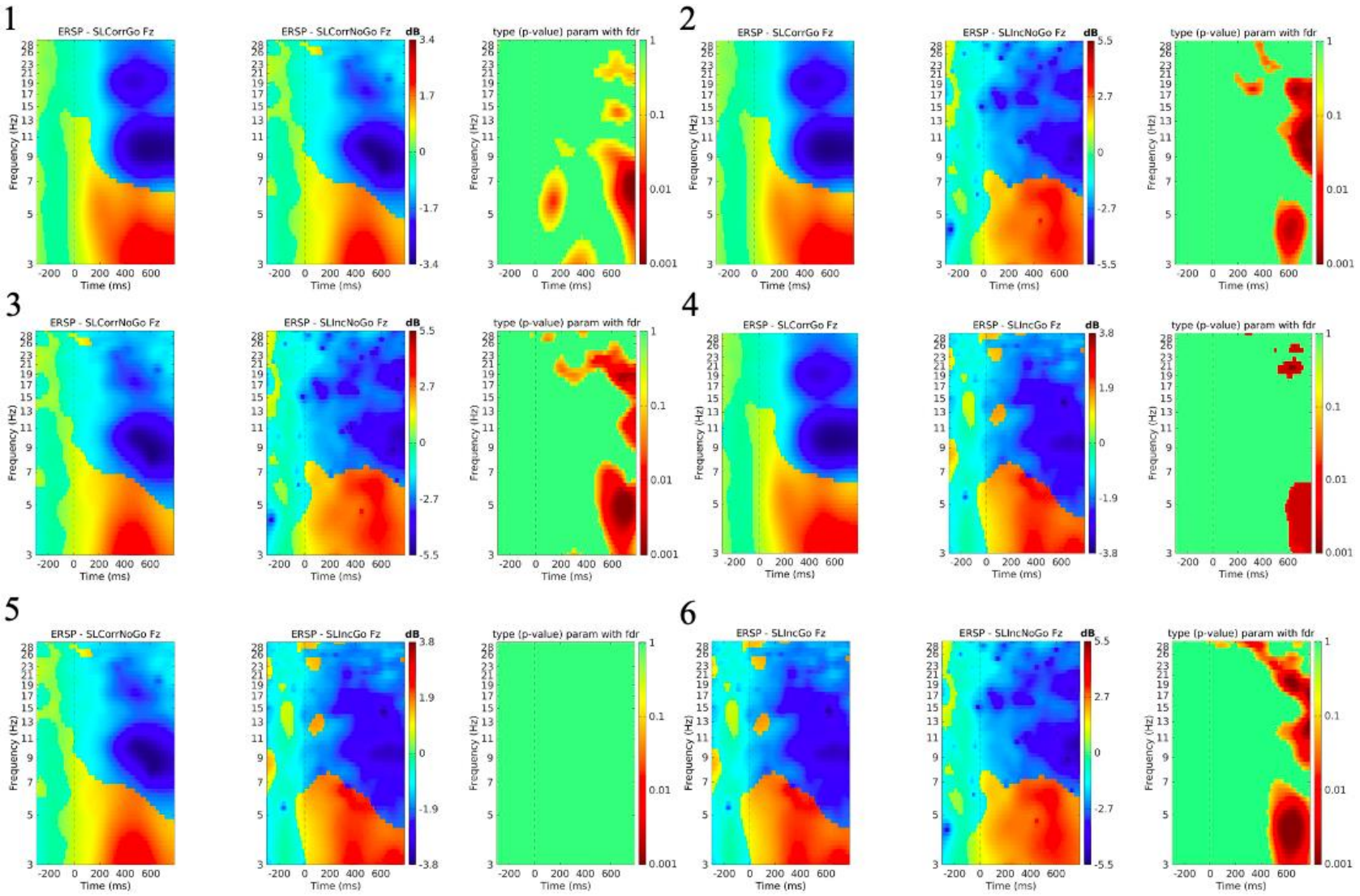
As a follow up to the RM ANOVA results presented in Figure 7, we conducted a series of *t*-tests to evaluate pairwise ERSP differences across all four Go/NoGo conditions on alpha and theta frequency bands. The results of these *t*-tests are described below and can be seen in Figure 8.

We observed significant differences in ERSP (p <.05; contrast 1) on the theta frequency band from 20 to 215 ms between correct Go and correct NoGo conditions, such that correct NoGo responses showed lower ERSP. Significant differences in ERSP for correct Go vs. incorrect NoGo responses (p <.01; contrast 2) were observed on the theta frequency band from 450 to 765 ms, and alpha from 590 to 800 ms, with incorrect NoGo responses having greater theta ERSP, and lower alpha ERSP (Figure 14). Significant differences in ERSP for correct NoGo vs. incorrect NoGo responses (p <.01; contrast 3) were observed on the theta frequency

band from 500 to 800 ms, and alpha from 750 to 800 ms, with correct NoGo responses having lower alpha ERSP. When comparing correct Go and incorrect Go responses, significant differences in theta ERSP were observed from 600 to 800 ms (p <.05; contrast 4), with incorrect Go responses showing lower ERSP. No significant differences were observed between correct NoGo and incorrect Go responses (p >.05; contrast 5). Significant differences in ERSP for incorrect Go vs. incorrect NoGo responses (p <.05; contrast 6) were observed on the theta frequency band from 440 to 800 ms, and alpha from 560 to 800 ms, with incorrect NoGo responses having lower theta ERSP, and greater alpha ERSP.

Figure 8.

Stimulus-locked ERSP Comparisons at Fz Across Go/NoGo Conditions



Linear Regressions.

Similar to the response-locked data, we conducted a series of linear regressions using the stimulus-locked N2 waveform to investigate if patterns of ERSP activity could predict dimensions of ACE exposure. Prior to conducting this series of regressions, we investigated the collinearity of ERSP and ITC predictor variables, ensuring variables did not exceed VIF thresholds (Appendix A; Table A3). Upon confirmation of non-concerning collinearity, we conducted our series of regression models. Those reaching statistical significance are shown in the tables below (Tables 3–5), the remainder can be found in Appendix C.

Table 3.

Stimulus-Locked N2 Alpha ERSP Across Trial Conditions and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	1.20	1.02	.29	1.18	.24
Correct NoGo ERSP	-2.12	.86	-.55	-2.46	**.02**
Error Go ERSP	.52	.48	.18	1.07	.29
Error NoGo ERSP	-.05	.37	-.03	-.15	.88

Table 4.

Stimulus-Locked N2 Alpha ERSP Across Trial Conditions and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	.14	.33	.10	.44	.66
Correct NoGo ERSP	-.57	.28	-.40	-2.03	**.048**
Error Go ERSP	.19	.16	.18	1.18	.24
Error NoGo ERSP	.22	.12	.27	1.84	.07

Table 5.

Stimulus-Locked N2 Alpha ERSP Across Trial Conditions and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	.98	.65	.37	1.51	.14
Correct NoGo ERSP	-1.23	.55	-.50	-2.22	**.03**
Error Go ERSP	.17	.31	.09	.54	.59
Error NoGo ERSP	-.25	.24	-.18	-1.05	.30

Table 6.

Stimulus-Locked N2 Alpha ERSP Across Trial Conditions and Subjective ACE Rating

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	7.26	9.10	.19	.80	.43
Correct NoGo ERSP	-16.05	7.71	-.45	-2.08	**.04**
Error Go ERSP	4.80	4.28	.19	1.12	.27
Error NoGo ERSP	2.00	3.10	.10	.65	.52

We conducted a similar series of regressions to evaluate N2 theta ERSP and dimensions of ACEs. We found that N2 theta ERSP for correct NoGo trials was significantly related to abuse, such that as N2 theta ERSP decreased, abuses increased. The result of this linear regression is reported below in Table 7. All other regressions for theta ERSP did not reach significance and are included in Appendix C.

Table 7.

Stimulus-Locked N2 Theta ERSP Across Trial Conditions and Abuse

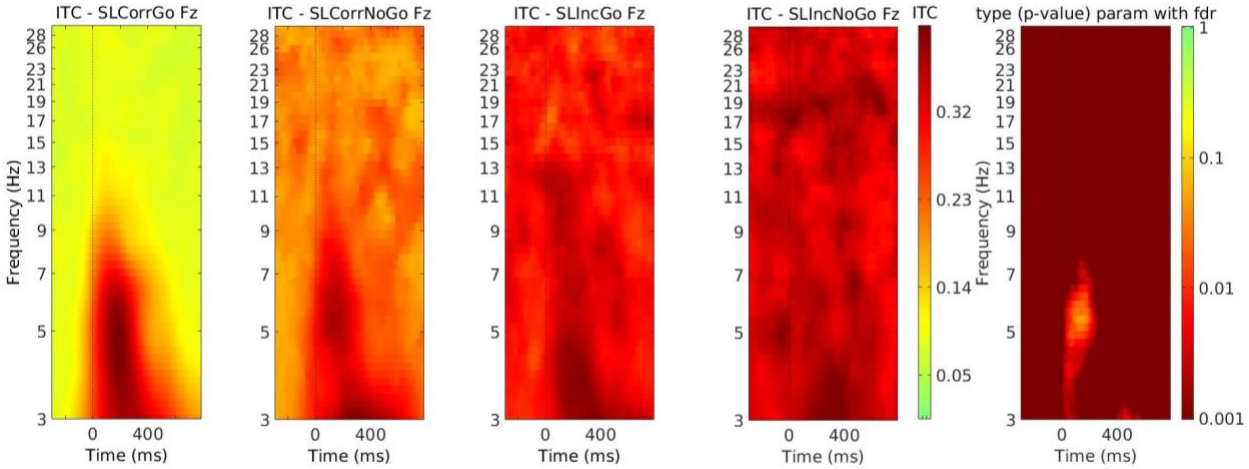
	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	-.10	.33	-.04	-.30	.77
Correct NoGo ERSP	-.35	.14	-.40	-2.42	**.02**
Error Go ERSP	.11	.11	.14	1.04	.30
Error NoGo ERSP	.08	.10	.11	.75	.46

2.8 Stimulus-Locked ITC

To further explore the relationships among ITC and dimensions of ACEs, we extracted stimulus-locked ITC activity for correct Go, correct NoGo, incorrect Go, and incorrect NoGo responses at Fz. Across the four conditions, we observed significant ITC differences in theta and alpha throughout nearly the entire -300 to 800 ms epoch. Interestingly, alpha ITC for correct Go responses was significantly reduced when compared to correct NoGo, incorrect Go, and incorrect NoGo responses across all frequency bands. However, when isolating the N2 waveform on the theta frequency band, ITC for correct Go responses is significantly greater than the other three conditions (see Figure 9).

Figure 9.

