ABSTRACT

Title of Dissertation:	ASSESSING PERSONALITY, PSYCHOPATHOLOGY, AND NEUROPHYSIOLOGY RELATIONSHIPS: A WITHIN-SUBJECTS META-ANALYTIC APPROACH
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Background. Emerging transdiagnostic frameworks now provide compelling classifications to characterize the core structure of latent systems underlying both psychopathology and neurophysiology measures indexing cognitive-affective systems. For psychopathology, a primary transdiagnostic model is the Hierarchical Taxonomy of Psychopathology (HiTOP), which describes a broad internalizing factor (INT; characterizing comorbidity between fear and distress disorders), externalizing factor (EXT; aggression, disinhibition, and substance use), and general psychopathology factor (p-factor; indexing the shared variance between INT and EXT). To frame cognitive domains, the proposed work will utilize the Research Domain Criteria (RDoC), a transdiagnostic framework for indexing brain systems underlying cognitive-affective processing. A specific focus will be on positive valence (PV), negative valence (NV), and cognitive processes (COG) domains by

utilizing the Multidimensional Personality Questionnaire (MPQ) and neurobiological EEG measures. Based on previous work in the lab and field, we expect all measures of psychopathology to relate similarly to the RDoC measures: increased NV, and decreased COG and PV. The project aimed to review this relationship in more traditional self-report measure only analyses, as well as through a novel within-subjects meta-analytic approach to, importantly, incorporate neural markers as an objective measure beyond the self-report measures.

Methods. The project will utilize an archival dataset. The participants completed a self-report questionnaire battery and underwent a multi-task protocol of cognitive-affective tasks while EEG data was collected. Regression analyses were conducted with the collected self-report measures. A correlation table of the self-report measures correlated with the neural measures was used as the base data table with which meta-analyses (t-tests, regressions, and general linear models) were conducted.

Results. Although p-factor related to the RDoC measures as expected, there were distinctions in the INT and EXT relationships. Of note, the regression results were consistent across both self-report and meta-analytic approaches.

Discussion. The establishment of these relationships advances the goals of both transdiagnostic models by relating psychopathology (HiTOP) to cognitive processes (RDoC). The novel meta-analytic approach valuably adds objective neurophysiological measure information to the self-report data. Although not without its considerations, the meta-analytic approach can add to current analytical approaches, and future studies could aim to replicate and improve upon this methodology.

ASSESSING PERSONALITY, PSYCHOPATHOLOGY, AND NEUROPHYSIOLOGY RELATIONSHIPS: A WITHIN-SUBJECTS META-ANALYTIC APPROACH

by

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Dedication

To my maternal grandfather (Thatha), Vishnu Vardhana Rao, who prioritized, valued, and encouraged the highest of educational pursuits for his three daughters and three granddaughters, as much as he did for his two grandsons.

To my maternal grandmother (Ammamma), Sita Mahalakshmi, who taught me what strength, patience, and sacrifice looked like even in the hardest moments of life…and for always playing with me – a person who always saw the lighter and brighter sides of life.

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Finally, and importantly, thank you to the individuals whose participation in the study made this work possible. My hope is that this work can advance mental health knowledge and treatments, to improve the lives of those affected, and the lives of their loved ones.

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HiTOP: Hierarchical Taxonomy of Psychopathology

- INT: Internalizing
  - IDAS: Inventory of Depression and Anxiety Symptoms
- EXT: Externalizing
  - ESI: Externalizing Spectrum Inventory
- p-factor: General psychopathology factor

MPQ: Multidimensional Personality Questionnaire

- CON: Constraint
- PEM: Positive Emotionality
- NEM: Negative Emotionality

RDoC: Research Domain Criteria

- COG: Cognitive Control
- PV: Positive Valence
- NV: Negative Valence

EEG: Electroencephalogram

- ERP: Event-related potentials
- ICPS: Inter-channel phase synchrony
- ITPS: Inter-trial phase synchrony

Chapter 1: Introduction

Psychopathology and its impact on society

The prevalence of mental illness has been on the rise, noting a 13% rise in mental health conditions and substance use disorders from 2007 to 2017 (World Health Organization, 2022a). Around the world, as of 2019, 1 in every 8 people were living with a mental health disorder (World Health Organization, 2022a). This was further exacerbated by the pandemic resulting in a 25% increase in prevalence of anxiety and depression worldwide (World Health Organization, 2022b). The American Psychiatric Association reported in December 2022 that 37% of Americans rated fair or poor mental health (up from 31% a year ago), and that for 2023, 26% expected more stress (up from 20% a year ago). At the same time, the utilization of mental health resources has increased from 2019 to 2021, from 19.2% to 21.6% (Centers for Disease Control and Prevention, 2022). Given this rise in mental health concerns and the increase in those seeking care, it is important to find efficient and meaningful ways to understand disorders to then more impactfully diagnose and treat those affected.

Hierarchical Taxonomy of Psychopathology (HiTOP)

Research on disorder dimensionality and comorbidity suggests that many mental disorders are manifestations of a relatively few core underlying dimensions (Krueger & Eaton, 2015). Beginning several decades ago, investigations of common symptoms and behaviors across mental health disorders in children (Chu, 2012; Bilek & Ehrenreich-May, 2012; Weersing et al., 2011; Ehrenreich-May & Bilek, 2011) and in adults (Fairholme et al., 2012; Bernstein et al., 2011), have repeatedly replicated theories to substantiate an underlying cross-cutting

transdiagnostic structure: hierarchical taxonomy of psychopathology (HiTOP) (Krueger, Caspi, Moffitt & Silva 1998; Krueger 1999; Krueger & Markon 2006; Kotov et al., 2017).

The HiTOP model is a data-driven, transdiagnostic, hierarchical classification system. General psychopathology factor (p-factor) is the highest order of classification representing the common underlying influence or dimension of all mental health disorders (Caspi et al., 2014). Among the lower order factors contributing to p-factor are internalizing (INT) and externalizing (EXT) classifications. Although it is important to note that p-factor accounts for the shared variance of other diagnoses (e.g., thought disorders, like mood disorders with psychosis or schizophrenia spectrum) in addition to the shared variance between INT and EXT, the majority of disorders can be classified under the INT and EXT factor umbrellas (Caspi et al., 2014; Kotov et al., 2017). The INT construct is a latent measure incorporating diagnoses categorized as fear (e.g., phobias, OCD, panic) and distress (e.g., depression, anxiety, PTSD) (Kotov et al., 2017). The EXT construct characterizes the comorbidity between antagonistic behavior (e.g., conduct disorder, borderline personality disorder) and disinhibited behavior (e.g., substance abuse) (Kotov et al., 2017).

Studies have shown that the INT-EXT model is stable over time, contributing to an understanding of disorder continuity (Kotov et al., 2021). Additionally, transdiagnostic variance (i.e., captured by the factor) appears to drive disorder persistence. On the other hand, the unique variance of disorders – disorder-specific variance that makes each disorder different from its related disorders – tends to show comparatively low, often negligible, stability. In other words, transdiagnostic factors appear to serve as the primary pathway for homotypic disorder persistence over time (Bromet et al. 2011, Shea et al. 2002). Generalized anxiety disorder, for instance, appears to persist because the INT-EXT model variance saturates the diagnosis, and it

is this transdiagnostic variance that is stable, not the disorder-specific variance of generalized anxiety disorder (Kessler et al., 2011; Eaton, Krueger, Markon et al., 2013). In terms of predicting new related disorder onset, evidence supports the idea that elevation in transdiagnostic INT-EXT model predicts the onset of new disorders longitudinally (e.g., sequential comorbidity) (Kessler et al., 2011). In terms of the distributions for these factors, multiple studies now indicate that these factors are continuously distributed dimensions (vs. liability classes, or dimension-class hybrids) (Eaton et al., 2013).

Currently, the INT-EXT model demonstrated invariance across cultures (Krueger, Caspi, Moffitt, Silva 1998; Vollebergh et al., 2001; Krueger, Chentsova-Dutton, Markon et al., 2003; Slade and Watson 2006; Kessler et al., 2011; Røysamb et al., 2011), gender (Eaton, Keyes, Krueger et al., 2012), ethnicity (Eaton et al., 2013), age (Eaton, Krueger & Oltmanns 2011), sexual orientation (Eaton 2014), and time (Krueger et al., 1998; Vollebergh et al., 2001; Measelle, Stice & Hogansen 2006; Eaton et al., 2011). The stability of this model could help address the lack of constancy across diagnosticians (Regier et al., 2013).

The model also provides insight into how key psychopathological processes map onto shared pathology versus unique components of individual disorders (South, Krueger & Iacono 2011; Conway, Hammen & Brennan 2012; South and Miller 2014). Furthermore, shared genetic and environmental risk factors for experiencing psychopathology are parsimoniously accounted for in this transdiagnostic model, supporting the notion that the INT-EXT model mediates the likelihood of developing additional related diagnoses across the lifespan (Kendler, Prescott, Myers et al., 2003; Kendler, Aggen, Knudsen et al., 2011; Kessler et al., 2011; Lahey, Van Hulle, Singh et al., 2011; Kendler and Myers 2014). Lahey and colleagues (Lahey, Zald, Hakes et al., 2014), for example, found evidence for widespread heterotypic continuity of mental

disorders during adulthood, indicating that mental disorders are not fixed, independent entities. Suggesting, instead, that disorders are robustly related to one another in a correlational structure that is manifested both concurrently and across time.

These findings suggest that underlying transdiagnostic liabilities represent an important avenue for identifying appropriate psychopathology relationships. The latent p-factor, INT, and EXT factors will serve as the primary psychopathology outcome measures we plan to evaluate.

Research Domain Criterion (RDoC): Multidimensional Personality Questionnaire (MPQ) and Neurophysiological Measures

The National Institute for Mental Health (NIMH) developed the Research Domain Criterion (RDoC) system in 2009 as a research framework for investigating mental disorders (NIMH, About RDoC 2009). Although not meant to be used as a diagnostic model, it can provide important biological and cognitive process information that will help develop screening tools, diagnostic systems, and treatment options. RDoC was developed to address problems with the heterogeneity of symptom presentation, bring attention to comorbidity (i.e., transdiagnostic nature) of diagnoses, encourage enrollment of individuals representing the larger spectrum of functioning (i.e., those without "pure" diagnoses), and understand the full spectrum of mental health and illnesses (NIMH, About RDoC 2009; Cuthbert & Insel, 2013). The RDoC model will help address these issues by moving away from reliance on a disorder-based categorical research framework to one that is transdiagnostic and looks at the interactions between biological, physiological, and behavioral components.

There is emerging evidence for studying the HiTOP model using both the behavioral and neurobiological measures of transdiagnostic RDoC constructs (Michelini et al., 2021; Davis et al., 2025). Although all measures are of interest, given constructs that may currently be of

primary interest related to psychopathology (Sainslow et al., 2010; Shankman & Gorka, 2015), the focus will be on cognitive (COG), positive valence (PV), and negative valence (NV) systems. In the current study, a combination of neural and self-report measures will aim to capture these systems and relate them to psychopathology measures. Although COG, PV, and NV may be related to specific measures in the two tasks of interest, as will be detailed, they will be related to both tasks uniformly to ensure a thorough transdiagnostic approach.

Cognitive systems domain addresses various cognitive processes (NIMH, About RDoC 2009), with the cognitive control construct as a primary measure of interest in this study. Cognitive control, a construct of the cognitive systems domain, refers to a system that guides goal-directed behavior by regulating responses, particularly in unfamiliar or demanding situations (NIMH, About RDoC 2009). The go/no-go task evaluated in this study (Logan, Cowan & Davis 1984; described in detail in Methods), is a primary measure to evaluate such cognitive control, namely participants' inhibitory control or ability to suppress automatic responses when they are inappropriate. The EEG data collected during the response feedback portion of the task is intended to measure activity that relates to the three components of cognitive control: response selection: inhibition/suppression, goal selection: updating, representation, and maintenance, and performance monitoring (NIMH, About RDoC 2009). First, participants will need to select and execute responses during the go trials based on this feedback, while selecting to inhibit responses during the no-go trial. Second, they will need to understand, remember, and maintain these goals throughout the task. Finally, as they are provided feedback for their responses, they will need to recognize errors, and track them in order to better adapt behavior in future trials. Similar response selection, goal selection, and performance monitoring functions will likely be utilized by participants in the gambling task (Gehring & Willoughby, 2002; described in detail in

Methods). There may be some relation to the other constructs of COG and these tasks (attention, perception, declarative memory, language, working memory), however, cognitive control in RDoC is likely most closely related to the constraint measure of the MPQ questionnaire reviewed in this study.

Positive valence systems and negative valence systems are most related to the response feedback portion of the gambling task. PV systems are primarily responsible for positive motivational systems, like reward-seeking and reward learning (NIMH, About RDoC 2009). Some constructs and subconstructs of this system, related to the task, are reward anticipation, initial response to reward, and reward satiation. NV systems are primarily responsible for threat, loss, or frustrative non-reward (NIMH, About RDoC 2009). The participants' feedback to both loss and gain trials in the task will serve as a neurobiological measure to interpreting the PV and NV constructs. Although not as apparent, feedback during the go/no-go task can conceptually relate to PV and NV constructs, such as reward learning or frustrative non-reward, respectively. Feedback related to whether a participant got the response correct or incorrect could be similar to that of when they earn or lose a monetary reward, as the processes may relate to each other (Kertzman et al., 2008).

Finally, although not a direct replication of the three RDoC domains mentioned, the Multidimensional Personality Questionnaire (MPQ) self-report measures of constraint (CON), positive emotionality (PEM), and negative emotionality (NEM) (Tellegen, 1982; Patrick et al., 2002; Tellegen and Waller, 2008) will be included as they measure similar underlying theories related to the domains (COG, PV, and NV, respectively). CON is a measure of tendencies toward high versus low behavioral restraint: control – planfulness vs. impulsivity, harm avoidance – intolerance vs. tolerance of risk or danger, and traditionalism – conventionality vs

nonconformity (Patrick & Kramer, 2017). PEM and NEM are counter scales that indicate the disposition to experience positive and negative affect, respectively, across situations and events (Patrick & Kramer, 2017).

A literature search with the collective terms “RDoC”, “HiTOP”, and “review”, provided a well-cited literature review as the top result on the topic, which described how current studies have typically looked at the PV, NV, and COG constructs, only independently, in relation to psychopathology, but not all the factors together (Carcone and Ruocco 2017). Of the forty-eight studies explored in this paper, only ten of them looked at the relationship between multiple constructs, and almost half of the studies (twenty) were only focused on the NV domain. Despite this domain being the most commonly explored, there is still not a singular study that explored this domain in conjunction with other domains (i.e., with PV and COG) (Carcone and Ruocco 2017). Among the studies looking at PV, these papers also focused on individual diagnoses, like schizophrenia and major depressive disorder, or they only looked at individual constructs of the PV domain, like reward learning or approach motivation (Arrondo et al., 2015; Webb et al., 2016; Karalunas et al., 2014). To that same effect, there are studies looking at COG, with the promising point of a considerable four of the six subconstructs having been studied, though still individually and independent of other domains (i.e., Constraint: Lopez-Garcia et al. (2016); Perception: Silverstein et al. (2015); Attention: Kleinman et al. (2015); and Cognitive Flexibility: Francazio and Flessner (2015)). Generally, across domains, this points to an imbalance in studying the constructs and, more importantly, in understanding the overall coordination among the constructs and the domains.

Across the neural tasks, gambling and go/no-go, and the behavioral measure, MPQ, we aim to combine neural and self-report measures as a means to broadly capture the transdiagnostic

RDoC domains of interest as they relate to transdiagnostic psychopathology measures (i.e., INT, EXT, and p-factor).

Importance of relating psychopathology and neural processes

Previous work has indicated several benefits in relating psychopathology to neurophysiology measures. To name a few benefits: identifying treatment targets, identifying risk factors, or identifying neural pathways, which can all lead to a better understanding of both the diagnoses and their relevant neurobiology (Krueger & DeYoung, 2016; Conradt et al., 2021; Cuthbert, 2020; Cuthbert, 2014; Michelini et al., 2021). This points to a growing perspective that HiTOP represents transdiagnostic psychopathology constructs with validated referents in genetics and neuroscience, and that the RDoC domains capture separable contributions to transdiagnostic measures of problem behavior. However, to our knowledge, there is currently no model that more comprehensively connects the two transdiagnostic models. The work that has been done in relating the two models has, either only considered specific diagnoses, or only studied the HiTOP model factors in relation to a specific physiological system (i.e., only a single RDoC domain or system models that were not framed in terms of the RDoC model). Although it is important to study the HiTOP-RDoC relationship in these ways, creating a more holistic picture of the relationship would benefit current understandings of the overall relationships.

Recent meta-analysis reports provide support for the idea that brain activity varies across broad arrays of diagnostic groups, consistent with a p-factor formulation. First, a meta-analysis of 537 task-fMRI studies including patients with schizophrenia, bipolar, major depressive, anxiety, obsessive-compulsive disorders, and controls from 21,427 participants found almost no support for differential contributions of regional brain areas unique to individual diagnoses (Sprooten et al., 2017). Rather, across all diagnoses, modulation of systems was consistent with

NIMH RDoC (Insel et al., 2010) domains (cognitive control, positive valence, negative valence, and social processes). A similar effort that examined 298 experiments including 5,427 patients and 5,491 control participants with diagnoses spanning schizophrenia, bipolar or unipolar depression, anxiety, and substance use disorders found differences between patients and controls across all disorders that were consistent with RDoC domains including PV, NV, and COG systems (McTeague, Rosenberg, Lopez, et al., 2020). These findings suggest that mental disorders may be better understood relative to the transdiagnostic modulation in RDoC systems.

Studies from the Carcone and Ruocco (2017) review, ranged from understanding mental health diagnostic approaches to understanding treatment effects in the context of the RDoC model. This holistic approach can have positive impacts in regards to care. For example, one paper mentioned in this literature review emphasized the ability of pre-treatment physiological (RDoC-related) measures in predicting SSRI treatment response and major depressive disorder symptom remission (Olbrich et al., 2016). By linking the physiological measurements with diagnostic data, there was a benefit of possibly understanding treatment probability better. There are reviews and studies indicating that linking INT (Vaidyanathan et al., 2012; Dickey et al., 2021), EXT (Lutz et al., 2021; Nelson et al., 2011), or p-factor (Bernat et al., 2020; Sprooten et al., 2022; Sprooten et al., 2017) to neurophysiological measures will help identify candidate biomarkers. Overall, mapping the relationship between the RDoC domains and HiTOP model will provide a transdiagnostic perspective of how neurophysiological activity and the disorders relate and allow for the development of new and efficient, diagnostic and treatment methods.