Stimulus-Locked ITC at Fz as a Function of Condition



Note. The right-most panel shows the results of the omnibus 4-level 1-way ANOVA F-test

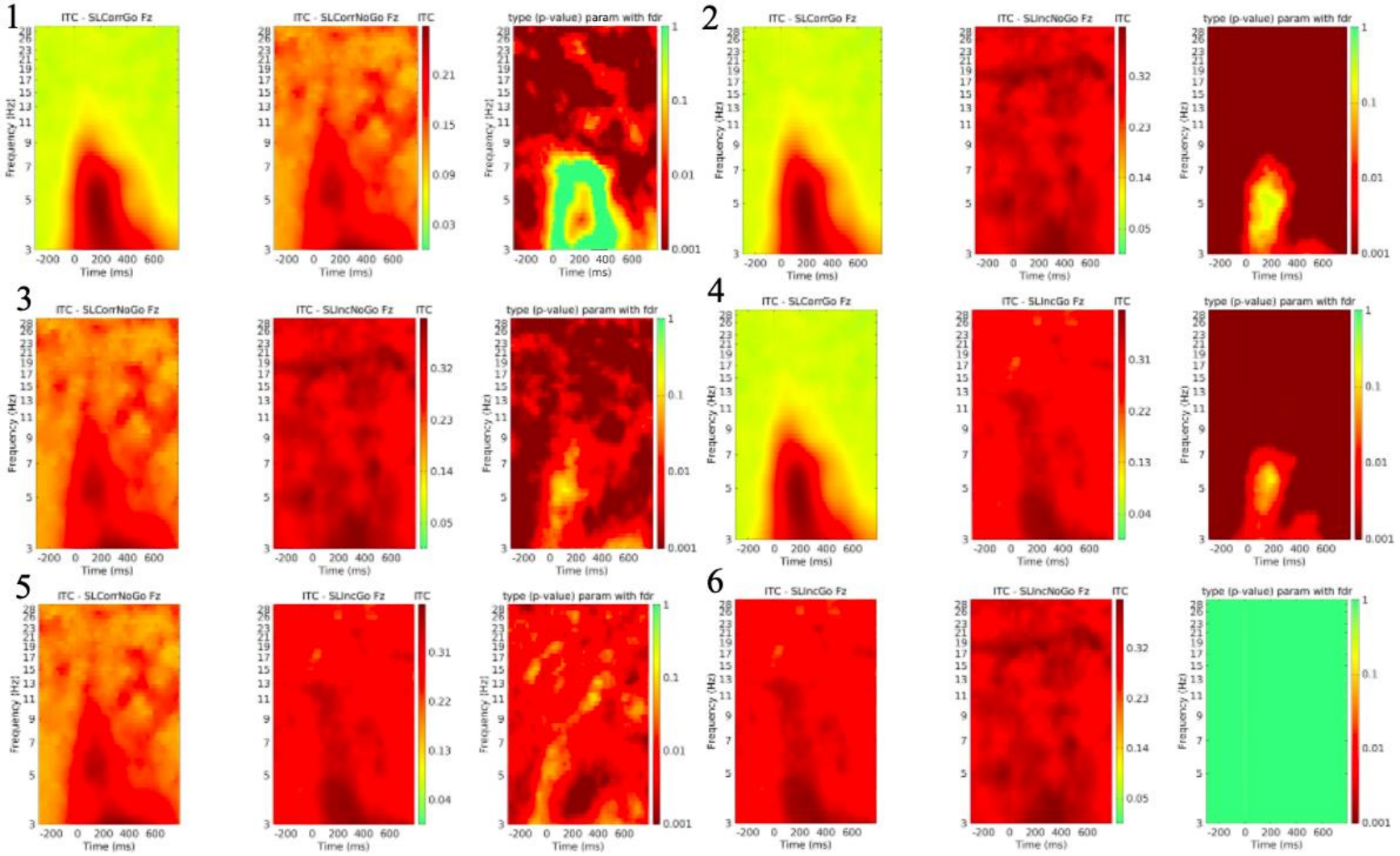
As a follow up to the RM ANOVA results presented in Figure 9, we conducted a series of *t*-tests to evaluate pairwise ITC differences across all four Go/NoGo conditions on alpha and theta frequency bands. Significant differences were observed in all ITC analyses; thus, only significant N2 differences on alpha and theta frequency bands are described.

We observed significant differences in ITC on alpha and theta frequency bands for correct Go responses compared to correct NoGo responses ($p < .01$; contrast 1), incorrect NoGo responses ($p < .01$; contrast 2), and incorrect Go responses ($p < .01$; contrast 4). In all of these relationships, correct Go trials showed greater ITC during the N2 waveform on alpha and theta frequency bands. When comparing incorrect NoGo trials to correct NoGo trials, significant differences were observed on the alpha frequency band, with incorrect NoGo trials showing greater ITC ($p < .01$; contrast 3). Between correct NoGo and incorrect Go trials, incorrect Go trials had significantly greater N2 ITC on the alpha and theta frequency bands ($p < .01$; contrast

5). Lastly, no significant differences in N2 ITC between incorrect Go and incorrect NoGo trials ($p > .05$; contrast 6). See Figure 10 for the full results of these *t*-tests.

Figure 10.

Stimulus-locked ITC Comparisons at Fz Across Go/NoGo Conditions



Linear Regressions.

Upon confirmation of ITC differences across conditions, we conducted a series of multiple regressions to assess whether N2 phase consistency could predict exposure to ACEs. Three relationships emerged as significant in this series of regressions. The first significant relationship was observed on the alpha frequency band; ITC for incorrect responses to Go trials were significantly related to abuse, such that as N2 alpha ITC decreased, the number of abuses increased (Table 8). The second significant relationship between ITC and ACEs emerged on the theta frequency band with N2 ITC for correct Go responses significantly related to abuse, indicating that as theta N2 ITC increased, so did the number of abuses (Table 9). Lastly, ITC for correct NoGo responses was significantly related to household dysfunction, such that as theta N2 ITC decreased, household dysfunction scores increased (Table 10).

Table 8.

Stimulus-Locked N2 Alpha ITC Across Trial Conditions and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	20.01	13.38	.23	1.50	.14
Correct NoGo ITC	4.48	2.51	.22	1.78	.08
Error Go ITC	-2.67	1.34	-.25	-2.00	**.049**
Error NoGo ITC	.75	1.56	.08	.48	.63

Table 9.

Stimulus-Locked N2 Theta ITC Across Trial Conditions and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	23.55	11.54	.28	2.04	**.045**
Correct NoGo ITC	1.58	2.90	.08	.54	.59
Error Go ITC	-1.93	1.34	-.21	-1.44	.16
Error NoGo ITC	-2.31	1.47	-.23	-1.57	.12

Table 10.

Stimulus-Locked N2 Theta ITC Across Trial Conditions and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	13.71	10.71	.19	1.28	.21
Correct NoGo ITC	-6.11	2.70	-.37	-2.27	**.03**
Error Go ITC	-1.13	1.24	-.14	-.91	.37
Error NoGo ITC	-2.72	1.36	-.31	-1.99	.05

These regression models show the associations among alpha and theta ERSP and ITC for the stimulus-locked N2 waveform across task conditions and our five ACE dimensions.

Chapter 3: Auditory Selective Attention Paradigm

Method

Participant information, study procedure, ACE questionnaire, EEG recording, and pre/post-processing were identical to that previously described; please refer to chapter two for the full details of these processes. The primary difference is that the selective attention task was counterbalanced by beginning with either attended left or right ear. For this paradigm, we determined that the most negative stimulus-locked N1 waveform fell between 69.07 to 131.13 ms. This maximal N1 timing N1 was used to evaluate ERSP and ITC differences across conditions, as well as for regression models evaluating how alpha and theta ITC and ERSP may predict dimensions of ACEs.

3.0 Selective Attention Paradigm

The auditory selective attention task, as described in Lackner et al. (2013), uses two distinct auditory stimuli - a 1000 Hz (88% probability, nontarget) and a 2000 Hz (12% probability, target) 200 ms tones presented in both ears with Etymotic ear inserts (Etymotic Research Inc.). An initial practice block where participants were presented with each of the sounds was used to ensure participants could accurately distinguish the four sounds from one another. Participants were instructed to attend to one ear at a time and press the ‘green’ button on a Cedrus response pad when the target tone was presented in their attended ear. As in Lackner et al. (2013), eight-hundred total sounds were presented, with forty-eight 2000 Hz tones presented to the attended ear (attended target), forty-eight 2000 Hz tones presented to the unattended ear (unattended target), three-hundred and fifty-two 1000 Hz tones in the attended ear (attended nontarget), and three-hundred and fifty-two 1000 Hz tones in the unattended ear (unattended nontarget). Upon completion of each 200-block section, participants were given a 20s break and instructed to attend to target 2000 Hz tones in the other ear for the next trial block.

Results

3.1 Behavioural Data

To determine if we replicated often-reported behavioural differences in the selective attention task, we examined response times and accuracy (Table 11). Those with accuracy or response time data that were greater than two and a half standard deviations from the mean were excluded from the behavioural analyses. For the selective attention task, we conducted ERSP and ITC analyses with stimulus-locked data only, as we are evaluating early stages of cognitive processing.

Table 11.

Auditory Selective Attention Task Accuracy and Response Time by Condition

	Accuracy (SD)	RT (SD)
Attended Nontarget Errors	.93 (.14)	351.52 (90.70)
Attended Target Correct	.97 (.03)	409.29 (68.22)
Unattended Nontarget Errors	.95 (.14)	396.72 (139.55)
Unattended Target Errors	.96 (.07)	424.35 (65.53)

Note. Response Time and Standard Deviation (in parentheses) are reported in milliseconds.

We ran two repeated measures ANOVAs to examine the factors of condition (attended targets, attended nontargets, unattended targets, and unattended nontargets) and measure (accuracy and response time). There were significant differences in accuracy, $F(3, 55) = 4.91$, $p < .01$, as well as significant differences in response time, $F(3, 24) = 3.73$, $p = .03$. It is worth noting that the df is significantly lower for response times due to unattended targets, unattended nontargets, and attended nontargets not requiring a response. As follow up we conducted a series of paired samples *t*-tests to investigate pairwise differences. Significant differences in accuracy were observed between attended target and attended nontarget trials $t(55) = 2.54$; $p = .01$) and

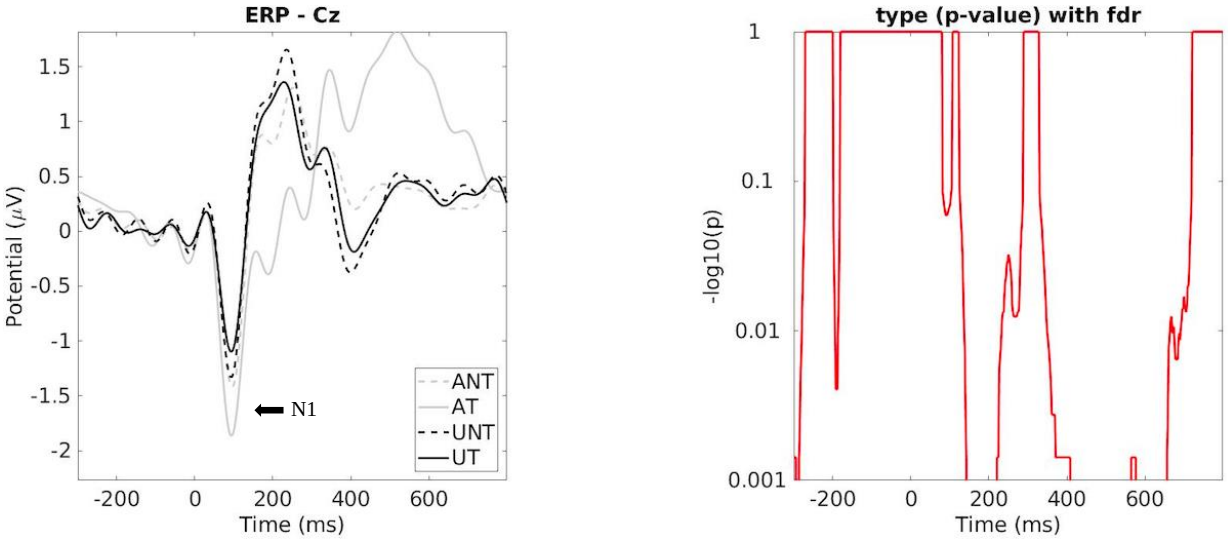
between attended target and unattended target trials $t(55) = 2.55$; $p = .01$). Significant differences in reaction times between attended target and attended nontarget trials $t(55) = 3.21$; $p < .01$) and attended target and unattended target trials $t(55) = -2.20$; $p = .03$) also emerged.

3.2 ERP Analysis

We then explored if there are amplitude differences between conditions (see Figure 11). The figure below highlights these ERP differences by condition.

Figure 11.

ERP Overlays at Cz as a Function of Auditory Selective Attention Condition



Note. ANT = Attended Nontarget; AT = Attended Target; UNT = Unattended Nontarget; UT = Unattended Target.