Other work focused on understanding an HiTOP-RDoC relation, but this was usually still done with one INT or EXT diagnosis and one RDoC domain. Additionally not all these studies utilized cost-effective, objective EEG methods. One such study looking at initial responsiveness

to reward attainment (IRRA) and whether it was a predictor of psychopathology in children and their parents utilized a self-report measure of IRRA (Albert et al., 2020). Although a valid method in understanding the construct, it bears the same issue of using only self-report measures to diagnose psychopathology mentioned previously. The proposed multi-task EEG data indexing transdiagnostic constructs will allow for a potentially more objective measurement in conjunction with the self-report measures. This will accomplish the goal of understanding dysregulated behavior across dimensions to then better relate this to transdiagnostic psychopathology data (Cuthbert, 2020). Another such study looked at the relationship between anxiety and executive function, which is categorized as an element of the COG domain. They found that an increase in delta power in the central region and left P3 was negatively correlated with the decrease in executive function (Bong et al., 2020). This paper is a prime example of utilizing EEG methodology to frugally and effectively develop measures of the domains and constructs. The current study partially focuses on addressing the gap that domains or constructs are not being studied cohesively. Utilizing simple and cost-effective EEG measures to develop a multi-domain understanding of the RDoC measures with psychopathology factors will provide a more comprehensive and objective model.

Clinical significance of relating HiTOP and RDoC

As noted earlier, given the prevalence of mental health disorders, it is vital to find proficient methods to treat disorders and transdiagnostic frameworks provide such an efficient and meaningful opportunity to do so. The aims of this study will, respectively, 1) identify direct relationships between self-report measures related to RDoC and HiTOP, and 2) identify direct relationships between HiTOP and RDoC in a neurophysiology context.

A transdiagnostic approach is central to all major components of the current study's focus on interpreting psychopathology, neurobiological processes, and their relationships: diagnosis, intervention, and outcome measures. The project utilizes a clinical diagnosis of various disorders and psychopathology measures, and can aid in providing a better interpretation of potential treatment targets. Recruitment was focused on elevated anxiety sensitivity (AS; Reiss et al., 1986), a transdiagnostic construct (Smits et al., 2019). In addition to AS, outcome diagnostic measures are based on established transdiagnostic models of INT, EXT, and p-factor (self-report measures for INT and EXT, factor score for p-factor). The proposed self-report (MPQ: Tellegen & Waller, 2008; Patrick & Kramer, 2017) and biological measures (EEG measured COG, PV, and NV domains: NIMH, About RDoC 2009; Randau et al., 2024) have all shown sensitivity across and to psychopathology diagnoses, consistent with RDoC ideals, and are selected to target known systems. Comprehensively, the utilization of both models (HiTOP and RDoC) is essential as RDoC provides a means of categorizing and identifying the underlying biological systems that can be used to relate to the underlying transdiagnostic mechanisms of treatment targets and find treatment targets for psychopathology.

Additionally, given that p-factor, INT, and EXT are stable and sensitive to change, they will present as meaningful potential treatment targets (Kessler et al., 2011; Eaton, Krueger, Markon et al., 2013). For instance, there has been growing interest in the application of the p-factor towards intervention work. This motivated a recent effort to assess whether the p-factor is stable and associated with change in problems over time. A recent analysis of 10-year follow-up data from 5001 participants in the US National Comorbidity Survey found that the p-factor was stable over time and that changes in diagnostic status were better accounted for by changes in the p-factor, relative to only modeling separate INT and EXT factors (Gluschkoff, Jokela, and

Rosenström, 2019). These findings suggest that the p-factor model could account for direct relationships, noting future analyses of any indirect relationships with INT and EXT, can offer an effective means of addressing underlying characteristics related to psychopathology.

Finally, the NIMH description of RDoC does not currently include comprehensive EEG-related measures for the various domains and constructs of RDoC. The current work will help facilitate that objective. EEG task data can be utilized to identify and build RDoC measures (i.e., COG, PV, NV). For instance, previous work has found a link between P300 reduction and the p-factor in an oddball paradigm, indicating a biological link to the psychopathology factor. (Bernat et al., 2020). This is just one task and one measurement, but offers insight to hypothesize that other tasks would also have such links between RDoC domains and HiTOP diagnoses and factors (Michelini et al., 2021; Hawn et al., 2022; Chetcuti et al., 2025). By building on these ideas, HiTOP variables (i.e., transdiagnostic self-report data) and viable EEG-related neurophysiological measured variables, which can be categorized in the RDoC domains, can be used to develop treatment targets for psychopathology that is grounded in the underlying biological mechanisms.

EEG data processing

Given the meta-analytic approach (to be described in the "Meta-analytic approaches" section), EEG measures were included inclusively across the two tasks. Although more measures could be included, all midline electrodes, all frequency measures, and various data reduction methods were utilized to provide a more wholistic approach. This level of analysis allows for both a broad, big picture review, as well as the potential for a more specific and detailed breakdown as appropriate. This section will focus on describing EEG techniques and background on why they were used.

Brief review: Analytical techniques

The current study employs various EEG-related analytical techniques. Given the complexity of these techniques, a brief review detailing the meaning and purpose of each method seemed beneficial to include. This section will summarize the different overall analysis types and power analysis types being used. Time-frequency analyses will be described more, but to note now, the frequencies are as follow: delta (0-4 Hz), theta (4-8 Hz), alpha (8-12 Hz), beta-1 (13-20 Hz), beta-2 (20-30 Hz), and gamma (30-60 Hz).

Analysis types can be based on amplitude, inter-trial phase synchrony or inter-channel phase synchrony. Amplitude analysis examines the magnitude of EEG activity, usually focused on event-related potentials (ERP; brief time-locked electrical activity patterns), or oscillatory power changes associated with cognitive events (e.g., during a behavioral task, like the gambling paradigm) (DaSilva, 1999). Inter-trial phase synchrony (ITPS) analysis measures the consistency or reliability of the EEG signal across multiple trials of the same stimulus or task, with the goal of reflecting neural response timing stability (van Diepen & Mazaheri, 2018). Inter-channel phase synchrony (ICPS) analysis assesses the functional connectivity between EEG signal activations recorded from different electrode sites, which is meant to evaluate neural network interactions (Aviyente et al., 2017; Obukhov et al., 2018). Amplitude analyses will be conducted across all frequencies, with ITPS and ICPS evaluations limited to delta and theta for this preliminary study analysis.

Power analyses could be total power analysis, evoked power analysis, or induced power analysis. Total power activity analysis encompasses all brain activity (stimulus-related and background activity, or evoked and induced, respectively), while evoked power activity analysis is specifically linked to activity during the presentation of a stimulus or event (David et al.,

2006). For reference, induced power analysis, though not reviewed in this study, is not time-locked to the stimulus (i.e., modulated by the stimulus, but not triggered by the stimulus) (David et al., 2006). For lower frequency amplitude analyses, like for delta and theta frequencies, total power analysis is preferred because these frequencies are often stable and sustained activity over a long period of time and total power will capture the brain states (e.g., general neural workload or emotional state), which these frequencies are more linked to, during the task and feedback process, more effectively (Harmony, 2013; Scheeringa et al., 2008). For relatively higher frequency amplitude analyses, like for alpha, beta-1, beta-2, and gamma frequencies, given their more transient and dynamic nature, evoked power analyses will better capture the specific cognitive or sensory events, which these frequencies are generally more linked to, that occur during the task and feedback portion (David et al., 2006). For ITPS and ICPS analyses (which were only conducted with delta and theta frequency bands in this study), regardless of frequency, an evoked power analysis is preferred to better capture the trial and channel synchrony as related to the actual presentation of the stimulus or event.

Data reduction techniques will be employed to interpret the data: time-frequency principal components analysis (TF-PCA) and a windowed time frequency analysis (TF-WIN). Time-frequency analyses will be detailed in the next section ("Advantages of time-frequency approaches"). A principal components analysis will identify components of the data (employed with the delta and theta bands in the current study) that explain the greatest percent of variance and will be helpful in reducing the dimensionality of the data for analysis, as has been shown with previous work in my group (Buzzell, Niu, Aviyente, and Bernat, 2022). This includes all the amplitude, ITPS, and ICPS analyses as related to total and evoked power, as mentioned. For the remaining frequency bands (alpha, beta-1, beta-2, and gamma), a windowed time frequency

approach will be employed, for many of the same reasons highlighted above related to an evoked power approach (i.e., the frequency bands exhibit a more transient and dynamic nature).

Advantages of time-frequency approaches

Time-domain approaches will allow for the extraction of ERP measures; however, traditional time-domain approaches will not parse apart overlapping frequency measures, and therefore can limit the understanding of each measure's meaning in the context of each respective task. To address this issue, a time-frequency (TF) approach will be utilized (Gröchenig, 2001; Boashash, 2015).

Time-frequency analysis will address an important challenge in measuring the different ERP measures that may overlap partially in time, complicating standard time-domain methods of response quantification. To better isolate distinctive stimulus-locked components, we will employ time-frequency analysis, which provides separation of ERP components that overlap in time but have differing frequency characteristics (Bernat et al., 2008; Bernat et al., 2011). For example, FN and P300 operate at different frequencies, but have some temporal overlap. P300 is largely delta activity and FN is largely theta activity (Bernat et al., 2008; Bernat et al., 2015; Watts et al., 2018; Ellis et al., 2018). Other instances of such overlap include: theta and delta bands underlying conventional ERP components such as the error-related negativity (ERN) (Bernat, Williams & Gehring 2005; Hall, Bernat, & Patrick, 2007), the N200/P300 in go/no-go (Harper, Malone & Bernat 2014) and oddball target (Bernat, Malone, Williams et al., 2007), and FN/P3 elicited during gambling feedback (Bernat, Nelson, Steele et al., 2011; Nelson, Patrick, & Bernat, 2011).

Important to note, the necessity for this approach expands between delta and theta frequency bands. Previous work in the lab has indicated the need for a complete review of all

frequency bands to interpret similarities and differences in neural activity. In the same gambling task used in this study, differences in components of interest were noted across lower (delta and theta) and higher (alpha, beta-1, beta-2, and gamma) frequency bands in the context of suicidality. There were notable neural activity similarities between the lower frequencies (delta and theta), separate from neural activity similarities among the three higher frequencies (beta-1, beta-2, and gamma), with alpha showing unique relationships to suicidal ideation different from all other frequency measures, which showed relationships with suicidal behavior (Pavuluri et al., 2023). Noting these similarities and differences provides a more comprehensive and detailed understanding of neural activity. Many studies that looked at EEG activity across frequencies have noted similar variations, generally, but also of specific interest, in neurobehavioral (Glass & Riding, 1999; Pino & Romano, 2022) and psychopathology related studies (Desarkar et al., 2022; Rommel et al., 2011; Jiang et al., 2023).

Meta-analytic approaches

Meta-analyses have been used across various domains of research since before they were first coined as a methodological approach in 1976 (Glass). Generally, they were developed to summarize and more accurately interpret the vast number of studies that fields, such as psychology, were producing in order to better grasp and simplify the full range of knowledge being produced (Glass, 1976). They have also become increasingly common and necessary in order to simplify and better interpret findings from the large number of studies produced in the present-day field of neuroscience, which is growing exponentially (Altimus et al., 2020).

Before interpreting these approaches in the context of the current study, it is important to establish some background related to meta-analytic approaches in the context of both psychopathology and neurophysiology. This understanding will better highlight the potential of

such analyses in advancing overarching theories, guiding future research, and refining experimental approaches.

Relevant to this study, meta-analytic approaches have been key to supporting and advancing the development of theoretical models, such as HiTOP and RDoC. One confirmatory factor analysis review of 35 studies with a total of 120,956 participants substantiated the HiTOP model both in the overall framework and placement of diagnoses (Ringwald et al., 2023). A similar systematic review (221 citations) related to RDoC NV systems supported the important clinical utility of the framework in evaluating antidepressant effectiveness. The review noted that the antidepressants helped treat NV system-related concerns such as acute threat, potential threat, and loss, while also identifying areas of antidepressant ineffectiveness related to acute fear (Hui, 2020). Finally, meta-analytic approaches have worked to summarize vast neurophysiological findings related to psychopathology. Of note, neuroimaging studies have faced concerns related to false positive reporting of activations (likely 10-20%; Wager et al., 2007), but meta-analytic techniques can work to minimize such concerns through an exhaustive and encompassing review of the literature. A review of methodological concerns related to meta-analyses conducted on neuroimaging data has posed concerns related to reliability, as well, but by ensuring appropriate statistical checks in review implementation, these concerns can be avoided and minimized (Uttal, 2012). This is promising given the knowledge to be gained from meta-analytic reviews. EEG findings related to psychopathology have demonstrated consistency in meta-analytic reviews related to both internalizing and externalizing disorders (Vanhollebeke et al., 2022; Rudo-Hutt, 2014). Such comprehensive reviews can help establish cohesiveness and clarity in the field to then conduct more substantiated and novel experiments.

Meta-analytic approaches can aid in guiding new research, while refining existing experimental and analytic methods. These approaches help look at the current field of research and identify central findings across several studies and/or note key areas that should be addressed in future research. For instance, a meta-analytic review of suicidal risk and protective factors in the context of RDoC helped identify both commonly studied factors, while addressing the need to branch out from solely examining those characteristics (Glenn et al., 2018). Notably, one proposed improvement when relating HiTOP and RDoC models is incorporating multimethod models (i.e., from various measurement methods; Patrick et al., 2019). Meta-analyses can work to compile data from several studies and methods, and the current work aims to expand on that to include multimethod models in a single study, as well. Compilation of such research related to these psychology and neuroscience transdiagnostic frameworks and neuroimaging also provides the benefit of identifying aberrations to then develop new treatments and locate treatment targets (e.g., depression targets in fMRI and EEG studies: Keren et al., 2018). One of the key benefits of meta-analyses is the summarizing of vast amounts of data. This summarization of commonly used research techniques has been utilized to interpret best practices for research methods from self-report measures (Achenbach et al., 2005) to neurophysiology measures (Fox et al., 2014; Müller et al., 2018).

Given these previous studies and their utilization in the field, we felt the methods could be expanded to within study analyses. As alluded to previously, the usage of these meta-analytic approaches in a single dataset is limited, if used at all. However, many studies engage participants in large questionnaire and task batteries. This leaves researchers with abundant data and information that spans across several different psychological and cognitive features, which are generally related (re: transdiagnostic constructs). In order to better interpret this feature in a

single context as they relate to each other, an experimental within-subjects and within-study meta-analytic approach was considered for this current study.

Data considerations

Although meta-analytic approaches generally refer to evaluating summary statistics from several studies (e.g., meta-regressions using correlation value data derived from 100 different studies), they can also be used to conduct meta-statistics on single datasets (e.g., meta-regression using a single correlation matrix from a single study). The latter is far less common since meta-analyses usually involve multiple studies (Garg, et al., 2008; Williams & Tiedens, 2016; Pool et al., 2016). However, the latter meta-analytic approach, with a single study or as few as two studies ("mini-metas"; Goh et al., 2016) can be conducted with some considerations in mind – related to the overall data structure and related to each individual statistical analysis (McNemar, 1962; Valentine et al., 2010; Goh et al., 2016; Hansen et al., 2022). In other words, this would involve identifying statistical approaches to conduct and interpret meta-analyses on a single correlation table – a relatively novel approach.

The following considerations and methods will be detailed and explored more in "Methods" ("Meta-analysis" subsection), but describing them here provides important background on this attempt at a unique meta-analytic method to relate psychopathology and neurophysiology measures.. Before delving into these considerations, it is important to succinctly understand the current data structure being used in this study. The data is collected from a single study, but not from a single task or a single questionnaire. The data contains values from three collected questionnaires (three subscale totals from the Multidimensional Personality Questionnaire-Short Form for COM, PEM, and NEM; Inventory of Depression and Anxiety Symptoms average score for INT; and Externalizing Spectrum Inventory-Brief Form total

proportion score for EXT), a factor variable computed from two of the questionnaire's data (p-factor computed from an EFA of INT and EXT questionnaire data), and EEG collected ERP data from two separate behavioral tasks (gambling and go/no-go tasks). The questionnaire and factor data (six variables) were then correlated (Pearson's r correlation analysis) with the ERP measures from the two behavioral task data. Although all data used to compute the correlations is from a single study with the same participants, there are several different measures involved in the data analysis, and a meta-analytic approach could help assess this multi-task protocol.

First, correlation tables would need to be transformed using a Fisher's z transformation method in order to ensure data is normally distributed (Fisher, 1915). Raw correlations generally exhibit a non-normal distribution. This non-normal distribution is more commonly the case for small sample correlation tables (Bonett & Wright, 2000), but to minimize data concerns related to a potential non-normal distribution and related to this novel meta-analytic approach, it would be beneficial to apply Fisher's z transformation to the large correlation table before conducting meta-statistical analyses (Fisher, 1915; Borenstein et al., 2021). This is additionally a common practice in the case of needing to combine correlations when aggregating data from several studies in meta-analyses (Hedges & Olkin, 1985). In order to ensure best practice analytical methods, Fisher's z transformation will be applied to the correlational data.

Given the novelty of this analysis, it seemed especially important to establish resilient evaluation criteria and ensure assumptions are met for the analyses we intend to conduct (Cafri et al., 2010). The main analyses of interest were a one-sample t-test, paired samples t-test, multiple linear regression, and a univariate general linear model.

Second, for all analyses listed, a review of effect sizes is often seen as more valuable given that a large sample size is more prone to Type I error (Graf & Bauer, 2011; Shreffler &

Huecker, 2023). Of particular interest, studies have mentioned that including effect sizes along with typical p-value reporting will provide more clarity in disciplines like biology, psychology and neuroscience, and especially in the context of meta-analyses (where large datasets are more common; Nakagawa & Cuthill, 2007; Nakagawa, 2011; Nieuwenhuis, 2011; Hentschke & Stüttgen, 2011). It has also been noted that it is important to consider effect sizes in the context of the discipline and study question in addition to considering the general cutoff criteria established for effect sizes (Bakker et al., 2019). There are additional arguments that even a Bonferroni correction to the p-value, alone, may not be sufficient to counter the concerns associated with p-values in large datasets given how small a p-value could falsely be reported as (Nakagawa, 2004).