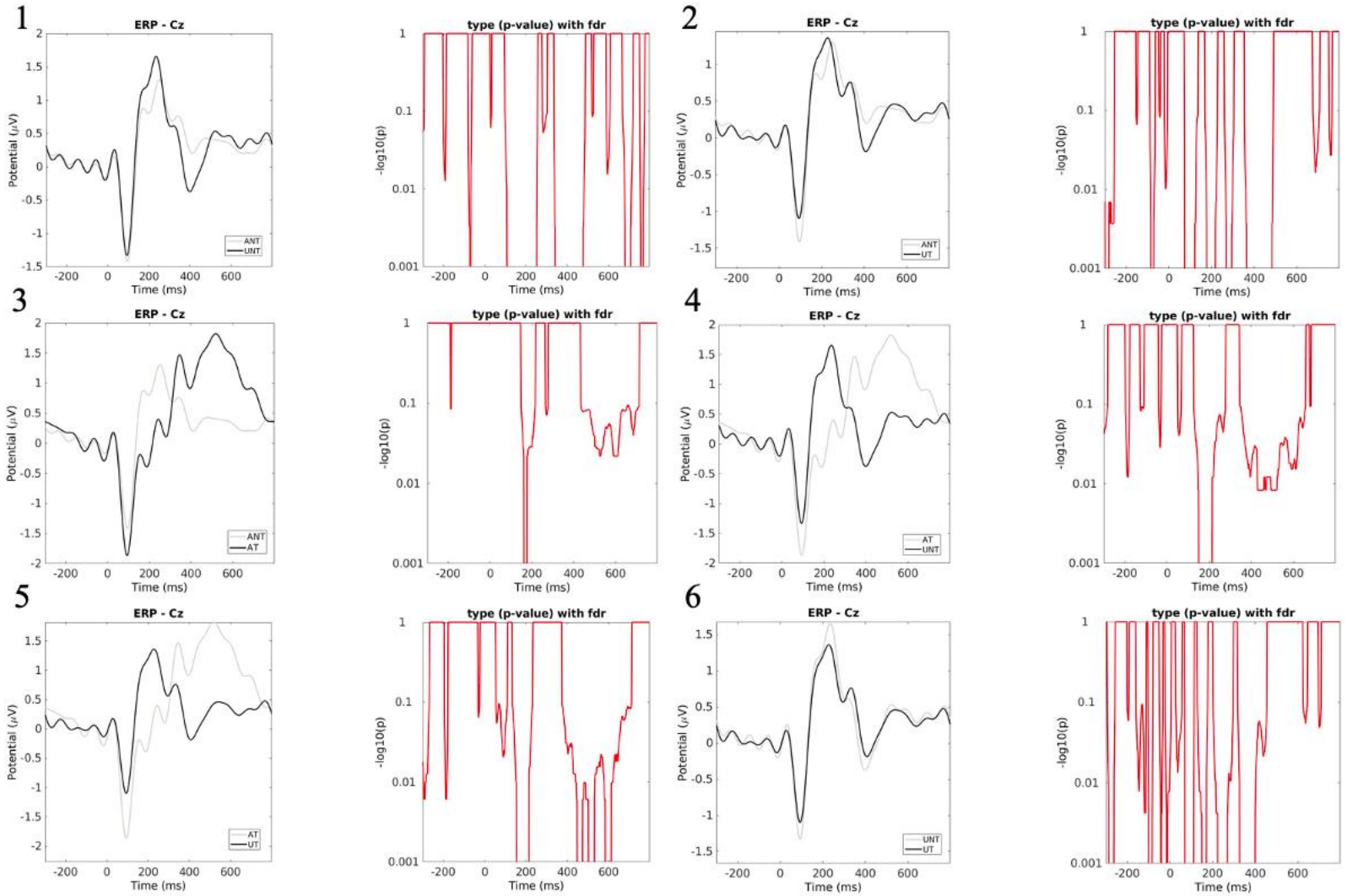
As seen in Figure 11, there are significant differences (FDR corrected, $p < .05$) in ERP amplitude across conditions, namely from 160 to 290 ms, and 375 to 760 ms post-stimulus. These findings warranted further investigation; thus, we conducted a series of paired *t*-tests with all possible condition permutations, these are described below.

Significant differences in N1 amplitude were observed between ANT and UNT ($p < .01$; contrast 1), with ANT N1 being more negative than UNT. Significant differences in N1 amplitude were observed between ANT and UT ($p < .01$; contrast 2), with ANT N1 being more

negative than UNT. No significant differences in N1 amplitude were observed between ANT and AT ($p > .01$; contrast 3). However, the ERP figure shows the AT N1 amplitude as more negative than the ANT. No significant differences in N1 amplitude were observed between AT and UNT ($p > .05$; contrast 4). However, the ERP figure shows the AT N1 amplitude as more negative than the UNT, with results approaching statistical significance. Significant differences in N1 amplitude were observed between AT and UT ($p < .05$; contrast 5), with AT N1 being more negative than UT. Significant differences in N1 amplitude were observed between UNT and UT ($p < .01$; contrast 6), with UNT N1 being more negative than UT. All *t*-test results can be seen in Figure 12.

Figure 12.

Stimulus-locked Condition Comparisons at Cz for the Selective Attention Paradigm with FDR Significance Values



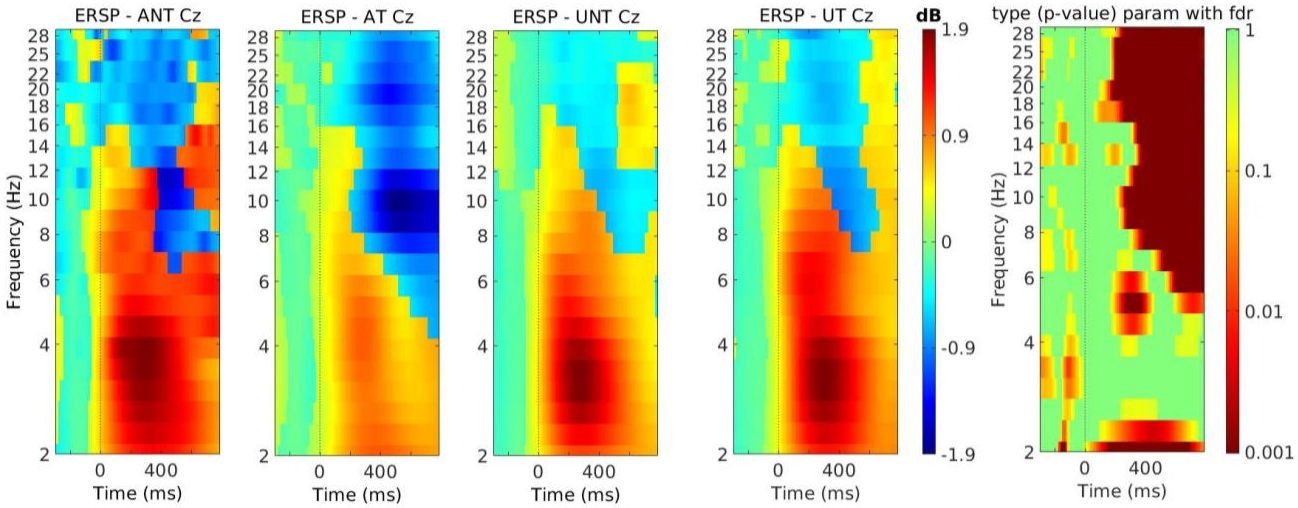
Note. Peak N1 amplitude can be viewed at 95 ms.

3.3 Stimulus-Locked ERSP

We then conducted ERSP and ITC analyses with stimulus-locked data from 1–30 Hz but focused on differences in alpha and theta during the maximal amplitude of the N1, between 69.07 to 131.13 ms. We calculated stimulus-locked ERSP and ITC data at Cz during this time window. Differences in ERSP across selective attention conditions can be seen below in Figure 13.

Figure 13.

ERSP at Cz as a Function of Condition



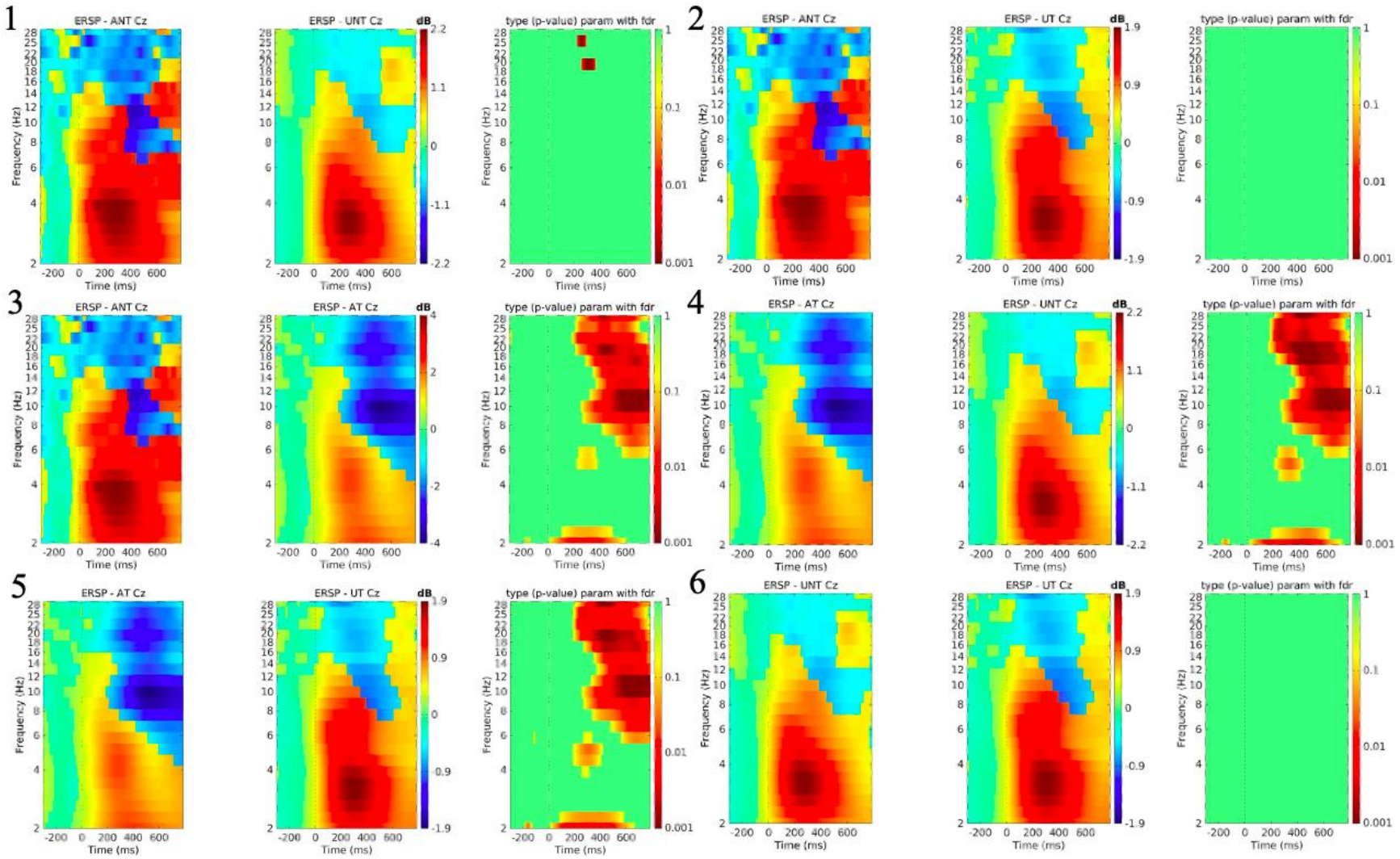
Note. The right-most panel shows the results of the omnibus 4-level 1-way ANOVA F-test

In order to broadly evaluate condition differences in ERSP, we ran a series of paired *t*-tests in EEGLAB. With our four response conditions, we had six possible pairwise comparisons. We found significant differences in ERSP for AT vs. UNT, AT vs. ANT, and AT vs. UT. Differences in ERSP for ANT vs. UNT, ANT vs. UT, and UNT vs. UT did not reach statistical significance. These results are described below and presented in Figure 14.

We observed significant AT vs. ANT ERSP differences in theta from 250 to 380 ms, such that greater theta ERSP was observed during attended targets. Further, significant differences in alpha ERSP were observed from 240 to 800 ms, such that lower alpha ERSP was observed during responses to the attended target (see Figure 14; contrast 3). We observed significant differences in ERSP between AT and UNT conditions. Specifically, we saw differences in theta ERSP from 220 to 400 ms, such that greater theta ERSP was observed during attended targets. Further, significant differences in alpha ERSP were observed from 250 to 800 ms, such that lower alpha ERSP was observed during responses to the attended target (see Figure 14; contrast 4). We also observed significant differences in ERSP between attended target and unattended target conditions. Specifically, we saw differences in theta ERSP from 200 to 410 ms, such that greater theta ERSP was observed during attended targets. Further, significant differences in alpha ERSP were observed from 230 to 800 ms, such that lower alpha ERSP was observed during responses to the attended target (see Figure 14; contrast 5). The remaining comparisons ANT vs. UNT (Figure 14; contrast 1), ANT vs. UT (Figure 14; contrast 2), and UNT vs. UT (Figure 14; contrast 6) did not reach statistical significance on our target frequency bands.

Figure 14.

Stimulus-locked ERSP Comparisons at Cz Across Selective Attention Conditions



Linear Regressions.