Third, although many of the same assumptions stand for each statistical test in the meta-analytic context as they do outside of this context (e.g., linearity, continuous data, normality, etc.), the data structure warrants some assumptions be more clearly assessed, especially in the context of the multiple linear regression analysis. A review of normality, multicollinearity, and outliers/influential points was particularly important given that the data was a correlation table, all six of the HiTOP and MPQ relevant variables were correlated with the same ERP measures, and the small range of correlational data, respectively.

Although the correlational values were Fisher's z transformed (Fisher, 1915; implying the data would be normally distributed), this is not a guarantee that the newly transformed data will display perfect normality, especially given special considerations attributed to larger datasets (Efron, 1982; Berry & Mielke, 2000; Öztuna, 2006; Mudholkar & Chaubey, 2007; Mameli et al., 2012; Biu et al., 2020; Demir, 2022). The normalization will reduce skewness and stabilize variance, but the transformed data may still deviate from a normal distribution.

Multicollinearity was another important consideration, especially given the meta-analysis data table was built on correlational data where the independent variables in the model were correlated with the same ERP variables and all the variables were collected as part of a single dataset. This indicated a possibility of multicollinearity, so it seemed important to verify. Within-study correlations, though leaving possibility for multicollinearity in this context, have been mentioned as a key statistic to review for in meta-analytic approaches, and another reason to consider relationships within the dataset in this study (Riley, 2009). Additionally, a key consideration in regression analyses is multicollinearity between the independent variables that are predicting the dependent variable (Vatcheva et al., 2016), usually considered when correlations are greater than .70 or a variance inflation factor (VIF) greater than 5 (Kim, 2019).

Finally, outliers and influential points should be reviewed in meta-analytic approaches (Viechtbauer & Cheung, 2010). This is especially true when considering the more compact range of correlation values in the data (e.g., -.25 to +.25 correlation range), the impact of outliers or influential points could be even more impactful given the limited explanatory power in the predictors (Osborne & Overbay, 2004). A thorough evaluation of these potentially influential points is necessary to ensure that the effects are seen regardless of the points and in the context of this correlational data (Stevens, 1984; Aguinis et al., 2013).

After considering the data transformation and evaluation methods above, we aim to complete a reasonable within-subjects or within-study meta-analysis. Building from this, though, individual meta-analyses can also later be reviewed similar to traditional meta-analyses to evaluate several within-subjects studies conjunctively.

Chapter 2: Study Overview

Using an archival dataset, RDoC-representative measures from the Multidimensional Personality Questionnaire-Short Form – constraint, positive emotionality, and negative emotionality – were examined as predictors of individual psychopathology measures – internalizing (Inventory of Depression and Anxiety Symptoms), externalizing (Externalizing Spectrum Inventory-160), and p-factor (factor computation from internalizing and externalizing scores). As a brief overview, the dataset consisted of electrophysiological measures across different tasks (EEG recordings) and self-report measures (individual questionnaires) both collected at the same time point. The data was correlated (i.e., EEG measure correlated with self-report measure) across different tasks, frequencies, time windows, and electrodes to then conduct a meta-analytic-like review with a multivariate correlation table.

Study design

A total of 281 adult participants were recruited near a southeastern American university. All participants underwent a baseline questionnaire battery assessment and a baseline computer task battery while electrophysiology recording was conducted. The two computer tasks that are evaluated here are the gambling and go/no-go tasks. The three questionnaires that are evaluated are the Inventory of Depression and Anxiety Symptoms, Externalizing Spectrum Inventory-160, and Multidimensional Personality Questionnaire-Short Form. These were selected to evaluate a possible relationship between psychopathology measures and relevant cognitive measures, both in the context of the neurophysiology data (re: multivariate correlation table analysis).

Motivation for current work

A comprehensive study from our research group conducted across several datasets found that, in self-report data, the relationships between psychopathology and personality domains are expected – psychopathology measures relate negatively with constraint and positive emotionality, but relate positively with negative emotionality (Mannava, 2025). Further, this investigation contributes to the use of transdiagnostic models and mechanisms as a means to understand these constructs. Building upon this ideology, we aim to interpret the relationships in this dataset, not only at the self-report level, but additionally through the incorporation of neurophysiology as a means to further include the transdiagnostic Research Domain Criteria.

Additional work in the lab that has motivated this study relates substance use to the HiTOP model (Pavuluri et al., 2022). Though conceptually different, this work relatedly focuses on understanding underlying psychopathology measures (internalizing and externalizing) and their relationships to the higher-order measure of p-factor. This study revealed a negative relationship between internalizing factors (fear and distress) and polysubstance use when accounting for p-factor, and a positive relationship between antagonism (a sub-factor of the EXT factor) and polysubstance use when accounting for p-factor. Given this, and other existing research that note the need to interpret psychopathology related to other constructs, this study focused on interpreting these relationships in different steps and levels. Although the current dataset was collected looking primarily at INT disorders, there is a presence of EXT disorders, as evidenced through both the diagnostic and self-report data collection, as well as the presence of shared diagnosis data.

The addition of neurophysiology data adds both the addition of biological factors for consideration, as well as the potential for a unique approach to review such data. Previous work

in the lab has indicated relationships between psychopathology and ERP measures. For instance, the majority of the P300 variance (in an oddball task) related to EXT is also related to shared variance between INT and EXT (i.e., a primary indicator of EXT is p-factor) (Bernat et al., 2020). Although the oddball task is not directly reviewed here, we will be able to conduct analyses across other cognitive tasks with future goals in mind to include this, and other tasks, for more comprehensive and diverse analyses.

Finally, the psychopathology and neurophysiology relationships detailed in the hypotheses are expected based on previous research conducted in the lab (Mannava, 2025), as well as previous work done in the field. Cognitive control (Goschke, 2014; McTeague et al., 2016; McTeague et al., 2017) and positive emotionality (Carl et al., 2013; Santos et al., 2013; Kendall et al., 2015) are expected to relate negatively with psychopathology measures, while negative emotionality (Boschloo et al., 2013; Watson & Naragon-Gainey, 2014; Osman & Wood, 2018) is expected to relate positively with psychopathology measures.

Summary and current aims

AIM 1: To confirm the relationship between transdiagnostic HiTOP and MPQ measures at the self-report data level.

Hypothesis 1: Constraint and positive emotionality will relate negatively to p-factor, and negative emotionality will relate positively to p-factor.

Hypothesis 2: INT and EXT will show the same pattern of relationships with the MPQ measures as p-factor does. However, these relationship patterns will be modulated to likely show reduced strengths of the same expected patterns in the relationships.

AIM 2: To review the same MPQ-HiTOP relationships, through a meta-analytic exploration, after integrating objective neurophysiology data.

Hypothesis: Multivariate correlatives of HiTOP and MPQ domains, both independently correlated with neurophysiology data, will present similar relationships (between HiTOP and MPQ) in both relative strengths and directionalities as the relationships seen in the self-report data.

Significant impact: This work will provide a novel contribution to understanding relationships between emerging transdiagnostic neurobiological and psychopathology frameworks.

Chapter 3: Methods

Participants

A total of 281 adult participants were recruited near a southeastern American university (Institutional Review Board Protocol #00000446). Recruitment focused on clinical criteria rather than a focus on recruiting a typical community sample (recruitment criteria will be detailed more in the "Procedure" section). A questionnaire battery and a multi-task protocol, which included a gambling task and a go/no-go task, was administered. For the gambling task, 273 participants remained since eight participants were excluded during data preprocessing due to incomplete data (five participants missing data files) or unusable data (three participants removed because >25% of trials rejected due to unreadable EEG artifacts). For the go/no-go task, 259 participants remained since twenty-two participants were removed during data preprocessing due to incomplete data (eight participants missing data files) or unusable data (nine participants with unreadable files and five participants due to issues with catcodes, electrodes, or epochs). From the participants remaining across both tasks, two participants were missing all demographic data. After merging the tables to only include participants found across both task datasets, who also had complete demographic data, 256 participants (mean age=35.63, SD=16.03; 55.9% female; 60.5% White, 24.4% Black, 2.3% Asian, .4% Pacific Islander, .4% Native American, 11.2% Other; 50.8% <$25,000, 36.1% >$25,000 and <$100,000, 10.1% >$100,000; 50.0% family history of mental health; note 2.3% missing data for income levels and 3.5% missing data for family history of mental health) remained for analysis.

The participants primarily met for internalizing disorders, with 68.0% meeting for depression disorder, 30.9% meeting for general anxiety disorder, 32.8% meeting for

posttraumatic stress disorder (PTSD), and 12.1% meeting for obsessive compulsive disorder (OCD) (recruitment criteria detailed in "Screening appointment" section). Overall, 87.1% of participants met for at least one of the following internalizing disorders: depression, anxiety, PTSD, OCD, eating, panic, specific phobia, social phobia, hoarding, and agoraphobia. Diagnosis data also looked at the externalizing measures of drug abuse and dependence, with 41.8% meeting criteria for at least one drug use (alcohol, cannabis, amphetamine/stimulant, hallucinogen/PCP, opioid, cocaine, and sedative, hypnotic, or anxiolytic). With the mentioned INT and EXT diagnosis data, 8.2% met for neither disorders, 37.1% met for both disorders, 50.0% met for only INT disorders, and 4.7% met for only EXT (drug use in this case) disorders.

The above values are based on a diagnosis of the participant by trained doctoral level therapists, with diagnoses determined by the Structured Clinical Interview for DSM-V (First, Williams, Karg, & Spitzer, 2015). All results were reviewed by a licensed clinical psychologist to verify accurate diagnosis (high inter-reliability, e.g., >80%, kappa=.77, Timpano & Schmidt, 2013). The participants were assessed for having ever (both current and past diagnoses assessed) meeting for either a primary or other (secondary, tertiary, etc.) diagnosis for the mentioned disorders. Since diagnosis data did not include other EXT-related disorders outside of drug use, self-report data was prioritized to represent both internalizing and externalizing measures to allow for better consistency and accuracy in representation of measure characteristics.

Procedure

Screening appointment

Participants who endorsed anxiety or mood disorders on an initial phone screen were invited to complete a more comprehensive screening appointment and participate in a clinical

trial. In order to increase generalizability of findings, the aim was to include a broad range of participants and minimize exclusion criteria. Inclusionary criteria were English speakers over the age of 18, who showed evidence of cognitive risk for psychopathology as indicated by scoring at or above the community sample mean on the Anxiety Sensitivity Index-3 (Taylor et al., 2007) or the Interpersonal Needs Questionnaire (Hill & Petit, 2013; Van Orden, Cukrowicz, Witte, & Joiner, 2012). Exclusionary criteria were significant medical illness that would prevent the completion of intervention (e.g., respiratory disorders, stroke, epilepsy), serious suicidal intent (e.g., intent that would indicate a need for hospitalization or immediate treatment), current substance dependence, current or past psychotic-spectrum disorders, or uncontrollable bipolar disorder.

During the appointment, participants underwent a diagnostic interview (First et al., 2015) with a trained therapist, and a thorough suicide risk assessment (Joiner et al., 1999). After the diagnostic interview, participants completed self-report measures to help determine study eligibility and inform diagnostic decisions. If participants were deemed ineligible based on the screening appointment they were thanked for their time and given relevant community referrals based on their needs. All participants went through an informed consent process before the start of the study.

Baseline appointment

Participants completed a battery of self-report questionnaires. The questionnaires included the Multidimensional Personality Questionnaire-Short Form, Inventory of Depression and Anxiety Symptoms, and Externalizing Spectrum Inventory-160. Upon completion, they were scheduled for a baseline neurophysiology assessment and provided with monetary compensation. The neurophysiology assessment involved a battery of tasks, including a gambling task and a

go/no-go task, which were assessed in this analysis (additional tasks included emotional picture viewing task, visual oddball task, and resting state task). Since treatment changes were not the focus and because the study aimed to test relationships at a single time point, only baseline questionnaire and neurophysiology data was used for the purposes of this study.

EEG data acquisition

All neurophysiological data was collected in a dimly lit sound attenuated room, where E-prime version 2.0 (Schneider, Eschman, & Zuccolotto, 2002) was used to present the computer tasks. Experimental stimuli were presented on a 21-inch Dell high-definition CRT color monitor, centrally placed in front of participants at a viewing distance of 100 cm.

Neurophysiological data was recorded using a BrainVision 96-channel actiCap (sintered Ag-Ag/Cl; international 10-20 system) as well as a 24-bit battery-supplied active channel amplifier. Horizontal electrooculogram activity was recorded from electrodes placed on the outer canthus of both eyes, while vertical electrooculogram activity was recorded from electrodes placed above and below the left eye. Impedances were kept below 10 kΩ. EEG signals were vertex referenced during recording, and referenced to average mastoid signals offline (electrodes TP9 and TP10). Recordings were collected using a 500Hz sampling rate, analog .05 to 100Hz bandpass filter, and digitized at 1000 Hz using BrainVision PyCorder (Brain Vision Analyzer).

Questionnaires

Inventory of Depression and Anxiety Symptoms (IDAS)

The Inventory of Depression and Anxiety Symptoms (IDAS) is a 64-item self-report questionnaire developed to provide a more nuanced assessment of mood and anxiety disorders (Watson, O'Hara, Simms, et al., 2007). The items were coded 1 to 5 (1=Not at all, 2=A little bit,

3= Moderately, 4=Quite a bit, 5=Extremely). Two items needed to be reverse scored. IDAS consists of the following subscales: appetite gain, appetite loss, dysphoria, general depression, insomnia, ill-temper, lassitude, panic, social anxiety, suicidality, trauma, and well-being.

Since each of the subscales consisted of different item numbers and because certain items captured characteristics of multiple subscales, an average of the subscales was taken, so all subscales are equally contributing and equally captured in the internalizing variable. In this study, the items displayed a strong internal consistency of α=.93. A review of Cronbach's alpha changes, given item removals, indicates that no item is meaningfully changing the alpha value (greatest increase is .003 and greatest decrease is .003).

Externalizing Spectrum Inventory (ESI)

The Externalizing Spectrum Inventory (ESI) is a 160-item self-report measure developed to assess externalizing psychopathology symptoms (Patrick et al., 2013). It is the brief-form version of the original 415 item full-form questionnaire (Krueger et al., 2007). ESI consists of the following twenty-three lower-order subscales: alcohol problems, alcohol use, alienation, blame externalization, boredom proneness, lacks dependability, destructive aggression, drug problems, drug use, lacks empathy, excitability, fraud, lacks honesty, impulsivity, irresponsibility, major problems, major use, physical aggression, lacks planful control, problem impulsivity, rebelliousness, relational aggression, and theft, and three higher-order subscales: general disinhibition, callous aggression, and substance abuse.

The items were coded on a four-level true/false scale (0=False, 1=Somewhat false, 2=Somewhat true, 3=True). Lacks planful control, lacks dependability, lacks honesty, rebelliousness, lacks empathy, drug use, and alcohol use were reverse scored. A proportion of the items from the total 160 items was computed to only include scores with 75% or greater valid

items. Finally, an ESI total proportion score, ranging from low to high externalizing (0 to 1, respectively) was computed for each participant. In this study, the items displayed a strong internal consistency of α=.97. A review of Cronbach's alpha changes, given item removals, indicates that no item is meaningfully changing the alpha value (no change seen for any items to the .000 place).

Multidimensional Personality Questionnaire (MPQ)

Multidimensional Personality Questionnaire-Short Form (MPQ) was administered and is a 155 item questionnaire that was developed to assess personality traits, such as temperament, affect, and social behavior (Tellegen, 1982; Patrick et al., 2002; Tellegen and Waller, 2008). Items were generally coded as true/false (0/1) or alternative B/alternative A (0/1; selecting one of two scenarios), and thirty-eight items were reverse scored.

The questionnaire has three broad higher-order scales that are reflective of the similar RDoC constructs: positive emotionality (PEM; RDoC: positive valence), negative emotionality (NEM; RDoC: negative valence), and constraint (CON; RDoC: cognitive systems). For each scale, a person must have completed 75% of the items, and if they have, but they've not completed all items on the scale, their scores are prorated by the number of items that were completed divided by the total number of items on the scale. The values were then transformed, using a t-score normalization (Patrick, Curtin, & Tellegen, 2002), to recode values of certain ranges to other values, so that all variables are on the same scale (22 to 78 response values, low to high, respectively).

For all three subscales, the same items were included, but the items were regression weighted to predict the three individual factor scores from the full version of the MPQ with the greatest precision compared to a factor analysis on the normative. In the study, the questionnaire

items that contributed to all has acceptable internal consistency coefficient of α=.78. A review of Cronbach's alpha changes, given item removals, indicates that no item is meaningfully changing the alpha value (greatest increase is .003 and greatest decrease is .008).

p-factor

General psychopathology factor or p-factor was measured by conducting an exploratory factor analysis of the IDAS average score and the ESI total score (following methods presented in previous work, Caspi et al., 2014. Principal axis factoring method was used to extract one factor that accounted for 61.44% of the variance in the data, with an eigenvalue of 1.23. The factor loading of each variable was moderate at .50. Overall, the factor is interpretable as a measure of the shared variance between internalizing and externalizing, or as a general psychopathology factor.

Behavioral tasks

Gambling

The gambling task (Figure 1) is a modified version of the Gehring and Willoughby (2002) task, in which a participant would be presented with two monetary values on the screen and be given feedback about whether they won or lost money after selecting one of the values. The four different combinations of values could be of equal or different values (i.e., 5 and 5, 5 and 25, 25 and 5, 25 and 25) in terms of cents with a random pattern (to prevent the possibility of prediction) of winning or losing. Feedback was presented for 1000ms after selection by the chosen box turning either red or green (with the color indicating winning or losing being counterbalanced across participants), and the unselected box would turn either the other color or the same color between red and green (i.e., it is possible that both values could have been losing

selections or that both could have been winning selections). Participants completed 7 blocks (each block contained two sets of these 16 trial types, randomly ordered) of 32 trials each, for a total of 224 trials. After each block was completed, participants received feedback regarding their win/loss ratio for that particular block. There was a version of this task that included pictures (same categories listed in the go/no-go section) between blocks.

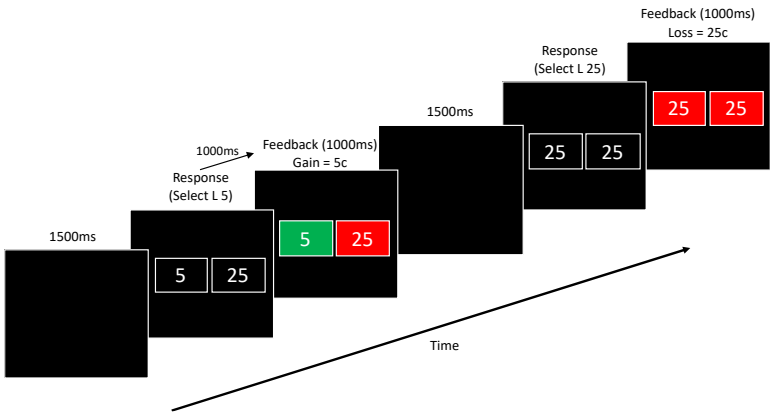


Figure 1. Sequence of stimuli and feedback events in the gambling task.

Go/No-go

The go/no-go task (Logan, Cowan & Davis 1984; Figure 2) is a stimulus-response selection task where participants decide whether to execute or inhibit a response. Participants were presented with two different white letters (e.g., S-F) displayed sequentially on a black background, and instructed to press the right or left button corresponding to the letter that appeared (go trials; e.g., S=left, F=right). However, when the stimulus repeated itself participants were asked to not press any button for the repeated stimulus (no-go trials; e.g., do not press for the second S or F in S-S or F-F, respectively). No-go trials were pseudo-randomly interspersed throughout the task such that one, three, or five go trials always preceded a no-go trial. Seven blocks of twenty-four trials each were completed, with eighteen go trials (75%) and six no-go trials (25%) in each block. Stimulus duration was 296 ms, the response window was 1150 ms,

and the inter-trial-interval was 900 ms. Participants also completed a practice version of the task consisting of 20 practice trials with different letters, but no electrophysiology data was recorded during the practice. Participants were provided with their overall accuracy after each block. Associations between the letter (e.g., S or F) and right/left button presses were counterbalanced across participants.

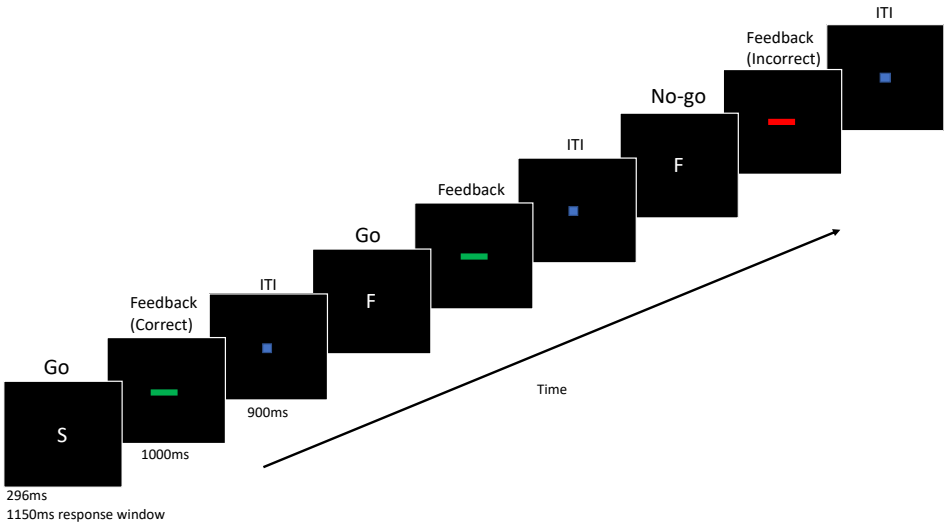


Figure 2. Go/no-go task sequence and feedback example.

Data processing

Preprocessing

EEG data preprocessing was conducted using a custom MATLAB script (version 9.3.0.713579 [R2017b]; The MathWorks, Inc.) utilizing both original and EEGLAB functions (Garland et al., 2021). ERP analysis was performed in the Psychophysiology Toolbox (PTB) developed by Dr. Edward Bernat (Buzzell et al., 2022).

The preprocessing steps are set up to clean and correct the EEG datafiles. The output set of preprocessed mat files that can then be analyzed through the PTB. Epochs of 3,000 ms were taken from 1,000 ms pre to 2,000 ms post-stimulus with a 500 ms pre-stimulus baseline

correction and 100 ms post-stimulus baseline correction. Electrodes were re-referenced to averaged mastoid sites. Data were corrected for ocular artifacts (Gratton et al., 1983) and downsampled to 256 Hz using the MATLAB resample function (Mathworks, Inc.), which applied an anti-aliasing filter during resampling. The respective triggers for each task were set in the respective task preprocessing script (i.e., loss and gain trials in the gambling task and go and no-go trials in go/no-go task).

A low-pass filter of 50 Hz was applied to the continuous data, and then ERP epochs were created as described previously. Epochs were computed separately for loss, gain, go, and no-go trials. For each individual, epochs were ranked according to number of extreme (>±150 mV) data points across all channels, and the worst 5% of epochs were removed. In addition, individual channels were interpolated across all data if they exceeded the threshold of 5 SDs in the domains of kurtosis and activity probability. After baseline (500–100 ms before stimulus) correction, each epoch was evaluated separately and channels with extreme (>±150 mV) data points were interpolated only for that epoch, while epochs with more than two bad channels were rejected and removed from the data. A final visual inspection was conducted to remove epochs with unusual artifacts. ERP component scores were extracted and exported for further statistical analyses.

Data reduction

Time-frequency analysis: Correlation table ERP measures

Using the PTB, principal components analyses and windowed time-domain analyses were conducted on frequency filtered TF surfaces of the EEG/ERP data (see Buzzell et al, 2022 for a detailed treatment of this approach). The data analyzed are time-locked to the gain and loss

feedback or the go and no-go feedback, 100 ms after a selection is made. Data were analyzed separately in gambling and go/no-go tasks, by frequency, electrode, condition, power, and principal component or time window analyses. The analyses include amplitude level analyses (all frequencies) and inter-channel phase synchrony level analyses (delta and theta, only).

For the 256 participants, correlations were conducted between the ERP measures and the individual difference measures. For the ERP measures, the following measures were included for each task: delta-time frequency principal components analysis (TF-PCA) evoked power amplitude review (126 measures), delta TF-PCA inter trial phase synchrony (ITPS) review (126 measures), delta-TF-PCA inter-channel phase synchrony (ICPS) review (288 measures; F6 and FCZ seed electrodes), theta-TF-PCA evoked power amplitude review (84 measures), theta TF-PCA inter trial phase synchrony review (126 measures), theta-TF-PCA ICPS review (192 measures; F6 and FCZ seed electrodes), alpha-window time frequency domain (TF-WIN) total power amplitude review (168 measures), beta-1-TF-WIN total power amplitude review (168 measures), beta-2-TF-WIN total power amplitude review (168 measures), and gamma-TF-WIN total power amplitude review (168 measures). For each of the measures, there were condition and condition differences (loss, gain, loss-gain differences for the gambling task and go, no-go, and go-no-go differences for the go/no-go task) and time windows for the TF-WIN analyses (250 ms cuts). The same analyses were run across tasks.

To separate and isolate frequency activity relevant to each component, the data were pre-filtered, before applying the TF-PCA, using the following filters for each frequency: delta – 0 Hz high-pass and 4 Hz low-pass filter; theta – 4 Hz high-pass and 8 Hz low-pass filter; alpha – 8 Hz high-pass and 12 Hz low-pass filter; beta-1 – 13 Hz high-pass and 20 Hz low-pass filter; beta-2 –

20 Hz high-pass and 30 Hz low-pass filter; and gamma – 30 Hz high-pass and 60 Hz low-pass filter.

TF-PCA was used to identify distinct frequency activity for delta and theta frequency bands (for details, see Bernat et al., 2005; Bernat et al., 2008; Bernat et al., 2011; Nelson et al., 2011; Bernat et al., 2015; Buzzell et al., 2022; and Watts et al., 2018). Scree plots identified the ideal number of components to review for each frequency band. Three PCs were identified for delta across all analyses, two PCs were identified for theta evoked power amplitude analyses and total power inter-channel phase synchrony analyses, and three PCs were identified for theta total power inter-trial phase synchrony analysis. The delta PCs, in order, accounted for 62.26%, 9.81%, and 7.85% of the variance in the gambling data. The delta PCs, in order, accounted for 59.36%, 10.31%, and 7.85% of the variance in the go/no-go data. The theta PCs for the two-factor solution, in order, accounted for 51.51% and 12.24% of the variance, with the third factor accounting for 7.35%, in the gambling data. The theta PCs for the two-factor solution, in order, accounted for 39.61% and 12.18% of the variance, with the third factor accounting for 7.49%, in the go/no-go data.

For higher frequencies (alpha, beta-1, beta-2, and gamma), TF-WIN approach was used because a PCA approach is generally not as sensitive to the transient and complex activity that higher frequencies tend to display (David et al., 2006). 250 ms window cuts were used for each of the higher frequencies given the transient and dynamic nature of the higher frequencies to capture the data more appropriately.

Data analytic methods

Statistical analyses were conducted using RStudio (Posit team, 2025) and IBM SPSS Statistics (Version 27) software. Correlation table computation, data descriptive statistics for the

correlation data, data assumption and distribution analyses for the correlation table (normality, homoscedasticity, and hexbin plots), one-sample t-test analyses on the correlation tables, and multiple linear regression analyses on the correlation table were all conducted in R. Univariate general linear model analyses on the correlation table, paired sample t-tests on the correlation table, data descriptive statistics for the self-report data, and the multiple linear regression analyses for the self-report data were all conducted in SPSS.

Self-report data descriptive statistics and multiple linear regression analyses were conducted. Listwise deletion methods were used to remove data for both analyses.

Electrophysiology activity data was exported using Matlab and an export script developed by Dr. Spencer Fix. After the data was exported it was merged, on subject number, with self-report data for the following measures: MPQ subscale total scores (constraint, positive emotionality, and negative emotionality), IDAS (internalizing) average score, ESI (externalizing) total proportion score, and p-factor (shared variance between IDAS and ESI).

Meta-analysis

Data set-up

A full sample correlation table contained 9,684 correlation values total (three MPQ subscales for CON, PEM, and NEM, an IDAS average variables, ESI total proportion variable, and a p-factor score were each correlated with 1,614 ERP components). Any analyses with time window cuts (alpha, beta-1, beta-2, and gamma) were at 250 ms only. Descriptive statistics were computed for the full sample correlation table (Mishra et al., 2016), and subsequent statistical analyses (e.g., multiple linear regression) are only provided for this full sample correlation table.

Since the data was a correlation table, there are no missing values that would warrant description of a removal process.

The correlations related a single ERP component with a single psychopathology or cognitive measure total score. For example, an ERP component that was from the gambling task, gain condition, theta frequency, FCZ electrode, evoked power amplitude reduction, principal component 1 would be correlated with p-factor psychopathology measure. For reference, another possible correlation is an ERP component that was from the go/no-go task, no-go condition, gamma frequency, CPZ electrode, total power amplitude reduction, 0-250 ms time window correlated with the PEM measure.

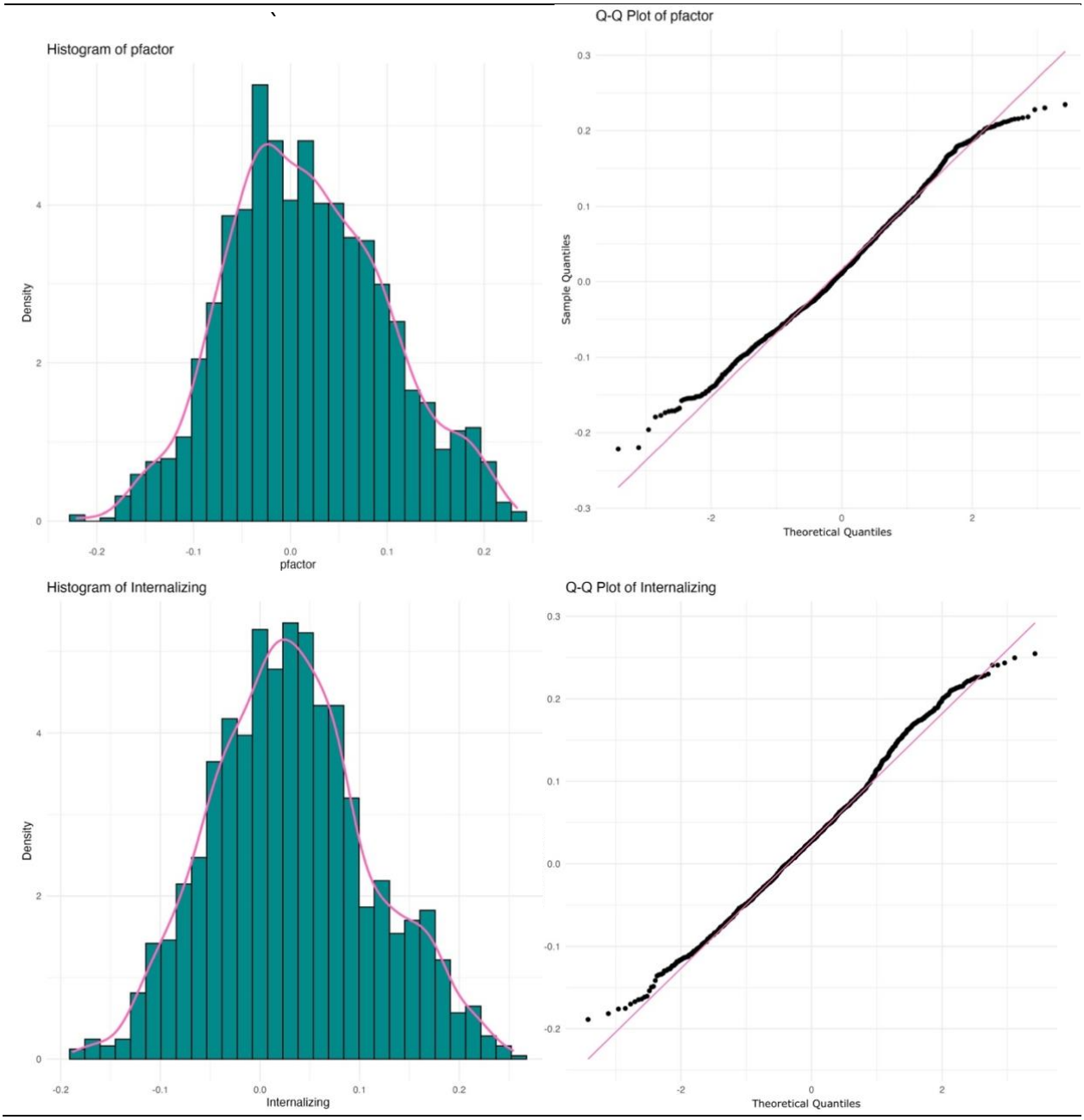
From these Pearson's r correlation tables, Fisher's z transformations were applied to perform the subsequent calculations (Fisher, 1915). All analyses were conducted on Fisher's z transformed data and all figures are representative of Fisher's z transformed data as it has been reviewed as having the least bias from sampling distribution skew (i.e., presents a more normal distribution), more stabilized variance (i.e., minimizes heteroscedasticity), and is beneficial when testing further hypotheses utilizing such multivariate correlational data (Corey et al., 1998). This is particularly helpful for correlation data to ensure reasonable symmetry of positive and negative values and ensuring they are treated similarly in further analyses, thereby increasing the reliability and validity of those tests.

Grouping variables were included after correlation table computations and transformations for the following groups in order to perform preliminary univariate general linear models: task (gambling and go/no-go), condition (loss, gain, no-go, go), frequency (delta, theta, alpha, beta-1, beta-2, gamma), and electrode (FZ, FCZ, CZ, CPZ, PZ, POZ, OZ, FCZ seed ICPS data, F6 seed ICPS data).

For each analysis, as needed, statistical values were converted back to Pearson's r for review (Borenstein, Hedges, Higgins, & Rothstein, 2009; Kim & Bottema-Beutel, 2019). Notably, given the large N and range of the correlation values in the data, variations between the raw correlation values, Fisher's z transformed values, and converted back Pearson's r values were minimal and essentially nonexistent (i.e., rounded values of statistics that needed to be converted back were identical to the transformed values). Therefore, all results tables and appendices contain the transformed results for consistency.

Also given the large N, all statistical significance is limited to values that have $p<.05$ and any trend level relationships are not considered beyond reporting in the tables. Furthermore, although significance is considered and measured in all analyses, more emphasis is placed on effect size statistics (e.g., Cohen's d for t-test, R^2 for multiple linear regression, or partial eta-squared for a univariate general linear model; Cohen, 2013) and relationships observed.

Given the data structure being analyzed, normality and homoscedasticity tests were conducted on the dependent variables of interest (internalizing, externalizing, and p-factor) to ensure conducting further statistical tests was viable. As a note, although statistics are provided relative to these assumptions (Appendix A), for such large datasets, a visual inspection (Figures 3 and 4) has been reviewed as generally more appropriate given the major impact even small deviations may have on these tests. Even tests seen as more effective for larger sample data, like the Kolmogorov-Smirnov normality test are affected by such large size and even small deviations from the normal distribution tend to provide significant results, so in this case it is suggested to visually evaluate histogram graphs (Biu et al., 2020, Öztuna et al., 2006; Demir, 2022; Kim, 2013; Mayers 2013).