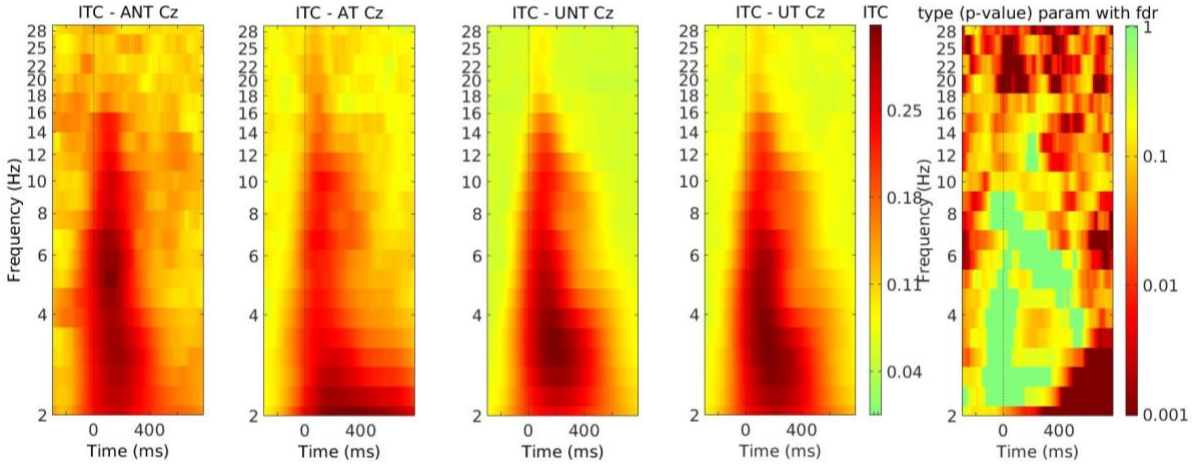
Prior to conducting this series of regressions, we investigated the collinearity of ERSP and ITC predictor variables, ensuring variables did not exceed VIF thresholds (Appendix A; Table A5). Upon confirmation of non-concerning collinearity, multiple linear regression analyses were conducted to explore the relationships of stimulus-locked N1 alpha and theta ERSP and number of ACEs, subjective rating of ACEs, abuse, household dysfunction, and other adversities. None of these regression models emerged as significant across both alpha and theta frequency bands (see Appendix D for results).

3.4 Stimulus-Locked ITC

To extend our time-frequency analyses, we explored the relationships among ITC and dimensions of ACEs by extracting stimulus-locked ITC activity for attended non-target (ANT), attended target (AT), unattended non-target (UNT), and unattended target (UT) responses at Cz. Across the four conditions, we observed significant ITC differences intermittently on theta and alpha throughout the -300 to 800 ms epoch (see Figure 15).

Figure 15.

ITC at Cz as a Function of Condition



Note. The right-most panel shows the results of the omnibus 4-level 1-way ANOVA F-test.

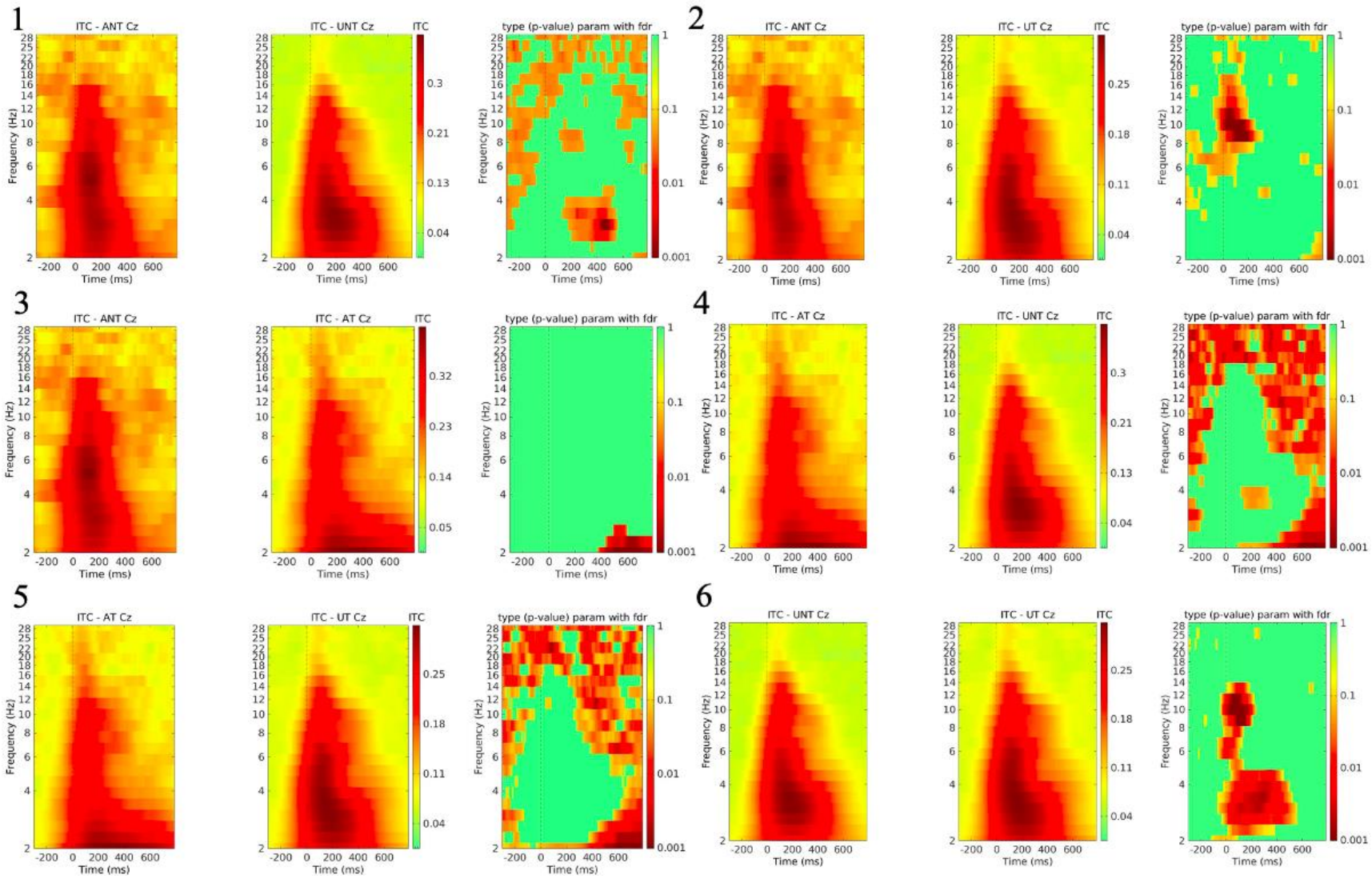
To follow up on the observed differences in ITC across conditions, we ran a series of paired *t*-tests in EEGLAB identical to those run for ERSP to assess differences in ITC in paired trial conditions. Paired *t*-tests reaching are described below.

Significant differences in ITC emerged between ANT and UNT conditions. Specifically, we saw differences in theta ITC from 170 to 580 ms, such that greater theta ITC was observed during unattended nontargets. Further, significant differences in alpha ITC were observed from 70 to 240 ms, such that lower alpha ITC was observed during responses to the unattended nontargets (see Figure 16; contrast 1). ITC differed between ANT and UT conditions. Specifically, greater theta ITC was observed during attended nontargets than unattended targets from -300 to 230 ms. Further, significant differences in alpha ITC were observed from -40 to 195 ms, such that lower alpha ITC was observed during unattended targets (see Figure 16; contrast 2). No significant differences were observed between ANT and AT (see Figure 16; contrast 3). We observed significant differences in ITC between AT and UNT conditions. Specifically, we saw differences in theta ITC from 85 to 380 ms, such that greater theta ITC was observed during unattended nontargets. Further, significant differences in alpha ITC were observed from 375 to 800 ms, such that lower alpha ITC was observed during responses to the unattended nontargets (see Figure 16; contrast 4). We observed significant differences in ITC between AT and UT conditions. Specifically, we saw differences in theta ITC from 550 to 800 ms, such that greater theta ITC was observed during attended targets. Further, significant differences in alpha ITC were observed from 305 to 800 ms, such that lower alpha ITC was observed during responses to the unattended targets (see Figure 16; contrast 5). Lastly, we observed significant differences in ITC between UNT and UT conditions. Specifically, we saw differences in theta ITC from -65 to 450 ms, such that greater theta ITC was observed during unattended nontargets. Further,

significant differences in alpha ITC were observed from -30 to 270 ms, such that lower alpha ITC was observed during responses to unattended targets (Figure 16; contrast 6). These results can be observed in Figure 16 below.

Figure 16.

Stimulus-locked ITC Comparisons at Cz Across Selective Attention Conditions



Linear Regressions.

We conducted a series of multiple regressions to assess whether phase consistency of the stimulus-locked N1 waveform could predict exposure to ACEs. N1 alpha ITC to the unattended nontarget stimuli were significantly related to other adversities, such that as N1 alpha ITC increased exposure to other adversities increased as well (Table 12). No additional regression models reached statistical significance (tables included in the Appendix D).

Table 12.

Stimulus-Locked N1 Alpha ITC Across Trial Conditions and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ITC	-1.44	3.62	-.05	-.40	.69
Attended Target ITC	3.16	6.12	.07	.52	.61
Unattended Nontarget ITC	29.83	12.53	.33	2.38	**.02**
Unattended Target ITC	-12.01	10.99	-.15	-1.09	.28

Chapter 4: Discussion

We investigated if patterns of ERP activity are related to childhood adversity by using a response inhibition auditory Go/NoGo paradigm and an auditory selective attention task. Specifically, we investigated whether ERSP and ITC values for response-locked (ERN) and stimulus-locked (N1 and N2) waveforms across alpha and theta frequency bands could predict the number of ACEs an individual has been exposed to, their subjective rating of those ACEs, as well as if they had experienced abuse, household dysfunction, or other adversities. Our findings suggest that alpha and theta ERSP and ITC activity for the N1 and N2 waveforms, particularly during successful response inhibition trials, left neural signatures that could significantly predict exposure to various dimensions of ACEs, with moderate effect sizes. Dimensions of selective auditory attention yield patterns of neural activation that are generally less predictive of childhood adversity.

4.0 Response-Locked ERN ERSP & ITC

Our first set of analyses examined the ERN, a frontally localized ERP that is generated by participant errors during a response inhibition task (Gehring et al., 1993). We found that there were significant differences in ERN amplitude as a function of condition, such that ERN amplitudes were more negative during erroneous than correct responses. This is a commonly observed effect, as the error-monitoring system does in fact generate a negative deflection following a correct response (CRN); however, this response is less negative than the ERN that reflects errors. Additionally, existing literature has consistently demonstrated more negative ERN amplitudes as a function of ACEs (Lackner et al., 2018; Letkiewicz et al., 2023). This suggests that ACEs may exacerbate neural sensitivity to errors during tasks requiring response inhibition. However, some of these differences may be more pronounced on higher frequency bands that are more dominant during cognitively demanding task requiring response-inhibition

(Wang et al., 2020). Here, we examined these purported deficits on alpha and theta frequency bands that contribute to the maintenance of appropriate levels of attentional engagement during tasks requiring cognitive control.

Following a preliminary analysis to confirm the commonly observed ERN – CRN difference, we conducted multiple regression analyses to investigate how ERN ERSP and ITC may differentially predict various dimensions of ACE exposure. Previous literature has demonstrated Notably, alpha and theta ERSP and ITC during the ERN were not significantly predictive of dimensions of ACE exposure. These findings could suggest a resilience of the alpha and theta time-frequency error-monitoring components to ACEs in our sample, which could be attributed to the importance of error monitoring and emotion regulation in the maintenance of cognitive stability across various contexts, leading to a nil effect (Holroyd & Coles, 2002). This may suggest that the effects observed in Lackner et al. (2018) were not due to alterations to alpha and theta activity. Instead, these differences may be driven by other frequency bands. For instance, beta activity significantly effects the negativity of the ERN (Wang et al., 2020). Thus, individuals who experience early adversity may develop compensatory neural mechanisms to retain functionality in neurological structures like the ACC, as well as on other frequency bands, despite encountering stressful events.

As the ERN is primarily generated in the ACC, a structure essential for adaptive behaviour and cognitive control, these findings may suggest variability in the impacts of ACEs in this region (Falkenstein et al., 2000; Gehring & Willoughby, 2002). Further, the stability in ERN ERSP and ITC despite exposure to ACEs provides evidence to support the adaptive nature of these structures. Despite the ACC containing an abundance of glucocorticoid receptors, it appears that deficits in the alpha and theta time-frequency domain are not readily observed in our

sample. However, this may suggest that commonly observed deficits may be the result of higher frequency neurological signals, such as beta or gamma waves.