Histogram of pfactor
Density
pfactor
Q-Q Plot of pfactor
Sample Quantiles
Theoretical Quantiles
Histogram of Internalizing
Density
Internalizing
Q-Q Plot of Internalizing
Theoretical Quantiles

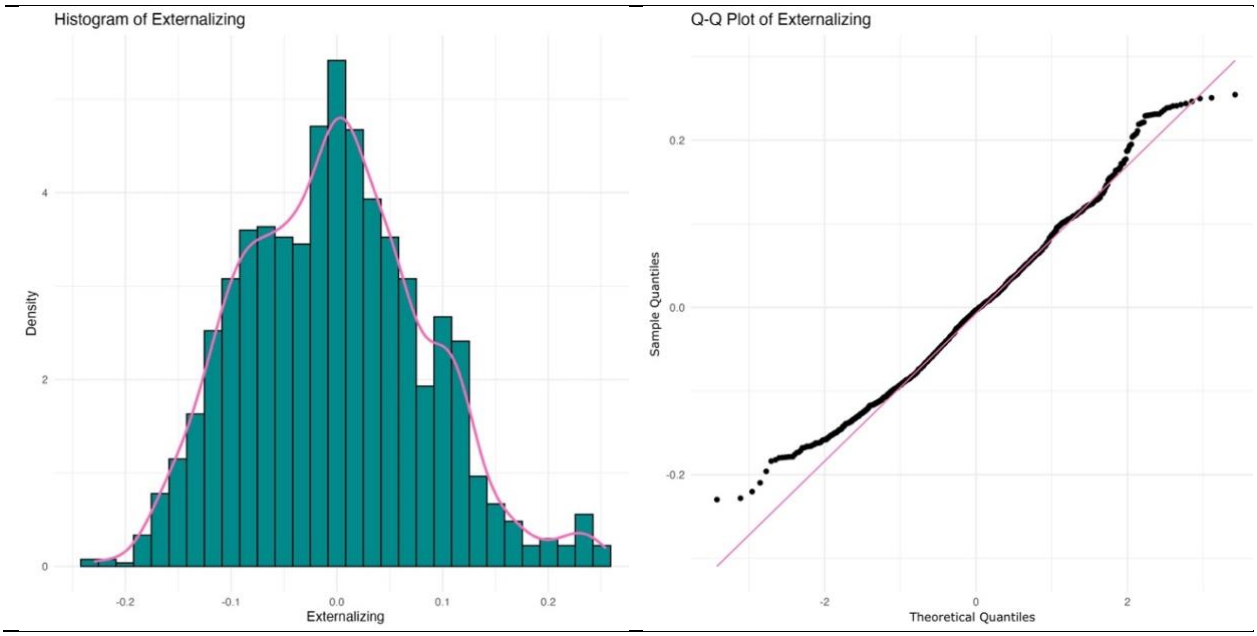
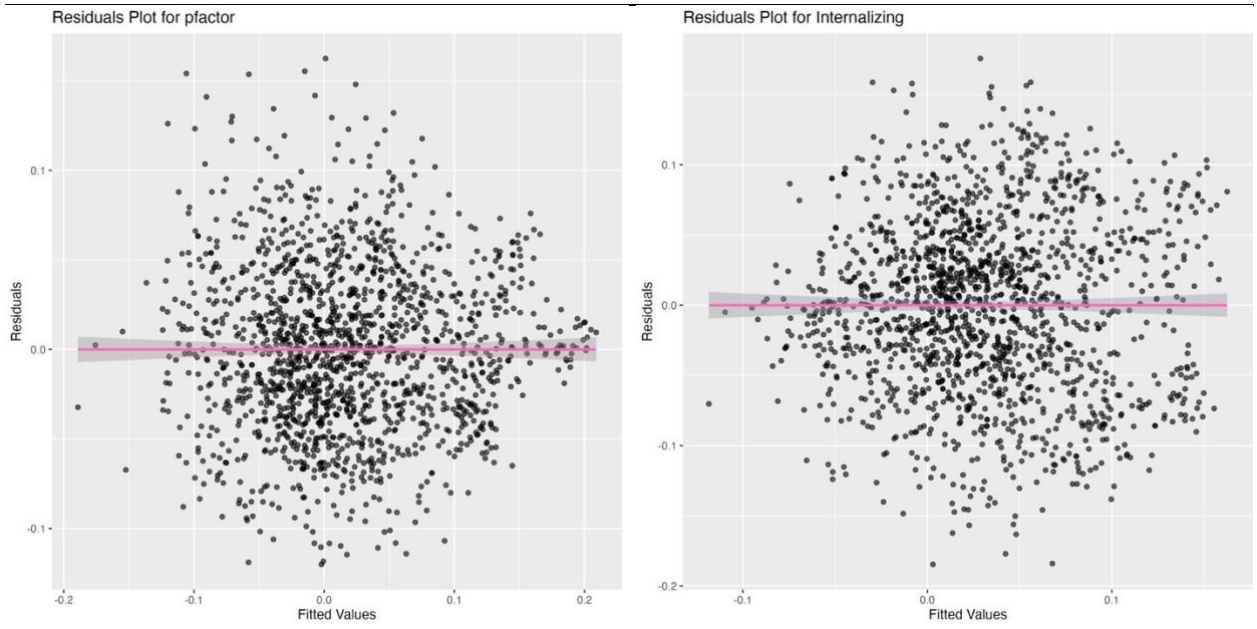


Figure 3. Normality tests for the dependent variables of interest.
Note. The visual tests verify that the transformed data will meet assumptions to conduct further analyses. Visual inspections of the histograms and Q-Q plots indicate relatively stable normal distributions for all variables.



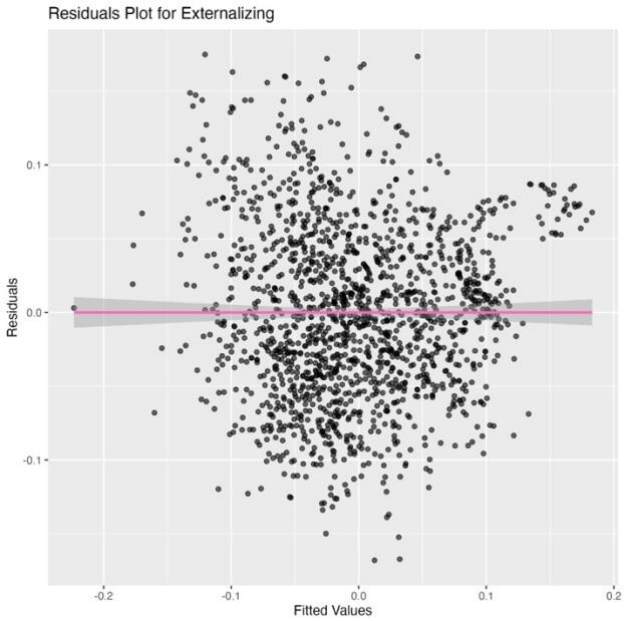


Figure 4. Homoscedasticity tests for the outcome variables of primary interest.
Note. The regression model has constraint, positive emotionality and negative emotionality as the independent variables and the respective dependent variables are indicated above each plot (p-factor, internalizing, and externalizing). The graphs indicate that there are no strong patterns in residuals, no major curvature in the residuals, and no strong funnel shape, but rather, a relatively random distribution of points. Although some mild outliers are present, they do not seem to have a strong influence or form a strong trend.

One-sample t-test

One-sample t-tests against zero were conducted on the Fisher's z transformed data for all 6 variables of interest. As mentioned, this, and other tests were less focused on the significance of the values.

Particularly for this analysis, it is important to note what a no difference or a difference from zero result could represent. First, no difference from zero could mean a more even distribution of positive and negative correlations rather than only implying no meaningful correlations at all. This means that, though the correlations are only minimally significantly different from zero in the negative direction, this does not mean that there are not meaningful correlations in the data at all; rather, that there may be an even distribution of positive and

negative correlations (with slightly more negative correlation values), or even that there are more positive correlations, but the negative correlations are generally stronger. Second, a difference from zero could represent stronger or more correlations in one direction indicating a possible direction of relationship between neurophysiology and the self-report data. For instance, if there is a positive t-value when comparing the correlation averages of internalizing-ERP to zero, this may indicate a different meaning than if there was a negative t-value.

This test aimed to understand these overall base relationships in the correlations. For more information on the data in the context of the above two point mentioned – primarily, to better understand the correlation values and their distribution – hexbin plots were included in Appendix B.

All analyses were conducted using R software, and significance was determined using an alpha level of .05, though effect sizes were considered more meaningful given the data structure and size. Effect sizes were reported using Cohen's d (general criteria: .20=small effect, .50=medium effect, .80=large effect).

Regression analyses

Multiple linear regression analyses were run on the Fisher's z transformed data. Since the regressions were conducted on Fisher's z transformed data, β values were already standardized. The regressions reported are limited to the full sample correlation data and to the primary psychopathology variables of interest. To expand, three regression analyses with internalizing, externalizing, and p-factor as the respective dependent variables were conducted, and all three analyses had the same personality domain independent variables – CON, PEM, and NEM.

Normality and homoscedasticity were visually tested above for the dependent variables of interest before conducting these regression analyses and did not indicate concerns with the

variables. Below is a test of multicollinearity relevant to the independent variables (Figure 5). Multicollinearity, generally with a standard cutoff at r >|.70|, indicated no multicollinearity concerns, and as a further statistical note, no VIF values met criteria (all values were <2, with ≥5 generally being an indication of multicollinearity) (Kim, 2019). Additionally, ten regression simulations of ten randomized correlation data, all with correlation values of a similar range of values to the actual correlation table, were conducted. This was done to examine whether the results occurred by chance (due to possible multicollinearity), or if there would be a meaningful difference between the actual data and the simulated data regression results. The simulated regression results indicated no meaningful results, different from the actual data.

Finally, tests related to influential statistics were conducted to ensure further interpret and validate the results. Several common attributes and concerns regarding the independent variables (CON, PEM, and NEM) warranted conducting an influence analysis to ensure the validity of the models: the possibility of influential points, outliers, and multicollinearity, in addition to the unique data structure. Influence plots for each of the three regression analyses were conducted based on all of the following: Cook's d, leverage values, and studentized residuals. Appendix C contains independent plots for each of these stats and a cumulative influence plot.

In addition to the plots, regressions were conducted to 1) cumulatively remove any points that may be considered influential (i.e., removing all influential points at once), and 2) remove points that may be influential in a stepwise fashion (i.e., remove most influential point, then remove top two most influential points, then remove top three most influential points, etc.). The first method compared the original regression results to the new regression results to note any differences, the second method looked at each step to note differences by a single point more specifically. Both methods tested each of the three measures of influence individually to be as

comprehensive as possible, and capture more points than be may be captured when looking only at data points that met all three criteria. Influence diagnostics were conducted to examine the robustness of regression models. Cook's distance (computed cutoff: > .0025), leverage (computed cutoff: > .0045), and studentized residuals (standard cutoff values: moderate ±2; extreme ±3) were used to identify potentially influential or extreme observations. The summary table for this analysis is provided in Appendix C, as well.

All analyses were conducted using R software, and significance was determined using an alpha level of .05, though effect sizes were considered more meaningful given the data structure and size. Effect sizes were reported using R^2.

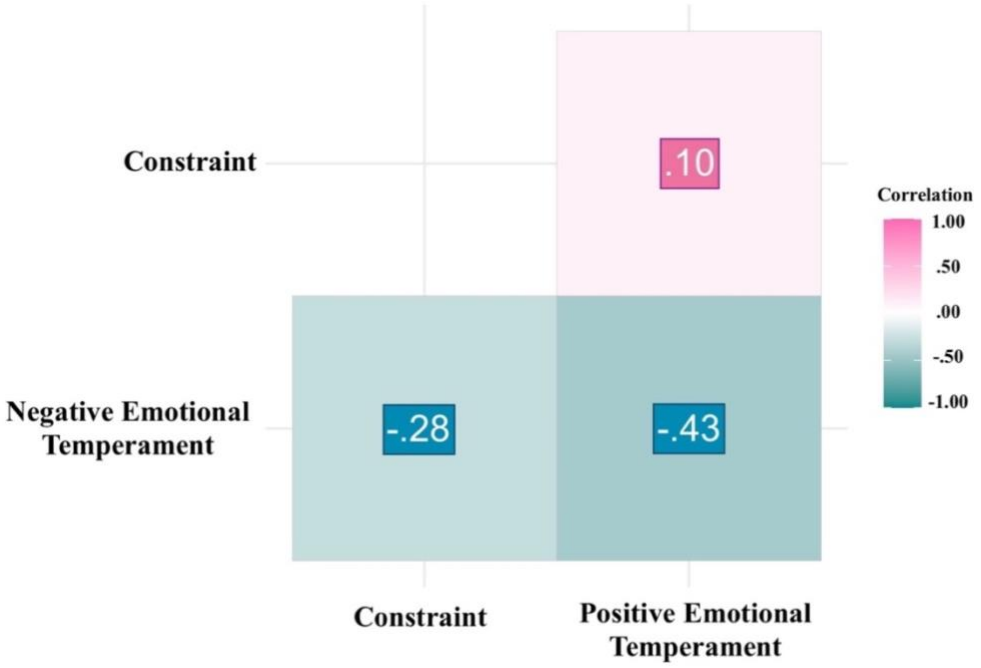


Figure 5. Multicollinearity review of the independent variables.
Note. Pearson's r correlations between independent variables of the multiple linear regression analysis.

ERP group differences: General linear model and paired samples t-tests

Given the complexity of the group data and unequal group sizes, a univariate general linear model was selected to interpret main effects. Main effects are detailed in results, while post-hoc analyses are detailed in Appendix D. These general linear model (GLM) analyses were conducted to examine the correlated neurophysiology and behavioral effects as determined by the groups – frequency, electrode, and condition – on each of the dependent variables (internalizing symptoms, externalizing symptoms, and p-factor).

Task group analyses were conducted separately, as paired sample t-tests, due to its potential collinearity with condition groups. Although the task data is somewhat identified through the condition differences, in order to interpret overall task differences, the t-test was conducted and indicated significant differences in the correlation results.

This univariate GLM approach was selected to assess the main effects among these categorical predictors while controlling for within-group variance. For significant main effects, Tukey's HSD post-hoc tests were conducted to identify pairwise differences between group levels while adjusting for multiple comparisons.

Frequency showed consistently high effect sizes, so additional paired-samples t-test analyses were conducted on the frequency group to compare each psychopathology against the other (internalizing vs externalizing, internalizing vs p-factor, and externalizing vs p-factor) across the six frequency measures (delta, theta, alpha, beta-1, beta-2, and gamma).

For frequency and electrode differences, estimated mean marginal plots were run to visualize the patterns across the psychopathology measures by either group. Additional plots for electrode groups were run by frequency group to further interpret these patterns.

All analyses were conducted using SPSS software, and significance was determined using an alpha level of .05, though effect sizes were considered more meaningful given the data structure and size. Effect sizes were reported using partial eta-squared (general criteria: .01=small effect, .06=medium effect, .14=large effect).

Chapter 4: Results

Self-report measures: Descriptive statistics

Before reviewing regression results for the questionnaire data, descriptive stats were reviewed for all of the self-report measures. Additionally, although, 256 participants had full data for ERP measures and demographic data, data was missing for the self-report questionnaires as mentioned in Table 1 (removal processes for missing data is detailed in Methods).

The MPQ measures ranged from a possible minimum of 22 to a possible maximum of 78 (an average score would equal 50). CON scores had an observed range that spanned 22 to 71 (M = 46.32, SD = 8.75), indicating that participants generally reported moderate levels of CON. PEM scores had an observed range of 22 to 69 (M = 44, SD = 10.97), indicating a generally moderate, although slightly lower than average level, of positive emotional functioning. NEM scores had an observed range of 31 to 78 (M = 59.66, SD = 9.12), indicating moderate susceptibility to negative emotions that is also slightly above an expected median value.

INT had a possible minimum of 7 and possible maximum of 35, with observed values between 8.92 and 27.17 (M = 16.32, SD = 3.53). This mean may indicate that, most individuals in this sample reported low to moderate levels of INT symptoms, the presence of higher scores in some participants suggests that a subset of individuals may be at increased risk for INT disorders.

EXT was a proportion computation and ranged from 0 to 1, with observed scores spanning .04 to .72 (M = .27, SD = .15). The relatively low average EXT scores in this sample suggest that most participants exhibit few behavioral disinhibition tendencies, though the range of values indicates some individual differences in EXT traits that trend towards higher scores.

Finally, p-factor scores were derived using factor analysis, with an observed range of -1.16 to 1.80 (M = .00, SD = .63). Because factor scores are standardized, a score of 0 represents the sample mean, rather than representing a balanced or even level of psychopathology. The observed range suggests that while most participants may cluster around the mean, some individuals exhibit higher general psychopathology risk (1.80 as max value), warranting further investigation of the present psychopathology.

Table 1. Self-report: Descriptive statistics.

Variable	*N*	Missing	Mean	SD	Min	Max
MPQ Measures						
CON	230	26	46.32	8.75	22	71
PEM	230	26	44	10.97	22	69
NEM	230	26	59.66	9.12	31	78
HiTOP Measures						
INT	229	27	16.32	3.53	8.92	27.17
EXT	231	25	.27	.15	.04	.72
p-factor	229	27	.00	.63	-1.16	1.80

Self-report measures: Regression – Personality domains predicting psychopathology

Multiple regression analyses were conducted to confirm whether the relationships between HiTOP and MPQ measures exist as hypothesized and as seen in previous similar studies. The results generally confirm the expected directionality of relationships: both CON and PEM will be negatively related to the HiTOP variables, and NEM will be positively related to the HiTOP variables. Standardized beta values and r-squared values are provided to indicate a measure of effect size for these results.

For the p-factor (Table 2), CON and PEM both showed significant negative relationships, with $\beta = -.25$ ($p<.001$) and $\beta = -.17$ ($p<.001$), respectively, aligning with the expected negative associations. In contrast, NEM exhibited a significant positive relationship with $\beta = .54$ ($p<.001$), supporting the hypothesis that NEM would be positively associated with the p-factor. The model

explained 47% of the variance (R^2 = .47, adjusted R^2 = .46), indicating the importance of MPQ traits, especially NEM, in capturing individual differences in general psychopathology.

For INT (Table 3), PEM showed a significant negative effect (β = -.22, p<.001), while NEM demonstrated a significant positive effect (β = .47, p<.001), both consistent with expectations. However, CON did not show a significant effect on INT (β = -.09, p=.26). The model accounted for 33% of the variance (R^2 = .33, adjusted R^2 = .32), supporting the notion that MPQ traits, particularly NEM, contribute meaningfully to INT symptomatology.

For EXT (Table 4), CON was significantly negatively related to EXT (β = -.31, p<.001), as hypothesized. NEM also showed a significant positive relationship with EXT (β = .38, p<.001), supporting the positive association expected. However, PEM did not show a significant effect on EXT (β = -.05, p=.43), contrary to the hypothesized negative relationship. The model explained 30% of the variance (R^2 = .30, adjusted R^2 = .29), indicating that while MPQ traits are relevant to EXT symptoms, their predictive power may be slightly weaker than they are for INT and general psychopathology.

Table 2. Self-report regressions: p-factor as the dependent variable.

Variable	β	95% CI	t	SE
Intercept	-.01	[-.10, .09]	-.13	.05
CON	-.25***	[-.35, -.16]	-5.10	.05
PEM	-.17***	[-.27, -.07]	-3.47	.05
NEM	.54***	[.44, .64]	10.85	.05

† .10≤p<.05,*p≤.05, **p≤.01, ***p≤.001
Note. F(3, 224) = 64.93***, R^2=.47, Adj. R^2=.46. Standardized beta reported for all values, except the intercept value.