These findings align with research focused the ability of neural networks to change both functionally, and structurally, in response to experiences and injuries (Bernhardi et al., 2017). Naffaa (2024), investigated the role of the ACC in neurogenesis and neural plasticity and found a relationship between postnatal and adult neurogenesis in the ACC in conditions related to memory and cognitive behaviour. Thus, the ACC may utilize these mechanisms to preserve some aspects of cognitive control in these regions despite the other consequences of ACEs. For instance, Bluschke et al. (2022) found that response inhibition and cognitive control could be improved using theta neurofeedback protocols in individuals with ADHD. Despite ACEs exerting negative effects on the ACC via glucocorticoids, we found no evidence that alpha and theta rhythms are disrupted during error-monitoring processes associated with the ERN.

4.1 Stimulus-Locked N2 ERSP

Our next series of analyses involved the stimulus-locked N2 ERP that also plays a role in conflict monitoring and cognitive control. The same Go/NoGo paradigm was used to elicit this waveform, with the key difference being the epoch windows were time locked to the stimulus presentation, rather than an overt participant response (Folstein & Van Petten, 2008). We used this stimulus-locked waveform to probe earlier phases of the information processing stream that represent response selection rather than the overt response. Similar to the ERN, we conducted a series of regression models to explore if alpha and theta ERSP and ITC during the timing of the N2 were related to five dimensions of ACEs. We observed patterns of alpha and theta N2 ERSP and ITC for correct NoGo trials that were significantly predictive of exposure to early childhood adversity.

We found that alpha N2 ERSP during correct NoGo trials (i.e., successful inhibitions) was significantly related to the total number of ACEs, abuse, other adversities, and subjective rating of ACEs in our study sample. Specifically, reduced alpha power during these trials was indicative of a higher number of ACEs, more frequent abuses, other adversities experienced, and higher subjective rating of these ACEs. Klimesch (2012) highlights the role alpha oscillations play in modulating cortical excitability and facilitating a multitude of attentional processes, including inhibitory control. This is the key aspect of how the N2 differs from the ERN despite both being involved in conflict monitoring. The ERN is primarily involved in the detection of errors and is the immediate neurological response to mistakes; whereas the N2 represents the inhibition of inappropriate responses and occurs earlier in the information processing stream (van Noordt & Segalowitz, 2012). During cognitively demanding tasks where response inhibition is required, alpha suppression is also commonly observed (Benedek et al., 2011), our results suggest that this suppression may be more pronounced as a function of ACEs. The negative relationship observed between N2 alpha ERSP and these ACE dimensions implies that individuals who have experienced a greater number of ACEs may exhibit altered neural responses during tasks that require inhibitory control.

These findings align with previous neurodevelopmental literature exploring global alterations in neurophysiologic structure and function, while affecting dimensions of cognition via prolonged glucocorticoid exposure (Chapman et al., 2007; Koss & Gunnar, 2017; Teicher et al., 2016). Further, patterns of reduced alpha activity as a product of ACEs may point to an early biomarker for those at risk of experiencing a variety of psychological issues associated with ACEs, such as anxiety and depression (Knyazev, 2007). Here, we provide further evidence of

how neurophysiology may be altered as a product of the ACE dimensions that a child is exposed to.

Notably, the subjective rating of various ACE dimensions played an integral role in the resulting neural signatures. This expands on previous research that has primarily focused on dose-dependent relationships between ACEs and ERPs rather than looking at the subjective impacts of the ACEs. These findings suggest that individual differences in the perceptions and interpretations of ACEs can significantly affect the observed neurophysiological differences. Subjective experiences of stress and early adversity can vary widely among individuals, such that two individuals who may have experienced identical adverse events at the same point in time may be impacted in an entirely different way (Giano et al., 2020; Jones et al., 2022). Thus, this supports our overarching theme in exploring the individual differences in ERSP by considering personal contexts, and how these neural signatures may relate to the impacts of early adversity.

Expanding on these findings, negative associations between subjective ACE rating and alpha ERSP suggests that those who perceive ACEs to be highly stressful or traumatic may exhibit more significant neural dysregulation on the alpha frequency band. Previous literature has demonstrated that subjective stress is an integral component in the development of stress-induced cognitive and neurophysiologic deficits (McEwen, 2012). Not only do these results point to potential biomarkers of early adversity that could lead to future psychopathology, but they also highlight the importance of incorporating subjective measures of stress and adversity. By doing so, we can elucidate how individual differences can impact neural and cognitive outcomes.

After evaluating the relationships among alpha N2 ERSP and dimensions of ACEs, we conducted a similar series of regressions exploring theta N2 ERSP and the same five ACE dimensions. Notably, we observed a significant negative relationship among theta N2 ERSP for

correct NoGo responses and abuse. Individuals exposed to higher levels of abuse showed a decrease in theta ERSP during successful response inhibition trials. Again, this finding suggests that childhood abuse can have a significant negative effect on neural mechanisms that underly cognitive control. Cavanagh and Frank (2014) explored the role of frontal theta oscillations in coordinating fluid communication among neural networks while simultaneously facilitating cognitive control. This synchronized firing of neurons from the PFC is thought to represent conscious awareness of the need for exercising cognitive control (Cavanagh & Frank, 2014). This is supported by our finding of lower theta ERSP during correct NoGo responses related to abuses, suggesting a dysregulation of this inherent neurophysiological mechanism.

Further, the generation of theta oscillations in the PFC have also been heavily implicated in emotion regulation and executive function (Cavanagh & Shackman, 2015). The inability for the PFC to adequately generate theta ERSP during the correct NoGo condition suggests that the regions of the PFC responsible for conflict detection and cognitive control, such as the VLPFC (Aron et al., 2004) and the dorsolateral prefrontal cortex (DLPFC; Macdonald et al., 2000), may be particularly susceptible to the effects of early abuse (Carpenter et al., 2010). For instance, Gold et al. (2004), found that alterations in grey matter occurs in the DLPFC as a product of early abuse and may result in deficits in effortful emotion regulation. This could include the early stages of cognitive processing when selecting an appropriate response and lead to reductions in N2 amplitude while the ERN remains intact.

Expanding on this, Carpenter et al. (2010) found that women who experienced childhood physical abuse exhibited significantly blunted cortisol responses during the Trier Social Stress Test when compared to those who had not. These findings highlight the differential effects of early childhood abuse on the neuroendocrine system, which can lead to functional differences in

these neural regions, as we observed in the present study. This blunted cortisol response could represent an innate neuroprotective mechanism to prevent further cortisol induced damage to these brain regions. Altered development in these regions can manifest as increased impulsivity, difficulties regulating emotions, and deficits in global cognitive control (Pechtel & Pizzagalli, 2011).

4.2 Stimulus-Locked N2 ITC

Following the exploration of how ERSP across alpha and theta frequency bands relates to ACEs, we extended our analyses to evaluate phase consistency of these signals. Specifically, we explored whether the consistency of neurological responses, measured via ITC, to task conditions could differentially predict number of ACEs an individual was exposed to, levels of abuse, household dysfunction, other adversities, and the subjective rating of ACEs. Phase consistency of alpha N2 during incorrect Go trials was significantly related to childhood abuse, such that as alpha ITC scores decreased, abuses increased. ITC is commonly indexed alongside ERSP to evaluate the consistency of cognitive engagement and performance across trials (Klimesch, 2012). Interestingly, decreases in alpha N2 ITC during incorrect Go responses suggests an enhanced susceptibility of neural phase consistency to error responses, potentially due to the role of alpha waves in inhibitory control and attentional processes.

This observed lapse in phase consistency of alpha N2 ITC suggests a breakdown in the cognitive control mechanisms that monitor performance (i.e., an attentional lapse). An incorrect Go response suggests that participants unintentionally inhibited their response to a Go stimulus, thus reflected by disruptions in ITC that significantly predict increased levels of abuse. Not only are medial (Aron et al., 2004), and lateral (Aron et al., 2014) PFC regions implicated in these findings, but primary motor cortices are also engaged during response inhibition tasks

(Ridderinkohof et al., 2011). Not only must participants select an appropriate response, but they must synchronize these responses across regions to effectively respond to target stimuli (Burle et al., 2016). Our analyses investigated the early phases of decision making by time-locking and analyzing ITC to the presentation of a stimulus. Cumulatively, the disruptions in phase synchrony in frontal sites suggest that these mechanisms of cognitive control are affected at the early stages of information processing.

In exploring dimensions of theta ITC, we observed a significant positive relationship between theta N2 ITC during correct responses and number of ACEs, showing that as theta N2 ITC increased, abuses also increased. This observed relationship may be a function of an induced state of hypervigilance seen in individuals who have ACEs (Knyasev, 2007). This could be a direct result of the increased cognitive load associated growing up in an abusive environment, or experiencing instances of abuse (Cavanagh & Frank, 2014). The neural systems responsible for exercising cognitive control on the theta frequency band may enhance synchronization as a compensatory mechanism to deal with stressful environments (Cicchetti & Rogosch, 2012).

Lastly, a significant negative relationship was observed between N2 theta ITC for correct NoGo trials and household dysfunction. Dimensions of household dysfunction include living with family members that abuse illicit substances, experience mental illness, have suicidal thoughts and behaviours, divorce or parental separation, incarcerated family members, and intimate partner violence or domestic violence (Felitti et al., 1998). In line with nature (genetics) and nurture (environment) discourse, this suggests that the environment in which a child is raised plays a role in future cognitive function, measured via theta phase consistency during response selection. Individuals in our study sample with some, or a combination, of these experiences demonstrate progressive reductions in phase synchrony on the theta frequency band as the

number of these experiences increase. Specifically, these differences emerged during successful response inhibition trials (i.e., during correct NoGo trials).

As correct NoGo trials indicate a successfully inhibited response, (Shalgi et al., 2009), our findings highlight a disruption in this cognitive control mechanism as a product of household dysfunction. Studies suggest that chronic stress from childhood poverty impacts self-regulation and coping mechanisms (Evans & Kid, 2013), in addition to affecting the development of neural circuitry in the PFC that contributes to behavioural inhibition (Chen et al., 2022). These findings highlight the attention needed to ensure children grow up in safe household environment. By doing so, the risk of alterations to neural circuitry during critical developmental periods can be reduced (Baker et al., 2019).

4.3 Stimulus-Locked N1 ERSP

Next, we used an auditory selective attention task to evaluate the stimulus-locked N1 waveform on alpha and theta frequency bands. As with the other analyses, we explored whether particular neural signatures of the N1 ERP could predict dimensions of ACEs. N1 ERSP did not relate to the number of ACEs, abuses, household dysfunction, other adversities, and the subjective rating of these ACEs on either frequency band. The N1 indexes early sensory processing and attention allocation, and therefore, may not be as sensitive to the effects of ACEs as other ERP components. For example, we observed several significant relationships among the N2 ERSP and ITC and dimensions of ACEs. These findings align with previous literature suggesting that the N2 is more directly involved in conflict monitoring and cognitive control when compared to the N1 (Folstein & Van Petter, 2008, but see Lackner et al., 2013 for an exception). Alpha ERSP during the N1 may be more closely related to generalized neural processes that are less susceptible to ACEs. For example, alpha waves comprise the greatest

percentage of the EEG signal (Jensen & Mazaheri, 2010), thereby implicating the alpha frequency band across a multitude of tasks. This suggests that despite the reflection of alpha in gating inhibition, the robustness of alpha activity in early sensory processes may supersede the potential negative effects of childhood adversity. This could result in an absence of alpha N1 ERSP neural signatures that correspond to dimensions of ACEs in our study sample.

These results appear to be consistent across frequency bands, as theta N1 ERSP also left no neural signatures related to dimensions of childhood adversity. Similar to alpha activity, theta N1 ERSP may be less affected by ACEs as it represents early stages of sensory processing. To further explore if this is the case, it may be valuable to explore the associations among sensory gating, that occurs prior to the N1, using the P50 waveform. The P50 waveform represents the immediate response to sensory stimuli, and attention filtering following repeat stimuli, occurring 50 ms following stimulus presentation (Li et al., 2023). Li et al. (2023) found that the P50 waveform is predictive of depressive symptomology in those with schizophrenia. When considering the interconnectedness of ACEs and depression, as well as ACEs and risk behaviours associated with schizophrenia (e.g., marijuana use; van Zyl et al., 2023), the P50 could assist in providing a more complete picture of how the information processing stream may be altered by childhood adversity.

Beyond this, current research suggests that the impacts of ACEs on neurological function may be more pronounced during high order cognitive tasks involving error processing and cognitive control (McLaughlin et al., 2014; Pechtel & Pizzagalli, 2011). As a result of the broad ACE categories, alterations in neural oscillations may not be uniformly captured by alpha and theta ERSP during the early stages of sensory processing. Perhaps, individuals who are exercising enhanced cognitive control while focusing on task demands exhibit patterns of high

frequency neurological oscillations (i.e., beta and gamma) that govern early aspects of attentional processes. This points to the complexity and variability of how early sensory processing may be affected by ACEs; thus, suggesting the integration of higher frequency waveforms in future studies may be beneficial for examining these purported deficits, or lack thereof.

4.4 Stimulus-Locked N1 ITC

For the auditory selective attention task, we also evaluated the phase consistency of the N1 ERP on the alpha and theta frequency bands by calculating ITC. Despite no significant findings of theta ITC, there was a significant positive relationship between alpha N1 ITC during responses to the unattended nontarget and other adversities. Research suggests that alpha oscillations are involved with selective attention and are readily observed in the PFC while filtering out irrelevant stimuli and focusing on relevant information (Foxe & Snyder, 2011; Klimesch, 2012). Importantly, the alignment of these waveforms represents consistent information processing, and can be seen as a reflection of maintaining focused attention over time.

This suggests that early experiences of other adversities, such as losing a pet, bullying, and serious medical procedures or illnesses, may not impact the brain's ability to maintain phase consistency during less cognitively demanding attention tasks, particularly when attention is being drawn elsewhere. Another perspective to consider is that other adversities, as captured by our ACE questionnaire, may have been more benign in nature when compared to abuses, and household dysfunction. For instance, the average subjective rating of abuses was 8.4/10, and 7.8/10 for household dysfunction, compared to an average rating of 6.9/10 for other adversities, and therefore, it can be inferred that household dysfunction and abuses are perceived as more severe and impactful forms of childhood adversity than other adversities in our study sample.

Thus, a sensitization effect may occur where individuals are presented with more frequent and less severe ACEs, resulting in attentional resources being drawn to irrelevant stimuli (UNT). A state of hypervigilance may be induced in which individuals constantly monitor their environment to detect threats, even if what they are paying attention to in the environment may not be relevant.

The observed positive association between alpha N1 ITC and other adversities may indicate that individuals who have been exposed to these adverse events could exhibit heightened neural synchronization, i.e., a more consistent early attentional response. Further, these increases in alpha N1 ITC may implicate a compensatory mechanism used to maintain cognitive function despite increases in environmental stress during childhood. Much of the research surrounding positive outcomes despite ACEs focuses on unique qualities of children who succeed, with a common theme of resilience emerging (Masten, 2001). Importantly, the increased neural phase consistency observed in the present study may reflect an adaptive response that assists individuals in maintaining attentional control and early sensory processing despite experiences of childhood adversity.

4.5 Limitations and Future Directions

One limitation is the sex imbalance of our sample, with only nine men completing all parts of the study compared to fifty-three women. This imbalance may affect the generalizability of our results to the population more broadly. We suspect that this sex imbalance arose naturally as a product of the makeup of the student body at MSVU during the primary period of recruitment (76% female, 23% male, 1% non-binary). While we did recruit individuals from SMU and the general public, where the sex distribution is closer to fifty-percent male and fifty-percent female, our sample remained skewed toward women. It may be valuable for future

research to aim for a more balanced sex distribution. By doing so, it would help increase the generalizability of these results to a broader population, representative across sexes. While ACEs affect both sexes, research suggests that on average women report a greater number of ACEs than men (Dube et al., 2003). Further, women are more likely to experience internalizing disorders (e.g., depression or anxiety), where men are more likely to exhibit externalizing behaviours (e.g., aggression or substance abuse) as a result of ACEs (Wegman & Stetler, 2009).

Another important factor to consider is the interconnectedness between psychological factors, such as depression and anxiety, and ACEs (Chapman et al., 2007). Given that these psychological conditions are common outcomes of ACEs, it is challenging to disentangle the direct impacts of ACEs on spectral power and phase consistency. Recall, or persistent rumination on these early childhood experiences may also contribute to the observed changes in ERSP and ITC into adulthood. Not only can these mechanisms amplify emotional distress, but they could also alter neurophysiological outcomes independent of the initial traumatic experiences. In order to address this potential confound, it would be valuable for future studies to integrate assessments of psychopathology, particularly anxiety, depression, and PTSD, to better isolate the impacts of ACEs on neurophysiology.

The exclusive focus on the ERN, N1, and N2 waveforms generated by an auditory selective attention and auditory Go/NoGo task may be limiting. While these three ERPs provide insights into mechanisms of early attention and cognitive control, it may be valuable to investigate additional domains of the information processing stream. For instance, other ERP components such as the P50 or the error positivity may complement our analyses investigating individual differences in neural signatures as they relate to ACEs during response inhibition tasks. Furthermore, future research could use a visual task to evoke the N1 waveform to evaluate

if differences in observed ERSP and ITC to a visually evoked N1 differ from those generated by an auditory selective attention task. Beyond this, we also averaged ERSP and ITC across participants for our target waveforms. This approach may not fully account for temporal variability among participants and could mask individual timing of neural responses. While the N1 and ERN waveforms reflect more instantaneous cognitive processes, waveforms like the N2 (reflecting response selection) may vary temporally across individuals. This temporal variation could lead to inaccuracies in capturing the true neural dynamics reflected by these ERPs in some participants. To address this, future studies should consider analyzing peak timings on an individual level. While this approach may be more computationally intensive, it would allow for a more accurate and individualized evaluation of neural responses.

Lastly, the sequence of events during the EEG testing session may have introduced a priming effect where participants entered the recording chamber in a negative affective state. Upon arrival to the electrophysiology lab, participants were instructed to review and sign the consent form, as well as complete the ACE questionnaire prior to the EEG session. As the ACE questionnaire requires individuals to reflect on experiences of early childhood adversity, this could lead to a negative or dysphoric affective state prior to the testing session. While the application of the EEG net requires approximately thirty minutes, in addition to the initial twenty-five-minute P50 task prior to the paradigms described in the present study, it does not completely eliminate the risk of priming effects. Participants may not return to baseline affective states and could still be influenced by the emotional content of the questionnaires, thus affecting neural responses. It may be valuable for future research to consider the timing of questionnaire measures that could induce negative affective states, and ensure they are completed prior to neurophysiological recording sessions.

4.6 Implications

The current findings offer several insights that contribute to a better understanding of the relationship between patterns of ERP activity and childhood adversity. By uncovering significant relationships among ACEs and neural oscillatory dynamics on alpha and theta frequency bands, we highlight the differential effects of ACEs on the information processing stream. Specifically, the observed effects manifest as reduced alpha power and impaired theta synchronization, these deficits were primarily observed during tasks requiring conflict monitoring and inhibitory control. The identification of these neural signatures may provide a tool for clinicians to promptly detect and manage potential deficits in cognitive function for individuals exposed to increasing levels of childhood adversity. These insights into early markers of adversity, and the subsequent effects on cognition, can support the integration of timely interventions for those experiencing ACEs to support healthy neurological development and cognitive functioning into adulthood (Cavanagh & Frank, 2014; Klimesch, 2012).

The integration of intervention strategies using both neuroimaging and electrophysiologic measures could be used to support children and adults who have experienced ACEs. This could include neurofeedback and mindfulness-based programs–both of which enhance neural synchronization and cognitive control mechanisms (Gruzelier, 2014; Tang et al., 2015). Further, these interventions can be individualized to address specific deficits in neural activity and promote overall well-being. Notably, mindfulness-based interventions enhance neural synchronization and improve overall attention; thus, making mindfulness training a viable option for mitigating the negative effects of ACEs (Zeidan et al., 2010). Neurofeedback training is a non-invasive procedure that can be used to monitor neural oscillations and provide feedback to individuals to help them regulate neural activity (Gruzelier, 2014).

Our findings can be used proactively to identify specific patterns of alpha and theta neural activation and synchronization that put individual at risk of neurostructural and functional alterations. Neural signatures corresponding to dimensions of childhood adversity can be targeted through neurofeedback and mindfulness-based programs to help mitigate harmful effects (Gruzelier, 2014; Tang et al., 2015). In essence, this approach not only addresses neurocognitive deficits caused by ACEs but can also support long-term psychological health and well-being in those affected by early adversity.

4.7 Conclusion

The present study explored the relationships among dimensions of ACEs and neural oscillatory dynamics during a response inhibition auditory Go/NoGo, and an auditory selective attention paradigm. Specifically, we focused on evaluating individual differences in ERSP and ITC of the ERN, N1, and N2 waveforms on alpha and theta frequency bands and how they may represent neural signatures that relate to dimensions of childhood adversity. We observed several ERSP and ITC signatures across alpha and theta that correspond to experiences of childhood adversity; particularly, abuse and other adversities during the Go/NoGo task. Interestingly, we observed compensatory mechanisms during the auditory selective attention task, such that increased alpha ITC significantly corresponded to increased experiences of other adversities. Together, these results highlight the differential effects of ACEs on dimensions of neurophysiological activity and how individual differences in neural signatures may be predictive of early childhood adversity.

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Appendix A: Collinearity Diagnostics

Table A1.

Collinearity Diagnostics for ACE dimensions

	Tolerance	VIF
ACE Count	.02	16.48
Abuses	.36	2.80
Household Dysfunction	.60	1.67
Other Adversities	.32	3.14
Subjective Rating	.14	7.34

Note. VIF = Variance Inflation Factor

Table A2.

Correlations Among ACE Dimensions

	ACE Count	Abuses	Household Dysfunction	Other Adversities
ACE Count	--			
Abuse	.66**	--		
Household Dysfunction	.65**	.32*	--	
Other Adversities	.86**	.31*	.35**	--
Subjective ACE Rating	.92**	.71**	.58**	.75**

Note. *$p < .05$; **$p < .005$

Table A3.

Collinearity Diagnostics for ERN Alpha and Theta ERSP and ITC

	Tolerance	VIF
ERN ERSP Alpha Correct	.47	2.14
ERN ERSP Alpha Errors	.25	4.00
ERN ERSP Theta Correct	.48	2.11
ERN ERSP Theta Errors	.27	3.68
ERN ITC Alpha Correct	.21	4.67
ERN ITC Alpha Errors	.12	8.16
ERN ITC Theta Correct	.22	4.58
ERN ITC Theta Errors	.13	7.77

Table A4.

Collinearity Diagnostics for N2 Alpha and Theta ERSP and ITC

	Tolerance	VIF
N2 Alpha ERSP Correct Go	.37	2.67
N2 Alpha ERSP Correct NoGo	.51	1.98
N2 Alpha ERSP Error Go	.34	2.96
N2 Alpha ERSP Error NoGo	.32	3.11
N2 Theta ERSP Correct Go	.18	5.48
N2 Theta ERSP Correct NoGo	.62	1.62
N2 Theta ERSP Error Go	.37	2.68
N2 Theta ERSP Error NoGo	.54	1.86
N2 Alpha ITC Correct Go	.20	5.09
N2 Alpha ITC Correct NoGo	.21	4.70
N2 Alpha ITC Error Go	.36	2.80
N2 Alpha ITC Error NoGo	.76	1.32
N2 Theta ITC Correct Go	.20	5.08
N2 Theta ITC Correct NoGo	.26	3.82
N2 Theta ITC Error Go	.39	2.57

Table A5.

Collinearity Diagnostics for N1 Alpha and Theta ERSP and ITC

	Tolerance	VIF
N1 Alpha ERSP Attended Nontarget	.49	2.03
N1 Alpha ERSP Attended Target	.53	1.90
N1 Alpha ERSP Unattended Nontarget	.43	2.34
N1 Alpha ERSP Unattended Target	.31	3.22
N1 Theta ERSP Attended Nontarget	.46	2.16
N1 Theta ERSP Attended Target	.52	1.94
N1 Theta ERSP Unattended Nontarget	.38	2.67
N1 Theta ERSP Unattended Target	.24	4.18
N1 Alpha ITC Attended Nontarget	.22	4.53
N1 Alpha ITC Attended Target	.31	3.22
N1 Alpha ITC Unattended Nontarget	.44	2.29
N1 Alpha ITC Unattended Target	.53	1.91
N1 Theta ITC Attended Nontarget	.20	5.00
N1 Theta ITC Attended Target	.34	2.95
N1 Theta ITC Unattended Nontarget	.28	3.62
N1 Theta ITC Unattended Target	.24	4.10

Appendix B: Non-Significant ERN Regressions

Table B1.