Table 3. Self-report regressions: INT as the dependent variable.

Variable	β	95% CI	t	SE
Intercept	-.01	[-.12, .10]	-.14	.05
CON	-.09	[-.20, .02]	-1.59	.06
PEM	-.22***	[-.33, -.12]	-4.07	.06
NEM	.47***	[.36, .58]	8.49	.06

† .10≤p<.05,*p≤.05, **p≤.01, ***p≤.001
Note. $F(3, 224) = 35.97$***, R^2=.33, Adj. R^2=.32. Standardized beta reported for all values, except the intercept value.

Table 4. Self-report regressions: EXT as the dependent variable.

Variable	β	95% CI	t	SE
Intercept	-.00	[-.11, .11]	-.07	.06
CON	-.31***	[-.43, -.20]	-5.48	.06
PEM	-.05	[-.16, .07]	-.81	.06
NEM	.38***	[.27, .49]	6.59	.06

† .10≤p<.05,*p≤.05, **p≤.01, ***p≤.001
Note. $F(3, 226) = 31.51$***, R^2=.30, Adj. R^2=.29. Standardized beta reported for all values, except the intercept value.

Meta-analysis: Descriptive statistics

The descriptive statistics for the full sample correlation data between the ERP components and each of the six MPQ and HiTOP variables were computed (Table 5). The descriptive data is for the Fisher's z transformed data table. As a note, mention of all MPQ and HiTOP variables for all meta-analysis results are in the context of these six variables correlated with the ERP measures (e.g., p-factor in these sections is p-factor correlated with the ERP measures, and different from the self-report sections mention of p-factor).

The minimum and maximum correlation values indicate meaningful correlations in psychology and neuroscience, with the highest absolute value correlation at .25. NEM, INT, and p-factor trend more positive (M=.02, M=.03, M=.02, respectively) in their mean correlation relationships with ERP, while PEM trends more negative (M=-.03) in its mean correlation relationship with ERP. CON and EXT show a mean value of .00 in their correlational

relationships with ERP data. All variables show a relatively similar, and low spread of data (SD min =.07 and SD max= .09).

The second half of the descriptive statistics table is after computing the absolute value correlational values from the Fisher's z transformed correlation table, to provide a summary of the magnitude of the relationships regardless of directionality (Table 5).

Table 5. Meta-analysis: Original and absolute value correlation value descriptive statistics.

		Original Correlations				Absolute Value Correlations			
Variable	N	Mean	SD	Min	Max	Mean	SD	Min	Max
MPQ Measures									
CON	1614	.00	.07	-.24	.22	.05	.04	.00	.24
PEM	1614	-.03	.08	-.24	.22	.07	.05	.00	.24
NEM	1614	.02	.08	-.24	.22	.07	.05	.00	.24
HiTOP Measures									
INT	1614	.03	.08	-.19	.25	.07	.05	.00	.25
EXT	1614	.00	.09	-.23	.25	.07	.05	.00	.25
p-factor	1614	.02	.08	-.22	.23	.07	.05	.00	.23

Meta-analysis: One-sample t-test

T-tests against zero were run on the Fisher's z transformed correlation data to identify overall patterns (Table 6). Given that the data is correlation values, the output provides insight into not only the presence of this significant difference from zero, but also the direction of the difference, and the strength of that direction. For more information, distributions of the data for these six variables of interest are presented as hexbin plots in Appendix B.

CON correlations are different from zero in the negative direction ($t(1613) = -2.12$, $p = .053$), suggesting an inverse relationship, but with a very small effect size (Cohen's $d = -.05$). PEM correlations are different from zero in the negative direction ($t(1613) = -13.34$, $p < .001$), suggesting a strong inverse relationship with a small-to-moderate effect size (Cohen's $d = -.33$). NEM correlations are different from zero in the positive direction ($t(1613) = 7.83$, $p < .001$),

suggesting a positive relationship with a small effect size (Cohen's d = .19). INT correlations are different from zero in the positive direction (t(1613) = 15.72, p < .001), suggesting a positive relationship with a small-to-moderate effect size (Cohen's d = .39). EXT correlations are not different from zero (t(1613) = -1.41, p = .16), indicating no direction trend (Cohen's d = -.04). P-factor correlations are different from zero in the positive direction (t(1613) = 8.39, p < .001), suggesting a positive relationship with a small effect size (Cohen's d = .21).

Table 6. Meta-analysis: One-sample t-test against zero.

Variable	Mean	SD	*t*(1613)	CI	Cohen's *d*
MPQ Measures					
CON	.00	.07	-2.12*	[-.01, .00]	-.05
PEM	-.03	.08	-13.34***	[-.03, -.02]	-.33
NEM	.02	.08	7.83***	[.01, .02]	.19
HiTOP Measures					
INT	.03	.08	15.72***	[.03, .03]	.39
EXT	.00	.09	-1.41	[-.01, .00]	-.04
p-factor	.02	.08	8.39***	[.01, .02]	.21

† .10≤p<.05,*p≤.05, **p≤.01, ***p≤.001

Meta-analysis: Regression - Personality domains predicting psychopathology

Multiple regression analyses

Multiple linear regression analyses were conducted to confirm both the hypothesized relationship between HiTOP and MPQ variables in the ERP data, as well as confirm alignment with the patterns seen in the self-report data. Overall, the relationships are expected in relation to p-factor, however, CON was not significantly negatively related to INT and PEM was not significantly negatively related to EXT, as expected (detailed below). Standardized beta values and r-squared values are provided to indicate a measure of effect size for these results.

The p-factor has a positive relationship with NEM and a negative relationship with CON and PEM (Table 7). The overall model was statistically significant, $F(3, 1610) = 1179.65$, $p < .001$, with $R^2 = .69$ and an adjusted $R^2 = .69$, indicating that the model explains 69% of the variance in the p-factor. The relationships are as follows: CON, $\beta = -.15$, 95% CI [-.18, -.11], $t = -8.53$, $p < .001$; PEM, $\beta = -.26$, 95% CI [-.29, -.22], $t = -15.59$, $p < .001$; and NEM, $\beta = .66$, 95% CI [.63, .69], $t = 40.72$, $p < .001$.

INT has a positive relationship with NEM and a negative relationship with PEM (Table 8). The overall model was statistically significant, $F(3, 1610) = 356.65$, $p < .001$, with $R^2 = .40$ and an adjusted $R^2 = .40$, indicating that the model explains 40% of the variance in INT. The relationships are as follows: CON, $\beta = .05$, 95% CI [.00, .09], $t = 2.02$, $p = .04$; PEM, $\beta = -.39$, 95% CI [-.43, -.35], $t = -17.61$, $p < .001$; and NEM, $\beta = .37$, 95% CI [.33, .41], $t = 16.99$, $p < .001$.

EXT has a positive relationship with NEM and a negative relationship with CON (Table 9). The overall model was statistically significant, $F(3, 1610) = 620.61$, $p < .001$, with $R^2 = .54$ and an adjusted $R^2 = .54$, indicating that the model explains 54% of the variance in EXT. The relationships are as follows: CON, $\beta = -.27$, 95% CI [-.31, -.23], $t = -12.34$, $p < .001$; PEM, $\beta = .00$, 95% CI [-.04, .04], $t = -.16$, $p = .87$; and NEM, $\beta = .68$, 95% CI [.64, .72], $t = 32.79$, $p < .001$.

Table 7. Meta-analysis regressions: p-factor as the dependent variable.

Variable	β	95% CI	t	SE
Intercept	.00	[.00, .00]	-.35	.00
CON	-.15***	[-.18, -.11]	-8.53	.02
PEM	-.26***	[-.29, -.23]	-15.59	.02
NEM	.66***	[.63 .69]	40.72	.02

† .10≤p<.05,*p≤.05, **p≤.01, ***p≤.001
Note. $F(3, 1610) = 1179.65$***, R^2=.69, Adj. R^2=.69.

Table 8. Meta-analysis regressions: INT as the dependent variable.

Variable	β	95% CI	t	SE
Intercept	.02***	[.01, .02]	9.54	.00
CON	.05*	[.00, .09]	2.03	.02
PEM	-.39***	[-.44, -.35]	-17.61	.02
NEM	.37***	[.33, .41]	16.99	.02

† .10≤p<.05,*p≤.05, **p≤.01, ***p≤.001
Note. F(3, 1610) = 356.65***, R^2=.40, Adj. R^2=.40.

Table 9. Meta-analysis regressions: EXT as the dependent variable.

Variable	β	95% CI	t	SE
Intercept	-.01***	[-.02, -.01]	-9.62	.00
CON	-.27***	[-.31, -.23]	-12.34	.02
PEM	-.00	[-.04, .04]	-.16	.02
NEM	.68***	[.64 .72]	32.79	.02

† .10≤p<.05,*p≤.05, **p≤.01, ***p≤.001
Note. F(3, 1610) = 620.61***, R^2=.54, Adj. R^2=.54.

Qualitative comparison to self-report regression models

The model results of the correlation table values as the data emulate the model results of the self-report measure data in the directionality of the predictor measures. Notably, there is more variance explained in the meta-analytic approach across all three models run (p-factor model, INT model, and EXT model).

For the p-factor, the ERP-correlated data (Table 7) presented the following regression values, listed in strength order: NEM (β = .66), PEM (β = -.26), and CON (β = -.15). The self-report data model (Table 2) showed NEM (β = .54) as the strongest predictor, followed by CON (β = -.25), and PEM (β = -.17). Across both models, the predicted directionality of the independent variables in relation to p-factor was as expected. The adjusted R^2 was greater in the meta-analytic models: .69 versus .46.

In predicting INT symptoms, the ERP-correlated model (Table 8) yielded PEM (β = -.39) as the strongest predictor, closely followed by NEM (β = .37), and CON (β = .05) as the weakest with very modest significance. In contrast, the self-report data model (Table 3) ranked NEM (β =

.47) as the strongest predictor, PEM (β = -.22) second, and CON (β = -.09) third. Across both datasets, PEM and NEM were consistently significant predictors of INT in the expected direction. The adjusted R^2 was greater in the meta-analytic models: .40 versus .31.

Regarding EXT symptoms, the ERP-correlated model (Table 9) analysis clearly identified NEM (β = .68) as the strongest predictor, then CON (β = -.27), and PEM (β = .00) as negligible. The self-report data model (Table 4) followed a similar order, with NEM (β = .38) strongest, CON (β = -.31) second, and PEM (β = -.05) with a negligible relationship. Across both datasets, NEM and CON were consistently significant predictors of EXT in the expected directions. The adjusted R^2 was greater in the meta-analytic models: .54 versus .29.

Meta-analysis: Group main effects

Frequency, electrode, and condition group effects: General linear model

All three main effect models, for INT, EXT, p-factor, were overall significant. Of note, the frequency category was the only measure that was significant across all three models, with a consistently large effect size.

The overall p-factor model (Table 10) was significant, $F(16, 1059) = 98.22$, $p < .001$, with $R^2 = .60$ (adjusted $R^2 = .59$). Frequency had the strongest effect, $F(5, 1059) = 285.24$, $p < .001$, $\eta_p^2 = .57$, indicating a large effect. Electrode had a significant effect, $F(8, 1059) = 50.64$, $p < .001$, $\eta_p^2 = .28$, suggesting a moderate impact. Condition was also significant, $F(3, 1059) = 24.01$, $p < .001$, $\eta_p^2 = .06$, with a relatively smaller effect.

The overall INT model (Table 11) was significant, $F(16, 1059) = 73.49$, $p < .001$, with $R^2 = .53$ (adjusted $R^2 = .52$), indicating that 53% of the variance in INT was explained by the predictors. Frequency had a significant main effect on INT, $F(5, 1059) = 203.39$, $p < .001$, $\eta_p^2 =$

.49, indicating a large effect size. Electrode also had a significant effect, $F(8, 1059) = 52.41$, $p < .001$, $\eta_p^2 = .28$, suggesting a moderate effect size. Condition had a significant but smaller effect, $F(3, 1059) = 10.25$, $p < .001$, $\eta_p^2 = .03$, indicating a small effect size.

The overall EXT model (Table 12) was significant, $F(16, 1059) = 95.51$, $p < .001$, with $R^2 = .59$ (adjusted $R^2 = .58$), indicating that 59% of the variance in EXT was explained by the predictors. Frequency had a large significant effect, $F(5, 1059) = 280.52$, $p < .001$, $\eta_p^2 = .57$, suggesting a strong influence. Electrode showed a smaller but significant effect, $F(8, 1059) = 21.49$, $p < .001$, $\eta_p^2 = .14$. Condition also had a significant effect, $F(3, 1059) = 22.61$, $p < .001$, $\eta_p^2 = .06$, though the effect size was smaller than frequency.

Table 10. Meta-analysis general linear model: Analyzing main effects of frequency, electrode, condition groups predicting p-factor.

	Type III Sum of Squares	df	Mean Square	F	p	Partial Eta Squared
Corrected Model	4.70	16	.29	98.22	<.001	.60
Intercept	2.18	1	2.18	728.31	<.001	.41
Frequency	4.27	5	.85	285.24	<.001	.57
Electrode	1.21	8	.15	50.64	<.001	.28
Condition	.22	3	.07	24.01	<.001	.06
Error	3.17	1059	.00			
Total	8.75	1076	.00			
Corrected Total	7.87	1075	.00			

Note. p-factor, R^2=.60, Adjusted R^2= .59

Table 11. Meta-analysis general linear model: Analyzing main effects of frequency, electrode, and condition groups predicting INT.

	Type III Sum of Squares	df	Mean Square	F	p	Partial Eta Squared
Corrected Model	3.67	16	.23	73.49	<.001	.53
Intercept	3.77	1	3.77	1208.67	<.001	.53
Frequency	3.17	5	.64	203.39	<.001	.49
Electrode	1.31	8	.16	52.41	<.001	.28
Condition	.10	3	.03	10.25	<.001	.03
Error	3.31	1059	.00			
Total	9.29	1076	.00			
Corrected Total	6.98	1075	.00			

Note. INT, R^2=.53, Adjusted R^2 = .52

Table 12. Meta-analysis general linear model: Analyzing main effects of frequency, electrode, condition groups predicting EXT.

	Type III Sum of Squares	df	Mean Square	F	p	Partial Eta Squared
Corrected Model	5.44	16	.34	95.51	<.001	.59
Intercept	.16	1	.16	43.99	<.001	.04
Frequency	4.99	5	1.00	280.52	<.001	.57
Electrode	.61	8	.08	21.49	<.001	.14
Condition	.24	3	.08	22.61	<.001	.06
Error	3.77	1059	.00			
Total	9.20	1076	.00			
Corrected Total	9.20	1075	.00			

Note. EXT, R^2=.59, Adjusted R^2= .58

Task: Paired samples t-test

For INT, the gambling task (M = .04, SD = .08) showed significantly different correlations than the go/no-go task (M = .03, SD = .08), though both task correlations had slightly more positive means, t(806) = 3.00, p = .003, d = .11. However, the effect size was small. For EXT, the gambling task (M = .01, SD = .09) had significantly different correlations, specifically in terms of directionality, compared to the go/no-go task (M = -.01, SD = .08), t(806) = 8.17, p < .001, d = .29, with a moderate effect size. For p-factor, the gambling task (M = .03, SD = .08) also had significantly different correlations than the go/no-go task (M = .01, SD = .08), t(806) = 6.80, p < .001, d = .24, indicating a small-to-moderate effect. In this case, gambling correlations averaged slightly more positive, while the go/no-go correlations seemed to be more evenly distributed positive and negative (either in count, strength, or a combination of the two), with an average centering at 0 (Table 13).

Table 13. Meta-analysis paired samples t-test: Task group related to psychopathology measures.

	Gambling		Go/No-go		t(806)	p	Cohen's d
	Mean	SD	Mean	SD			
INT	.04	.08	.03	.08	3.00	.003	.11
EXT	.01	.09	-.01	.08	8.17	<.001	.29
p-factor	.03	.08	.01	.08	6.80	<.001	.24

Frequency relationships between psychopathology measures: Paired sample t-tests and EMM plot

Given the strong significant frequency main effects detected, a series of paired samples t-tests were conducted to compare the three psychopathology measures of interest (INT, EXT, and p-factor) across the six frequency bands (delta, theta, alpha, beta-1, beta-2, and gamma).

Although all psychopathology differences were statistically significant across all frequencies, the strongest effects were observed in alpha (Table 16), beta-2 (Table 18), and gamma (Table 19). For these higher frequencies, t-statistics ranged from 8.46 to 15.83, and Cohen's d values ranged from .65 to 1.22 (based on absolute values of the statistics), indicating strong differences and large effect sizes. The alpha frequency showed the largest differences, with INT vs. EXT yielding $t(167) = 15.25$, $p < .001$, $d = 1.17$. Similarly, INT vs. p-factor in the alpha frequency showed $t(167) = 15.83$, $p < .001$, $d = 1.22$, representing the largest observed effect size in the dataset.

In contrast, lower frequency bands (delta: Table 14, and theta: Table 15) and beta-1 (Table 17) showed moderate but significant differences. For example, in the theta band, INT vs. EXT showed $t(401) = 8.64$, $p < .001$, $d = .43$, in the delta band, the same comparison resulted in $t(539) = 8.22$, $p < .001$, $d = .35$, and in the beta-1 band, this comparison resulted in $t(167) = 3.73$, $p < .001$, $d = .29$.

The estimated marginal means plot (Figure 6) further illustrates these frequency-based differences. INT trends positive on average, with a general increase from lower to higher frequencies, except gamma, which sees a drop more to a value closer to alpha-INT average. EXT and p-factor, generally, trend negative in lower frequencies and positive for higher frequencies.

Table 14. Meta-analysis paired samples t-test: Comparison between psychopathology measures for delta frequency data.

	Delta Group 1		Delta Group 2		t(539)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	-.01	.07	-.03	.07	8.22	<.001	.35
INT vs p-factor	-.01	.07	-.02	.07	9.94	<.001	.43
EXT vs p-factor	-.03	.07	-.02	.07	-5.77	<.001	-.25

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with Delta Group 1 and Delta Group 2 column headers.

Table 15. Meta-analysis paired samples t-test: Comparison between psychopathology measures for theta frequency data.