ERN Alpha ERSP for Correct and Error Responses and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	.40	.49	.11	.82	.42
Error ERSP	-.21	.25	-.11	-.82	.42

Table B2.

ERN Alpha ERSP for Correct and Error Responses and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	.18	.18	.13	1.01	.32
Error ERSP	-.08	.09	-.12	-.89	.38

Table B3.

ERN Alpha ERSP for Correct and Error Responses and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	.23	.15	.19	1.49	.14
Error ERSP	-.04	.08	-.06	-.48	.63

Table B4.

ERN Alpha ERSP for Correct and Error Responses and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	-.01	.32	-.01	-.02	.98
Error ERSP	-.09	.16	-.071	-.54	.59

Table B5.

ERN Alpha ERSP for Correct and Error Responses and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	5.85	4.52	.17	1.30	.20
Error ERSP	-1.87	2.31	-.11	-.81	.42

Table B6.

ERN Theta ERSP for Correct and Error Responses and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	.38	.41	.12	.93	.36
Error ERSP	-.14	.24	-.08	-.59	.56

Table B7.

ERN Theta ERSP for Correct and Error Responses and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	.05	.15	.04	.33	.75
Error ERSP	-.09	.09	-.13	-.99	.33

Table B8.

ERN Theta ERSP for Correct and Error Responses and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	.12	.13	.13	.97	.34
Error ERSP	-.06	.07	-.10	-.79	.43

Table B9.

ERN Theta ERSP for Correct and Error Responses and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	.21	.26	.10	.79	.43
Error ERSP	.01	.15	.01	.03	.98

Table B10.

ERN Theta ERSP for Correct and Error Responses and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ERSP	4.44	3.72	.16	1.20	.24
Error ERSP	-2.02	2.18	-.12	-.93	.36

Table B11.

ERN Alpha ITC for Correct and Error Responses and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	16.09	47.61	.05	.34	.74
Error ITC	-2.61	3.92	-.09	-.67	.51

Table B12.

ERN Alpha ITC for Correct and Error Responses and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	14.77	16.78	.12	.88	.38
Error ITC	.88	1.38	.09	.64	.53

Table B13.

ERN Alpha ITC for Correct and Error Responses and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	-11.33	14.94	-.10	-.76	.45
Error ITC	-1.05	1.23	-.11	-.85	.40

Table B14.

ERN Alpha ITC for Correct and Error Responses and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	12.65	30.19	.06	.42	.68
Error ITC	-2.45	2.49	-.13	-.98	.33

Table B15.

ERN Alpha ITC for Correct and Error Responses and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	422.67	442.37	.13	.96	.34
Error ITC	-5.16	36.44	-.02	-.14	.89

Table B16.

ERN Theta ITC for Correct and Error Responses and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	-13.01	34.88	-.05	-.37	.71
Error ITC	-.63	3.60	-.02	-.17	.86

Table B17.

ERN Theta ITC for Correct and Error Responses and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	6.18	12.12	.07	.51	.61
Error ITC	1.25	1.25	.13	1.00	.32

Table B18.

ERN Theta ITC for Correct and Error Responses and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	-5.24	10.83	-.06	-.48	.63
Error ITC	-.09	1.12	-.01	-.07	.94

Table B19.

ERN Theta ITC for Correct and Error Responses and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	-13.95	22.11	-.08	-.63	.53
Error ITC	-1.79	2.28	-.10	-.79	.44

Table B20.

ERN Theta ITC for Correct and Error Responses and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct ITC	46.78	322.80	.02	.15	.89
Error ITC	7.40	33.30	.03	.22	.83

Appendix C: Non-Significant N2 Regressions

Table C1.

N2 Alpha ERSP Across Trial Conditions and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	.08	.32	.06	.25	.81
Correct NoGo ERSP	-.33	.27	-.27	-1.20	.24
Error Go ERSP	.17	.15	.19	1.10	.28
Error NoGo ERSP	-.03	.12	-.04	-.23	.82

Table C2.

N2 Theta ERSP Across Trial Conditions and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	-.08	1.02	-.01	-.08	.94
Correct NoGo ERSP	-.53	.44	-.22	-1.20	.24
Error Go ERSP	.33	.33	.15	1.00	.32
Error NoGo ERSP	-.14	.32	-.07	-.43	.67

Table C3.

N2 Theta ERSP Across Trial Conditions and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	-.30	.31	-.15	-.98	.33
Correct NoGo ERSP	-.10	.13	-.14	-.78	.44
Error Go ERSP	.01	.10	.01	.01	.99
Error NoGo ERSP	-.02	.10	-.02	-.16	.88

Table C4.

N2 Theta ERSP Across Trial Conditions and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ERSP	.32	.68	.08	.47	.64
Correct NoGo ERSP	-.08	.30	-.05	-.26	.80
Error Go ERSP	.22	.22	.15	.99	.33
Error NoGo ERSP	-.20	.21	-.16	-.93	.36

Table C5.

N2 Theta ERSP Across Trial Conditions and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	Std. Error	Beta	t	*p*
Correct Go ERSP	-5.48	9.19	-.10	-.60	.55
Correct NoGo ERSP	-4.48	3.98	-.21	-1.13	.27
Error Go ERSP	3.96	2.96	.20	1.34	.19
Error NoGo ERSP	-1.14	2.86	-.06	-.40	.69

Table C6.

N2 Alpha ITC Across Trial Conditions and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	-26.46	41.34	-.11	-.64	.53
Correct NoGo ITC	-.30	7.75	-.01	-.04	.97
Error Go ITC	-3.44	4.14	-.12	-.83	.41
Error NoGo ITC	-.40	4.81	-.02	-.08	.93

Table C7.

N2 Alpha ITC Across Trial Conditions and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	-8.44	12.96	-.11	-.65	.52
Correct NoGo ITC	-4.36	2.43	-.25	-1.80	.08
Error Go ITC	-1.85	1.30	-.21	-1.43	.16
Error NoGo ITC	-.33	1.51	-.040	-.22	.83

Table C8.

N2 Alpha ITC Across Trial Conditions and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	-38.04	26.52	-.25	-1.43	.16
Correct NoGo ITC	-.41	4.97	-.01	-.08	.93
Error Go ITC	1.08	2.65	.06	.41	.69
Error NoGo ITC	-.83	3.09	-.05	-.27	.79

Table C9.

N2 Alpha ITC Across Trial Conditions and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	-.86	382.65	.00	-.01	.99
Correct NoGo ITC	23.04	71.75	.05	.32	.75
Error Go ITC	-48.45	38.29	-.18	-1.3	.21
Error NoGo ITC	8.22	44.55	.03	.18	.85

Table C10.

N2 Theta ITC Across Trial Conditions and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	43.13	35.39	.19	1.22	.23
Correct NoGo ITC	-4.81	8.90	-.09	-.54	.59
Error Go ITC	-2.06	4.11	-.08	-.50	.62
Error NoGo ITC	-7.97	4.51	-.29	-1.77	.08

Table C11.

N2 Theta ITC Across Trial Conditions and Other

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	5.87	23.72	.04	.25	.81
Correct NoGo ITC	-.27	5.97	-.01	-.05	.96
Error Go ITC	.99	2.76	.06	.36	.72
Error NoGo ITC	-2.95	3.02	-.17	-.98	.34

Table C12.

N2 Theta ITC Across Trial Conditions and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Correct Go ITC	441.15	319.03	.21	1.38	.17
Correct NoGo ITC	-21.81	80.27	-.05	-.27	.79
Error Go ITC	-35.42	37.05	-.15	-.96	.34
Error NoGo ITC	-66.04	40.61	-.26	-1.63	.11

Appendix D: Non-Significant N1 Regressions

Table D1.

N1 Alpha ERSP Across Trial Conditions and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	-.85	1.44	-.09	-.59	.56
Attended Target ERSP	-.89	.72	-.17	-1.23	.23
Unattended Nontarget ERSP	-1.50	1.41	-.15	-1.06	.29
Unattended Target ERSP	2.73	1.60	.29	1.70	.09

Table D2.

N1 Alpha ERSP Across Trial Conditions and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	-.14	.53	-.04	-.26	.80
Attended Target ERSP	-.20	.27	-.11	-.76	.45
Unattended Nontarget ERSP	-.64	.52	-.18	-1.23	.22
Unattended Target ERSP	.69	.59	.20	1.17	.25

Table D3.

N1 Alpha ERSP Across Trial Conditions and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	.05	.46	.02	.11	.91
Attended Target ERSP	-.10	.23	-.06	-.43	.67
Unattended Nontarget ERSP	-.64	.45	-.20	-1.43	.16
Unattended Target ERSP	.32	.51	.11	.64	.53

Table D4.

N1 Alpha ERSP Across Trial Conditions and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	-.76	.93	-.13	-.82	.42
Attended Target ERSP	-.59	.47	-.180	-1.26	.21
Unattended Nontarget ERSP	-.22	.91	-.03	-.24	.81
Unattended Target ERSP	1.71	1.03	.28	1.66	.10

Table D5.

N1 Alpha ERSP Across Trial Conditions and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	-2.81	13.30	-.03	-.21	.83
Attended Target ERSP	-4.17	6.65	-.09	-.63	.53
Unattended Nontarget ERSP	-22.46	13.03	-.24	-1.72	.09
Unattended Target ERSP	21.40	14.77	.24	1.45	.15

Table D6.

N1 Theta ERSP Across Trial Conditions and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	-.56	.96	-.09	-.59	.56
Attended Target ERSP	-.23	.44	-.07	-.52	.60
Unattended Nontarget ERSP	.09	1.05	.01	.09	.93
Unattended Target ERSP	1.42	1.01	.25	1.41	.17

Table D7.

N1 Theta ERSP Across Trial Conditions and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	.25	.35	.11	.73	.47
Attended Target ERSP	-.09	.16	-.08	-.57	.57
Unattended Nontarget ERSP	-.02	.38	-.01	-.05	.96
Unattended Target ERSP	.29	.37	.14	.78	.44

Table D8.

N1 Theta ERSP Across Trial Conditions and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	-.05	.30	-.02	-.15	.88
Attended Target ERSP	-.06	.14	-.06	-.44	.66
Unattended Nontarget ERSP	-.18	.33	-.08	-.54	.59
Unattended Target ERSP	.46	.32	.26	1.47	.15

Table D9.

N1 Theta ERSP Across Trial Conditions and Other Adversities

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	-.77	.62	-.19	-1.26	.21
Attended Target ERSP	-.08	.28	-.04	-.27	.79
Unattended Nontarget ERSP	.29	.67	.07	.43	.67
Unattended Target ERSP	.67	.65	.18	1.03	.31

Table D10.

N1 Theta ERSP Across Trial Conditions and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	Std. Error	Beta	t	*p*
Attended Nontarget ERSP	2.03	8.49	.04	.24	.81
Attended Target ERSP	-1.34	4.35	-.04	-.31	.76
Unattended Nontarget ERSP	3.41	9.60	.05	.36	.72
Unattended Target ERSP	9.95	9.57	.18	1.04	.30

Table D11.

N1 Alpha ITC Across Trial Conditions and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ITC	.38	5.85	.01	.07	.95
Attended Target ITC	1.08	9.87	.02	.11	.91
Unattended Nontarget ITC	21.64	20.22	.15	1.07	.29
Unattended Target ITC	1.97	17.73	.02	.11	.91

Table D12.

N1 Alpha ITC Across Trial Conditions and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	.78	2.10	.05	.37	.71
Attended Target ERSP	-2.49	3.54	-.10	-.71	.48
Unattended Nontarget ERSP	-7.56	7.24	-.15	-1.04	.30
Unattended Target ERSP	11.65	6.35	.26	1.83	.07

Table D13.

N1 Alpha ITC Across Trial Conditions and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ITC	1.04	1.84	.08	.57	.57
Attended Target ITC	.41	3.10	.02	.13	.90
Unattended Nontarget ITC	-.63	6.35	-.01	-.10	.92
Unattended Target ITC	2.33	5.57	.06	.42	.68

Table D14.

N1 Alpha ITC Across Trial Conditions and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ITC	-3.74	54.08	-.01	-.07	.95
Attended Target ITC	16.53	91.26	.03	.18	.86
Unattended Nontarget ITC	151.00	186.93	.12	.81	.42
Unattended Target ITC	74.45	163.94	.07	.45	.65

Table D15.

N1 Theta ITC Across Trial Conditions and ACE Count

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ITC	1.75	5.31	.04	.33	.74
Attended Target ITC	.23	6.38	.01	.04	.97
Unattended Nontarget ITC	-2.61	10.01	-.05	-.26	.80
Unattended Target ITC	9.04	11.01	.17	.82	.42

Table D16.

N1 Theta ITC Across Trial Conditions and Abuse

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	.49	1.92	.03	.25	.80
Attended Target ERSP	-1.06	2.30	-.07	-.46	.65
Unattended Nontarget ERSP	-5.74	3.61	-.32	-1.59	.12
Unattended Target ERSP	5.79	3.97	.30	1.46	.15

Table D17.

N1 Theta ITC Across Trial Conditions and Household Dysfunction

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ERSP	1.27	1.65	.10	.77	.44
Attended Target ERSP	.98	1.98	.07	.49	.62
Unattended Nontarget ERSP	-2.11	3.10	-.14	-.68	.50
Unattended Target ERSP	2.94	3.41	.18	.86	.40

Table D18.

N1 Theta ITC Across Trial Conditions and Other

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ITC	.01	3.40	.00	.00	1.00
Attended Target ITC	.31	4.08	.01	.08	.94
Unattended Nontarget ITC	5.24	6.40	.17	.82	.42
Unattended Target ITC	.31	7.04	.01	.04	.97

Table D19.

N1 Theta ITC Across Trial Conditions and Subjective Experience

	Unstandardized Coefficients		Standardized Coefficients		
Predictors	B	SE	Beta	t	*p*
Attended Nontarget ITC	-15.16	49.08	-.04	-.31	.76
Attended Target ITC	-1.34	58.91	-.01	-.02	.98
Unattended Nontarget ITC	-73.82	92.45	-.16	-.80	.43
Unattended Target ITC	109.47	101.72	.23	1.08	.29

Appendix E: Consent Form

CONSENT FORM	Date: _____	
TITLE OF STUDY: The Effects of Life Experiences on Brain Functioning, Cognition, and Emotion		
Principal Investigator		
Dr. Christine Lackner		
Department of Psychology		
Mount Saint Vincent University		
(902) 457-5981		
Christine.Lackner@msvu.ca		

Dear Participant:

This form provides you with the information you will need to make an informed decision about whether or not you would like to participate in our study on brain functioning. Please read it over carefully. If you have any questions, you are welcome to phone the principal investigator, Dr. Christine Lackner at 902-457-5981 for clarification.

The goal of the study is to investigate the effects of childhood experiences on the brain using EEG, a technology that allows us to measure brain waves. Additionally, we will be investigating how childhood experiences may affect growth of the legs and torso. We will then relate these measures to questionnaires focusing on anxiety, depression, cognitive and emotion regulation, stress, and executive functioning. We believe that this study will lead to a richer understanding of how childhood experiences are associated with brain and physical development, and whether these effects are long-lasting.

WHAT IS INVOLVED

Participants will come to the EEG lab located in Evaristus at Mount Saint University for a three-hour session. All the tasks and procedures will be explained, and we will review this letter together, so you will have a full understanding of what is involved before we begin.

You will be asked to fill out a series of questionnaires in which you will rate your general sense of well-being as well as the degree to which you may at times feel depressed, anxious, or stressed. **You will be asked to think about your childhood experiences, including ones that may cause you stress and anxiety to think about. For instance, we may ask you if you experienced the death of a loved one during childhood, or if you were sexually assaulted in childhood. We will ask how well you are currently dealing with those experiences, and can provide you with professional resources should these experiences be causing you ongoing distress.**

Exclusion criteria includes individuals with uncontrolled epilepsy (this makes EEG data uninterpretable. You need

to have normal or corrected-to-normal vision and speak and read English fluently. This ensures you can read and answer the questionnaires and see the stimuli for the computer task. You need to have normal hearing and not require the use of hearing aids. This impairs our ability to deliver auditory stimuli to you during testing.

Additionally, if you have type 3 (i.e., curly) or type 4 (i.e., coily) hair, to ensure the electrodes can be securely placed directly onto your scalp, we ask that you get your all of your hair braided in 10 to 20 straight-back cornrows with a middle part (see photo below) except if your hair is very short or buzzed. If you go to a hair salon to get your hair cornrowed, you can bring the receipt at your in-person appointment, and you will get reimbursed. Protective styles like braids, dreadlocks, weaves, and faux-locs hinder the attachment of the EEG cap and electrodes.

Image from Etienne et al. (2020) Novel electrodes for reliable EEG recordings on coarse and curly hair. *42nd Annual International Conference of the IEEE Engineering in Medicine & Biology Society (EMBC)* (pp. 6151-6154). IEEE.

While you are in the lab, a soft, elasticized sensor cap (or plastic comb-like clips if you have type 3 or 4 hair) and some gel is placed on your head to record naturally-occurring brain activity while you engage in a computer tassk. We will measure your head to ensure the accuracy of cap placement. The tasks involve listening to auditory stimuli (verbal and non-verbal sounds) through ear phones. In some tasks you will press a key in response to some sounds but not others. The session is expected to take three hours.

POTENTIAL RISKS AND BENEFITS

There is minimal risk associated with participation in the study. The following minimal risks may be experienced as part of participation in the research study: The EEG cap can become a little uncomfortable. If you feel the discomfort level is too high, we will remove the cap immediately upon your request. Your hair will be messy as a result of wearing the cap, and a small amount of gel will remain in your hair until it is washed.

Finally, you may feel some discomfort when filling out questionnaires due to their sensitive nature. If at any point you are upset by these questions, you can withdraw without penalty. Additionally, Mount Saint Vincent University offers counselling services, who can be contacted at 902-457-6567, or by email counselling@msvu.ca. The Nova Scotia Health Authority Mental Health Mobile Crisis phone line can be reached at 902-429-8167 or 1-888-429-8167. Community Mental Health Services, Bayer's Road location can be reached at 902-454-1661 ext 902. The Avalon Sexual Assault Centre can be reached at 902-425-0122 or 902-421-1188.

There are no direct benefits to participating in this study. Potential benefits include introducing participants to research. The techniques and procedures will be fully explained to you and you will be free to ask questions throughout. Most participants find it exciting to see their brain waves on the computer screen. As well, individuals often feel good about taking part in a project that could increase our understanding of the factors that influence development throughout childhood, adolescence, and adulthood.

REIMBURSEMENT/COMPENSATION

You may qualify for bonus points or course credit for certain participating psychology classes, and all participants will be given their choice of a $25 gift certificate to Amazon.ca, Sobey's or Tim Hortons. Those who have had their hair cornrowed will be reimbursed for this expense if a receipt is provided. Note that we can provide a blank receipt for you to have your hair stylist fill in.

CONFIDENTIALITY

All information gathered is kept completely confidential. Names are replaced with code numbers and it is these code numbers that are entered into our data base along with the physiological and behavioural information. If you receive to wish a bonus point for participation, you can fill out the sheet that will be provided to you by the researcher with your name and course number, or if you have signed up using SONA, you can provide this information there. The information will be used to inform your professor that you have participated in research and received a bonus point or course credit.

The electrophysiological (brain wave, EEG) data collected will be stored in two places (1) on CDs and hard drives in a controlled-access laboratory under the direction of Dr. Christine Lackner, and (2) data will be anonymized an uploaded to EEGNet, a Brain Canada funded platform that shares EEG data with neuroscientists and internationally to advance our understanding of the brain. The EEG data may be used for purposes other than described here. EEGNet servers are located in Canada, and are only accessible by researchers who have been given authorized permission. Given our current understanding of the brain, no one who views your EEG data will be able to identify you (i.e., we do not have reliable brain fingerprinting technology). Your file is not associated with identifying information on EEGNet. I agree to be re-contacted in case there are changes to REB protocols, amendments or extensions that might affect my willingness to have my EEG data stored on EEGNet.

All paper records (e.g., consent forms) of the information will be destroyed when no longer required, or after 5 years, whichever comes first. Anonymized, digital data (e.g., scores in an Excel file without your name associated with them) will be stored for indefinitely. You will never be identified in any way when the data are published. You will in no way be identified or associated with the findings of this study.

The protection of health, life and safety, may justify infringement of privacy and confidentiality. The researcher may be obligated by legal, professional, or ethical duties to disclose normally confidential information if it becomes evident that you are a threat to yourself or others, or that there is ongoing abuse or maltreatment. Student Health Services, Campus Security, Halifax Regional Police, or other required professional organizations may be contacted if such a threat is deemed to exist.

PARTICIPATION IS VOLUNTARY

Participation in this study is entirely voluntary. You may decline to answer any question or to refrain from participating in any component of this study. As well, you may decide to withdraw from this study at any time without penalty even after signing this form. Non-participating in this study will not affect your grades, or your instructor's evaluation of your performance.

CONTACT INFORMATION

If you have any questions about this study or if you would like further information, please contact the Principle Investigator Dr. Christine Lackner (contact information above). This study has received ethics clearance from the Research Ethics Board of Mount Saint Vincent University (#XXXXXX). If you have concerns about the conduct of this study and wish to speak with someone not directly involved with the research, please contact the University Research Ethics Board, c/o The Research Office at 457-6350 or ethics@msvu.ca.

Thank you for considering this project. If you would like to participate, please sign and date below and return to the research assistant.

CONSENT

I agree to participate in the study described above and to have my EEG data uploaded to EEGNet. I agree to be re-contacted in the future should changes to REB protocols occur that may influence my decision to have my data stored on EEGNet. I have made the decision to participate based on the information provided above and have had the opportunity to receive any further details and understand that I am welcome to ask any further questions in the future. I also understand that I can withdraw this consent at any time without penalty even after signing this form.

Signature: _____ Date: _____

Please provide your contact information should we need to re-contact you about changes to data storage on EEGNet.

_____

If you would like to be informed of the results of this study, please provide your email:

_____

Appendix F: ACE Questionnaire

Adverse Childhood Experiences Questionnaire

Remember that all of the information you provide will be kept strictly confidential and will be coded anonymously so that your name will never be associated with answers to any questions.

It is important for us to understand what may have happened to you in the past. The questions below describe some kinds of upsetting experiences. Since we give these questions to everyone, we list a lot of possible events that may have happened at any time in your life. If one or more of these experiences has happened at some time in your life, please circle yes and answer questions asked by circling appropriate responses.

1= no stress 10 = a great deal of stress

Death of a parent	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Badly frightened or attacked by an animal	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Death of a family member (other than a parent)	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
In a bad car accident	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)

Divorce or separation of parents	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Experienced a natural disaster (e.g. fire, tornado, earthquake)	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
A stay in the hospital (at least one night)	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Lost a pet you really cared about (died, killed or lost)	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)

A stay in a foster home	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Saw someone get badly hurt or die suddenly	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Separation from parents	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Family member or residence was robbed	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Experienced a serious illness or injury	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)

Had someone in your household who abused drugs or alcohol	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Had someone in your household who tried to hurt or kill themselves	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Had a family member who was depressed or mentally ill for a long time	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Had someone in your household be physically violent towards you	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Been threatened or picked on by a bully	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)

Had a parent swear at you, insult you, or put you down	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
An adult who said/acted like they were going to hurt you really bad or kill you	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Heard or seen family members act like they were going to kill each other or hurt each other	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Seen a family member being hit, punched, kicked, or killed	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Had someone try to rob you or your family with a weapon	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)

Had a family member who was kidnapped, or have been kidnapped yourself	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Seen someone in the neighborhood being beat up, shot at, or killed	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Had a pet or animal that was killed on purpose by someone you know	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Seen a friend killed	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Had someone touch your private sexual body parts when you did not want them to	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)

Had someone make you touch his or her sexual body parts when you did not want to	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Had an adult lock you up, gag you, blindfold you, or lock you in a closet	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
Moved residences or schools	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)
A serious illness or injury of a family member	Yes	No	If yes, how much stress did this cause you? How often do you currently think about this? When did it occur? (Approximate Age) How long did the initial stress or trauma of this event last?	(1) (2) (3) (4) (5) (6) (7) (8) (9) (10) (Once a week) (Once a month) (Once a year) (Never) (0-3) (4-8) (9-13) (14-18) (Later than 18) (Minutes) (Hours) (Days) (Weeks) (Year) (Years)