	Theta Group 1		Theta Group 2		t(401)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.02	.08	-.01	.07	8.64	<.001	.43
INT vs p-factor	.02	.08	.01	.07	7.71	<.001	.38
EXT vs p-factor	-.01	.07	.01	.07	-8.29	<.001	-.41

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with Theta Group 1 and Theta Group 2 column headers.

Table 16. Meta-analysis paired samples t-test: Comparison between psychopathology measures for alpha frequency data.

	Alpha Group 1		Alpha Group 2		t(167)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.03	.04	-.07	.06	15.25	<.001	1.17
INT vs p-factor	.03	.04	-.03	.03	15.83	<.001	1.22
EXT vs p-factor	-.07	.06	-.03	.03	-14.16	<.001	-1.09

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with Alpha Group 1 and Alpha Group 2 column headers.

Table 17. Meta-analysis paired samples t-test: Comparison between psychopathology measures for beta-1 frequency data.

	Beta-1 Group 1		Beta-1 Group 2		t(167)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.08	.09	-.01	.06	9.52	<.001	.73
INT vs p-factor	.08	.09	.04	.06	8.46	<.001	.65
EXT vs p-factor	-.01	.06	.04	.06	-10.12	<.001	-.78

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with Beta-1 Group 1 and Beta-2 Group 2 column headers.

Table 18. Meta-analysis paired samples t-test: Comparison between psychopathology measures for beta-2 frequency data.

	Beta-2 Group 1		Beta-2 Group 2		t(167)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.09	.10	.06	.06	3.73	<.001	.29
INT vs p-factor	.09	.10	.10	.08	-2.56	.01	-.20
EXT vs p-factor	.06	.06	.10	.08	-7.92	<.001	-.61

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with Beta-2 Group 1 and Beta-2 Group 2 column headers.

Table 19. Meta-analysis paired samples t-test: Comparison between psychopathology measures for gamma frequency data.

	Gamma Group 1		Gamma Group 2		t(167)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.03	.04	.10	.09	-11.94	<.001	-.92
INT vs p-factor	.03	.04	.08	.07	-12.82	<.001	-.99
EXT vs p-factor	.10	.09	.08	.07	9.26	<.001	.71

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with Gamma Group 1 and Gamma Group 2 column headers.

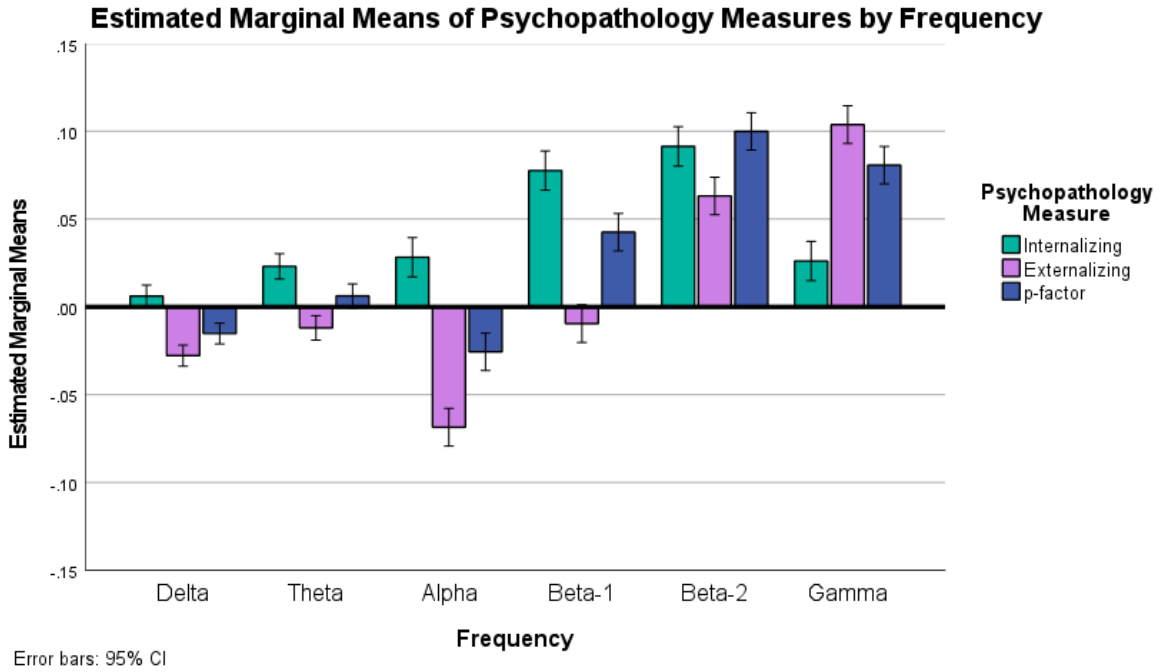
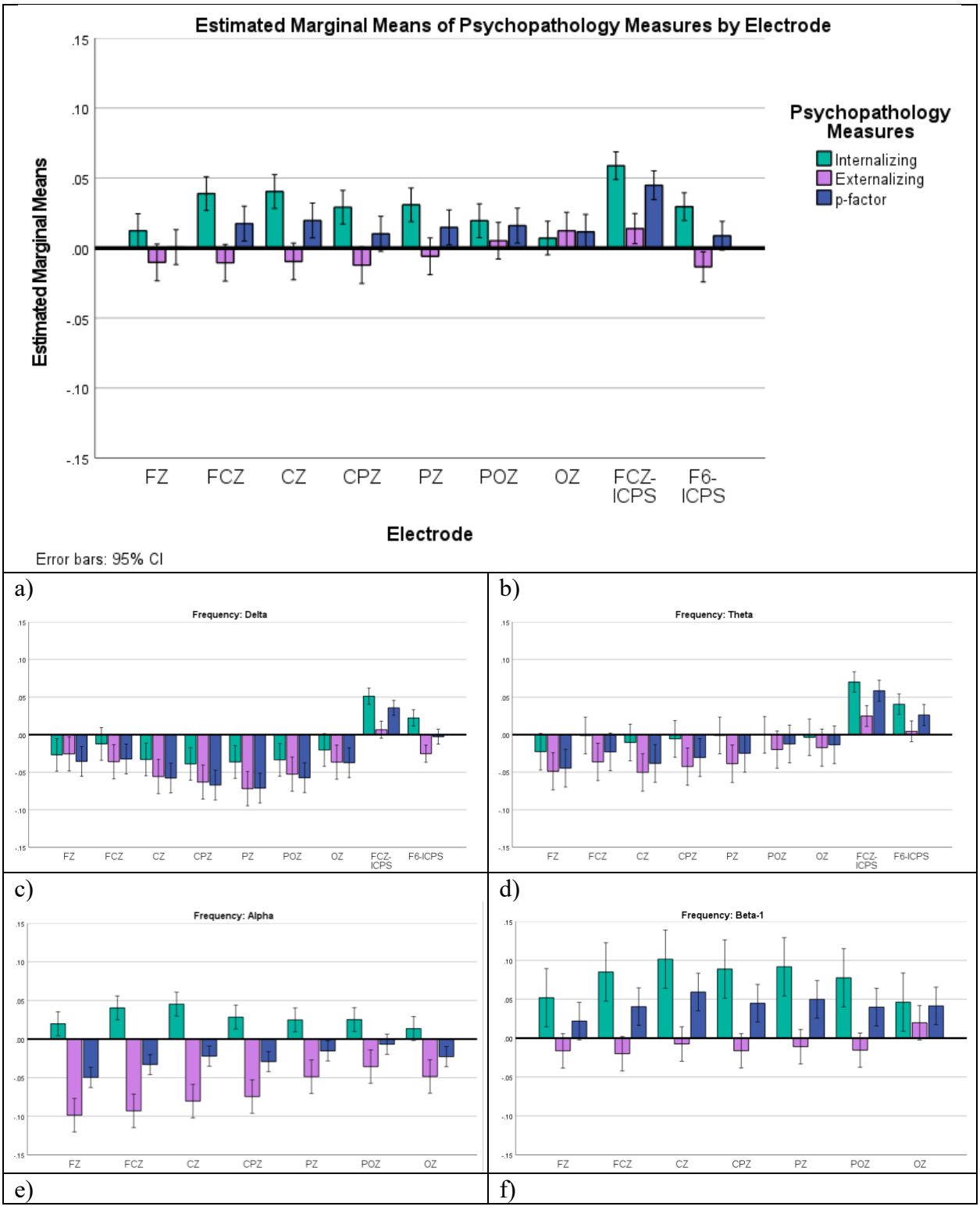


Figure 6. Depiction of adjusted means for psychopathology measures across frequencies to understand model-based group differences while accounting for other variables.

Electrode relationships between psychopathology measures by frequency: EMM Plots

Electrode differences were also consistently meaningful across the three psychopathology measures (p-factor: $\eta_p^2 = .28$; INT: $\eta_p^2 = .28$; EXT: $\eta_p^2 = .14$). An overall review of the electrode relationships and a breakdown of the electrode differences separated by frequency was conducted. Frequency separation was included given the differences seen among frequencies. Delta and theta generally showed reduced ERP deflection across electrodes, except for the ICPS seed electrode data. For alpha, beta-1, beta-2, and gamma all had positive deflections for INT, but p-factor and EXT displayed more variation. For alpha, EXT and p-factor displayed negative deflections. However there is a shift to positive deflections across all electrodes beginning with p-factor shifting for beta-1, and p-factor and EXT both displaying positive deflections for beta-2 and gamma. Also interestingly, INT did have positive deflections, generally increasing gradually from alpha to beta-2 frequencies, but deflections were differently decreased in gamma (particularly in parieto-occipital and occipital electrodes), though still largely positive. A series of paired samples t-test for the main electrode differences are included in Appendix D.


Estimated Marginal Means of Psychopathology Measures by Electrode
Estimated Marginal Means
.15
.10
.05
.00
-.05
-.10
-.15
FZ
FCZ
CZ
CPZ
PZ
POZ
OZ
FCZ-ICPS
F6-ICPS
Electrode
Psychopathology Measures
Internalizing
Externalizing
p-factor
Error bars: 95% CI
a)
Frequency: Delta
b)
Frequency: Theta
c)
Frequency: Alpha
d)
Frequency: Beta-1
e)
f)

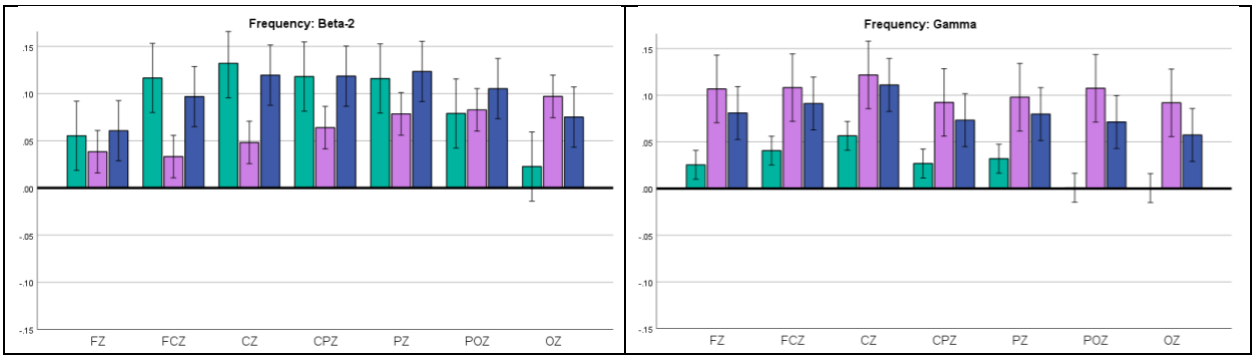


Figure 7. Depiction of adjusted means for psychopathology measures across electrodes to understand model-based group differences while accounting for other variables.
Note. Plots are shown overall (all data), and by frequency: a) delta, b) theta, c) alpha, d) beta-1, e) beta-2, and f) gamma.

Chapter 5: Discussion

The study focused on confirming and assessing how transdiagnostic RDoC-relevant measures collectively predicted transdiagnostic psychopathology measures. This was done both with only individual self-report measures, and further extended to incorporate neurophysiological measures correlated with the self-report measures in a novel meta-analytic approach.

Previous work in the lab conducted with self-report data compiled across several datasets supported a priori hypotheses that PEM and CON would negatively relate to psychopathology measures, while NEM would relate positively to the psychopathology measures (Mannava, 2025). The self-report analyses conducted with the current dataset, with MPQ representing PEM, NEM, and CON, IDAS representing INT, ESI representing EXT, and a factor analysis of IDAS and ESI representing p-factor, generally confirmed these relationships, with some distinctions from the hypotheses related to INT and EXT, which will be further discussed.

A key extension to the above analyses was constraining the self-report measures to neurophysiology data utilizing a within-subjects meta-analytic approach that correlated 1,614 ERP components with the three HiTOP and three MPQ self-report measures of interest. This approach allowed the initial self-report measure only analyses to be grounded with the inclusion of objective neurophysiological measures. Similar to the original regression analyses, when neurophysiology measures were constrained to the HiTOP and MPQ measures, the expected relationships generally persisted, again with the same, to be discussed, distinctions from the hypotheses related to INT and EXT.

The current study established psychopathology and cognitive processes relationships across valuable transdiagnostic models and, newly, in a within-subjects meta-analytic approach.

HiTOP-RDoC relationships

Overall, the models presented generally confirm that the a priori psychopathology and cognitive processes relationships persist in the current dataset's self-report analyses, as well as in the subsequent meta-analytic approaches.

First, the confirmation of relationships in the self-report measures from the current clinical dataset provides further support for previous findings, while providing a base and verified comparative model with which to relate the new meta-analytic approach.

Second, the meta-analytic approach provides insight into the persistence of these relationships after incorporating a relatively comprehensive set of neurophysiological measures (across frequencies and electrodes). Although there were differences in aspects of the relationships, the overall models were similar to the self-report model results, and generally were as hypohesized.

Self-report: Results review

The self-report measure results generally support previous findings and literature regarding the expected relationships between the HiTOP and MPQ measures. Namely that psychopathology is negatively related to CON (Goschke, 2014; McTeague et al., 2016; McTeague et al., 2017) and PEM (Carl et al., 2013; Santos et al., 2013; Kendall et al., 2015), but positively related to NEM (Boschloo et al., 2013; Watson & Naragon-Gainey, 2014; Osman & Wood, 2018). This preliminary step was important to validate the relationships in the current dataset before expanding the analysis to include neurophysiological measures and before attempting a novel approach in the dataset.

The relationships of MPQ with p-factor were entirely as predicted (negative with CON and PEM, positive with NEM), additionally accounting for the largest explanation in the data

results, likely due to equal contributions from both INT and EXT characteristics. INT, however, followed expected relationships with PEM and NEM, but was not significantly related to CON. Similarly, EXT, followed expected relationships with CON and NEM, but was not significantly related to PEM. These variations will be discussed next.

Although there is work relating CON deficiencies to INT (Miller et al., 2013), CON is generally more related to EXT mechanisms. The sub facets of CON in MPQ include impulsivity control, harm avoidance, and traditionalism, which are generally more negatively related to the disinhibition and aggressive behaviors seen in EXT (Latzman et al., 2011; Hopwood et al., 2013; Walton et al., 2017; Creswell et al., 2019), and less related to the inward-focused INT symptoms, such as withdrawal or emotional distress (Eaton, Krueger, Markon et al., 2013). This inward-focused symptomology of disorders, such as depression or anxiety, would, however, closely relate to emotional experiences: increased NEM and decreased PEM (Anthony et al., 2002; Krueger et al., 2001), lending support for significant relationships with NEM and PEM, but not with CON. Additionally, this finding was different from previous similar work done in the lab, in which INT did exhibit a significant negative relationship with CON (Mannava, 2025). This previous work was done compiling data across several studies with likely more representatively distributed psychopathology population characteristics. This current study, however, focused on an INT population. In the past study, the INT-CON relationship was still weaker relative to PEM and NEM's relationship with INT, and relative to EXT-CON and p-factor-CON relationships. It is possible that the INT-CON relationship was further weakened in an INT-focused population, congruent with rationalizations described above about the INT-CON relationship.

EXT, while demonstrating a robust positive relationship to NEM and negative relationship to CON, did not show a significant relationship with PEM. Although there is work

relating decreased PEM with EXT (Humbad et al., 2010), it is not as closely related in the same way that decreased CON (Meehan et al., 2013; Hall et al., 2021) and increased NEM are related to EXT (Krueger, Markon, Patrick, Benning & Kramer, 2007). This lack of a directional relationship between PEM and EXT is not necessarily a new finding (Krueger et al., 2001). Further, studies have even indicated that some EXT behaviors, like sensation-seeking, may actually be associated with increased PEM, meaning that the assumption that PEM is solely low in EXT may be too simplistic (Moore et al., 2024). Additionally, previous work specifically in gambling and decision-making tasks indicated that risky behaviors may be more motivated by reward and less revised by losses (Ba et al., 2019). Due to this complexity of opposing operations in EXT related to PEM, PEM has greater potential to not be considered a significant predictor of EXT in either direction.

Finally, across all three psychopathology measure regression analyses, NEM presented as the strongest predictor in each respective analysis (always a positive predictor). One reason for this may be that NEM reflects a general emotional vulnerability that uniformly cuts across nearly all forms of psychopathology (Stanton & Watson, 2014) in a way that, as mentioned, PEM and CON may not. Increased NEM related mechanisms appear to be the strongest predictors of psychopathology measures, providing evidence that NEM should be viewed as a valuable indicator of all three transdiagnostic psychopathology measures.

Meta-analysis: Results introduction

The meta-analyses produced important findings with unique theoretical and methodological implications. Given the novel meta-analytic approach it seemed important to discuss those, as well as statistical characteristics surrounding the approach. Therefore, the following discussion will review both statistical takeaways that can be applied to

psychopathology and RDoC-relevant theory, and relevant features regarding the meta-analyses, themselves.

Meta-analysis: Descriptive statistics and t-test analyses

In order to review the meta-analytic variables more comprehensively and to understand the structure of the variables (i.e., correlation coefficient values between ERP components and psychopathology/MPQ measures), descriptive statistics and one-sample t-tests were evaluated. Descriptive statistics indicated that the variables seemed to have a relatively even spread of correlations across positive and negative values (review: mean values, and minimum and maximum values in "Results" section). This implied a likely normal distribution, an important assumption to meet in order to conduct further analyses (Fisher, 1915; Borenstein et al., 2021; Hedges & Olkin, 1985), and one that was also verified in the "Methods" section. Given that the correlation values were in both the positive and negative direction, and the means centered at zero or very close to zero, further descriptive statistics were run with the absolute value numbers of the variables to understand the magnitude. This provides some more insight into the magnitude, with mean value numbers ranging from .05 to .07, and the correlation values as negative as -.24 and as positive as .25 . This correlation range and variation in the magnitude of values indicated a further need (beyond the large sample size reasoning) to review effect size values rather than simply reviewing significance values alone (Sullivan & Feinn, 2012).

The one-sample t-test further applied an understanding of the directionality of the correlation values for each variable. In other words, an indication of whether the correlations leaned more negative or more positive for each psychopathology and MPQ variable. For this one-sample t-test, and all analyses, more consideration was given to the effect sizes, rather than the significance levels (Sullivan & Feinn, 2012). Given the large sample size, an artifact of the

meta-analytic approach, it is likely that significance could be seen where it is not meaningful (Lantz, 2013).

This effect size review indicated that PEM and INT variable correlation values lean meaningfully negative and positive, respectively. The following descriptions related to specific ERP components are based on previous understandings and previous studies of ERP components in the relevant tasks related to psychopathology constructs. The present study did not conduct analyses based on specific ERP components related to each measure, but rather was a compilation of measures across spatial, temporal, and frequency distributions.

Increased PEM may be more negatively related to ERP activity for several reasons. Individuals with higher PEM may have less internal conflict related to response selection (Zinchenko et al., 2017; e.g., less augmented no-go N200 or no-go P300, Gajewski et al., 2007), less effortful inhibition or more approach bias (Pandey & Gupta, 2022; e.g., less augmented no-go P300, Salisbury et al., 2004), or less error sensitivity or concern over mistakes (Fishman & Ng, 2013; e.g., less augmented error-related negativity, Wang et al., 2020) in the go/no-go task. Related to gambling, increased PEM may relate to less loss sensitivity due to an optimism bias (Borkenau & Mauer, 2006; e.g., less augmented feedback-related negativity, Isheqlou et al., 2023; Zheng & Mei, 2023) or a positivity baseline (e.g., moderated reward positivity, Paul et al., 2020) that flattens a response to gain feedback. Across both tasks, this may be indicative of reduced engagement to the task, due to lower potential stress from the tasks resulting in the tasks being repetitive or boring (e.g., P300 amplitude reduction, Lammers & Badia, 1989).

Almost inversely, individuals with INT may relate positively with the tasks reviewed in this study due to increased stress or salience. Individuals may have greater sensitivity to errors in go/no-go (Gorka et al., 2017; e.g., enhanced error-related negativity or decreased no-go P300,

Olvet & Hajcak, 2009; Pasion & Barbosa, 2019; Trayvick et al., 2024) or loss feedback (Tobias & Ito, 2021; e.g., larger feedback related negativity, Mason et al., 2012) in gambling, while having an over-attention to all feedback in general (Radoman et al., 2019; e.g., increased P300, Huang et al., 2015). In some cases, positive feedback could lead to, for example, a heightened P300 (Huang et al., 2015) or moderated reward positivity in those expecting a negative result (Proudfit, 2015; Umemoto & Holroyd, 2017), a common symptom of depression or anxiety.

To a lesser extent, NEM and p-factor variable correlation values leaned more positive for both variables. For both of these measures, the ERP relationships can be quite similar to that of the reasoning for INT's positive relationship with ERP measures. However, there may be different reasons or influences. For instance, NEM features, such as sensitivity to failure may elicit similar activity to INT's sensitivity to errors (Johnson et al., 2017). For p-factor, features similar to INT may stem from the shared variance, especially given the current INT population. It is important to note that the effect sizes were small for these measures (smaller than the effect sizes for PEM and INT), so it did not seem appropriate to interpret the meaning of these values too extensively.

For CON and EXT, the effect sizes indicated no meaningful difference from zero. Although it is difficult to decipher this meaning, presently, this does not mean that correlations were only zero but rather that the distribution may be evenly positive and negative correlations, or that there may be fewer negative correlation values, but that they have a stronger magnitude than the large positive correlation values, or vice versa.

Rather than an analysis meant to generate concrete interpretations of the ERP data, this t-test step was considered a preliminary measure to descriptively understand the data and lay a base for further evaluations.

Meta-analysis: Regression models - Meta-analytic extension to self-report models

The meta-analytic multiple regression models served two main purposes: 1) to incorporate neurophysiology indicators as an objective variable alongside traditional psychological predictors into a meta-analytic regression framework, and 2) to evaluate the utility of a new analytic approach while comparing it to more traditional self-report based analyses.

First, the purpose was to utilize this methodology to incorporate neurophysiological measures as an objective method, which lends support towards a current transdiagnostic methodological advancement aiming to integrate biological data into large-scale quantitative models (NIMH, About RDoC 2009). Although there are benefits to self-report behavioral level analyses, there are also concerns (detailed more in the upcoming "Meta-analytic approach" section, as well) with relying solely on these methodologies (e.g., they are weakly correlated with each other indicating possible reliability issues across the measures, Dang et al., 2020). A primary goal and benefit of the RDoC model focuses on identifying biological markers that deviate from normal functions that could account for particular symptoms (NIMH, About RDoC 2009). This does not imply disregarding or denouncing existing self-report and behavioral methodologies, but rather incorporating them with biological markers to develop a more comprehensive model picture (Cuthbert & Insel, 2013; Morris et al., 2022; Patrick et al., 2019). This addition of neurobiological markers to the regression model reinforces both the validity of the constructs and lends support for theoretical models that recommend evaluating the neurobiological markers and mechanisms that underlie psychopathology. As mentioned in the introduction of this study, neuroscience and psychology research is moving towards and benefits from transdiagnostic approaches like the RDoC or HiTOP models (Morris et al., 2022; Kotov et al., 2017). This approach can open avenues and be employed to identify reliable biomarkers that

may inform diagnosis, intervention, or risk prediction, aligning well with the goals of the previously mentioned transdiagnostic RDoC and HiTOP models (Cuthbert & Insel, 2013; Morris et al., 2022; Insel et al., 2010; Kotov et al., 2017; Ruggero et al., 2019; Dalgeish et al., 2020).

Second, and equally important, the novel within-subjects meta-regression analysis approach aimed to extend upon the existing self-report model methods and theories. Specifically, this approach leverages the primary strength of typical meta-analytic approaches – integrating findings across studies – to then comprehensively evaluate several task data in a single study (Glass, 1976). By comparing these meta-analytic results with those derived from more conventional self-report based analyses, the current work provided a unique opportunity to test the robustness of previously established findings in this new approach, with the additional incorporation of relevant measures. The convergence of significant predictors across both analytic methods (self-report regressions: Tables 2-4, and meta-analytic regressions: Tables 7-9) emphasizes the potential utility of this unique meta-analytic approach, while also lending empirical support for the theoretical constructs and relationships of interest.

Meta-analysis: Regression models and comparison to self-report regressions

Given the similarities of the significant predictors and the directionalities of the HiTOP-RDoC relationships between these meta-analytic regression analyses and the self-report measure regression analyses, previously mentioned theoretical descriptions (review: "Self-report: Results review" section) of the predictors related to each psychopathology measure can be applied here, as well. Given the complexity of the neurophysiological data, it is difficult to interpret specific concepts from the regression analysis alone (the following review of the general linear models may be more helpful in this regard).

However, a critical difference after the addition of the neurophysiological measures is a larger effect size, or adjusted R^2 value, across all three regression models. This improvement in model fit and variance explained of the dependent variable by the independent variables indicates enhanced reliability and generalizability of the observed relationships (Gupta et al., 2024). Although it is difficult to posit the exact impact of adding neurophysiological measures at this time, it is possible that including these measures may capture more stable (in measurement and over time; Yoon et al., 2015) and objective associations between the HiTOP and RDoC-relevant variables of interest (Pasion et al., 2023; McLoughlin et al., 2014; Leiser et al., 2011; Widge et al., 2019; Bel-Bahar et al., 2022; Woo & Wager, 2015). The increased adjusted R^2 value may highlight the value of this meta-analytic approach in evaluating broader patterns across several data structures and the value of incorporating objective neurobiological measures.

Meta-analysis: General linear model and paired sample t-test analyses

General linear model analyses examining the effects of frequency, electrode, and condition on each of the three psychopathology measures were included to better interpret the individual relationships across characteristics of neurophysiological measures, which may offer possibilities for even more finely tuned neural biomarkers (McLoughlin et al., 2014). Across all three models, frequency emerged as the strongest contributor, suggesting that this variance among the six frequency bands plays a substantial role in explaining individual differences in the three psychopathology measures, aligning with previous interpretations of frequency-related disparities (Bernat et al., 2007; Newson & Thiagarajan, 2019; Knyazev et al., 2003; Pavuluri et al., 2023). Electrode locations also showed a large effect across all three psychopathology measures indicating that spatial differences in neural activation may meaningfully relate to different psychopathology measures or diagnoses (Du et al., 2023; Gerez & Tello, 1992; Gordon

et al., 2010; McLoughlin et al., 2022). Condition effects were small, pointing to modest contribution of task-related conditions to the psychopathology measures. Finally, task effects were reviewed separately, as paired sample t-test analyses, due to potential overlap with the condition measure. The t-test results indicated small effect size values, substantiating the condition effects seen in the general linear model analyses. Notably task-related differences are of interest in psychopathology (Rajna et al., 2003), and more details related to these differences will be meaningful to briefly discuss, however, the effects were minimal in the present study. Overall, the findings suggest the need to further evaluate and compare these different group and data subsets in the context of this meta-analytic approach here, and in future studies.

Frequency differences were the most meaningful across all three psychopathology measures. As mentioned earlier in the introduction, time-frequency analyses are vital in understanding the distinctions that exist between frequency measures related to cognitive processes (Bernat et al., 2008; Bernat et al., 2011). Utilizing this methodology was beneficial in identifying the substantial frequency differences that exist in relation to each psychopathology measure (INT, EXT, and p-factor). In the current study, based on the Cohen's d effect sizes in the paired sample t-tests conducted by frequency measure, the differences among higher frequencies, particularly alpha, beta-1, and gamma bands, appear to be more influential than differences seen among lower frequencies, particularly delta and theta bands. Most interesting, based on the estimated marginal means comparison graph, may be the beta-2 frequency band showing increases across all psychopathology measures and pointing to possible overall problems. There is work similarly discussing increased beta activity across studies related to grief and trauma, substance use, and disinhibited behavior (Jang et al., 2017; Kahn, 2021; Rangaswamy et al., 2002; Ceballos et al., 2009; Chojak et al., 2023). It is worth mentioning that

the studies do vary in their classification of beta, some aligning more with the current study beta-1 and beta-2 ranges (Kahn, 2021), and others not (e.g., beta 1: 12.5-16 Hz, beta 2: 16.5-20 Hz, and beta 3:20.5-28 Hz, Rangaswamy et al., 2002; or single beta band: 12-30 Hz, Jang et al., 2017). Notwithstanding this difference, collectively, this may indicate that beta-2 is a predictor of p-factor, INT and EXT. Related, beta-1 is positively related to INT here, possibly indicating a specific predictive quality of beta-1 to INT. There is some work relating beta-1 and INT (e.g., heightened anxiety related to increased beta; Stenberg, 1992), but generally this specific relation appears to be under-reviewed (Engel & Fries, 2010). The gamma band may be a predictive measure related to EXT and p-factor, and seemingly less related to INT than it is to EXT and p-factor. There appears to be contradictory outcomes related to the gamma band. There is work indicating increased gamma amplitudes in patients with ADHD (i.e., EXT disorder, Hermann & Demiralp, 2005). However, this positive relationship with EXT is inconsistent with some previous work noting alpha and gamma bands as marginally lower in EXT samples compared to a control sample (Rudo-Hutt, 2015), and review work noting decreases in gamma activity across psychiatric disorders (Newson & Thiagarajan, 2019). Notably many of these studies mentioned contradictions do exist, gamma is less studied, and further work related to gamma would be beneficial to pursue. Alpha, is generally considered an inhibitory signal (Uusberg et al., 2013), and decreased alpha in EXT psychopathology may indicate less inhibitory activity, consistent with general characteristics of EXT behaviors (McDonald et al., 2019). Additionally, except for beta-2 and gamma frequencies, INT seems to generally be more positive relationships, while EXT appears to be more negative relationships based on the estimated marginal mean graph. There have been varying results of internalizing and externalizing having different (i.e., unique variance, Beatty et al., 2023; Moadab et al., 2010) and similar (i.e., shared variance, Bernat et al.,

2020; Schettini et al., 2021) relationships with neurophysiology, and further dissection, like at this frequency level, would be beneficial.

Electrode location variability provides valuable spatial information about the neural correlates and activity. Since the electrode activity included both a representation of midline electrodes and ICPS electrode connections, these results indicate meaningful differences based on the underlying brain structures and neural connections. There is a vast amount of existing work indicating the relevance of brain regions in understanding psychopathology (Du et al., 2023; Gerez & Tello, 1992; Gordon et al., 2010; McLoughlin et al., 2022). For example, frontal electrode sites, often associated with executive function or emotion regulation (Dennis & Solomon, 2010), may be disrupted in INT disorders, such as depression and anxiety. One promising marker of depression vulnerability may present with frontal asymmetry (Allen & Reznik, 2015). Differently, EXT behaviors, like emotional disorder or disinhibition, may be better captured in the fronto-central electrode sites (e.g., anterior cingulate cortex or orbitofrontal cortex, Thijssen et al., 2021). This information can be reviewed to better identify disturbed regions contributing to the higher order p-factor, with previous studies noting a shared problem behavior between INT and EXT (i.e., p-factor) accounting for P300 amplitude reductions (Bernat et al., 2020).

Although the overall electrode results provided important spatial information across psychopathology measures, a further breakdown by frequency provided a more detailed review of the patterns. For instance, beta-2 and gamma both indicated increased activity across all included electrode sites and neural connections, which may be consistent with mentions of increased beta activity (Jang et al., 2017). For the midline electrodes, the patterns for delta and theta indicate negative deflections across all psychopathology measures, with increased

variability seen in alpha and beta-1where internalizing shifts in alpha and p-factor joins this shift in beta-1, both indicating positive deflections. There is currently limited work, to our knowledge, that unravels EEG data in the context of psychopathology at this level of detail, so our interpretations are limited. These patterns do present a valuable opportunity, however, and it would be beneficial to expand upon. By interpreting the various spatial processes, localized or network-based dysfunctions can be more deeply identified to develop a nuanced knowledge on neurobiological substrates of mental disorders.

Across task and condition differences for all three psychopathology measures, the effect sizes were present, but small relative to other measures. This could mean that, although the tasks are meant to index different cognitive processes, there may be cognitive mechanisms that are consistently impacted (i.e., compromised discrepancy between network functions), across conditions and tasks, due to psychopathology-related modulations. There is evidence to suggest that, in the presence of psychopathology, there could be impaired modularity, state-dependent processing, or network-level brain dysfunction, which could impact individual task or feedback processing, uniformly (Menon, 2011; Xia et al., 2018). Interestingly, although the effect sizes were small, a preliminary post-hoc analysis review did reveal significant differences between all the condition pairings, except loss and gain conditions (Appendix D). This was true across all three psychopathology measures, noting a possible similarity in all feedback processing, whether positive or negative. This may again point to a possible dysregulation in neural systems in those with psychopathology, leading to this consistent response to both feedback types (Johnson et al., 2020).

This review of ERP feature groups is limited as it was not a primary goal of the present study, but it is an important benefit of incorporating neurophysiology measures and of this meta-analytic method that could be replicated and extended, with seemingly considerable potential.

Meta-analytic approach considerations

The meta-analysis approach aimed to enhance the past work and current self-report analyses by including a second measure related to RDoC constructs: neurophysiology. The self-report measures provide important and validated insight into the relationships, but there are several concerns with only reviewing self-report measures. Some of the concerns with only relying on self-report measures may include: shared method variance, response bias, insight limitations, retrospective bias, and a lack of multi-method variability (Ben-Porath, 2003; Pavlova & Uher, 2020). Shared method variance is the concern that when all variables come from the same methodology, the associations may be inflated due to method bias. Response bias, insight limitations, and retrospective bias all relate to concerns with the participant's reporting. Participants may underreport or overreport, lack insight into their own emotional processes, or inaccurately reflect on past behavior or mood, respectively. The attachment of the neurophysiological measures to the self-report measures to the correlations aimed to attach objective processes when interpreting the relationships, and may alleviate concerns mentioned when only self-report measures are used.

This methodology further enhances the goals of the RDoC framework by involving psychological or biological systems into the variables and model analyses (NIMH, About RDoC 2009; Cuthbert & Insel, 2013). Self-report measures do offer a validated base and understanding, but given the reasons mentioned, it is important to include an objective measure, which in this case was EEG measures.

Limitations

A primary limitation and possible concern with the data is the correlational values in the dataset. The descriptive analyses indicate that the mean of the correlation values for all variables is weak and close to zero. In order to avoid bias, there was no selective process in including ERP components to correlate with the self-report measures. The primary goal was to be as inclusive and comprehensive as possible, which did lead to a large number of indiscriminate correlation values. The only selection related methodology was in the principal components analysis conducted related to delta and theta frequency bands. This methodology allowed us to select the number of meaningful components, since there is not a standard number of components to select components outside of this process.

Apart from this selection, ERP components were included across tasks, conditions, condition differences, frequencies, electrodes and time windows (for alpha, beta-1, beta-2, and gamma), uniformly. Given this nonselective process there was no consideration for significant, large correlations, but rather a methodology to include all possible characteristics of neurophysiological measures. This meant small, and even nonsignificant, correlation values would be included in the analyses. Generally, this is concern for Type 1 (detecting a non-robust effect) or Type II (failing to detect a real effect) errors. This does not seem to be the case given the effect size results, but it is an important limitation and future replications could aim to review more meaningful correlations in addition to replicating the present methodology.

Additionally, the study was conducted in a dataset of primarily INT subjects, which could skew the results differently from what may be seen in a typical population or in a population with more symmetrically distributed psychopathology features (i.e., distribution of HiTOP-related diagnoses). There was a mention of an area where this could be a concern in the

discussion section – minor variation in regression results compared to past work regression results in the lab – so it may be important to review these analyses and this new methodology in a less restrictive population.

Related, p-factor was computed as a factor score from INT and EXT related measures. This may be influenced in an INT-focused population, but additionally, p-factor consists of other components that do not fall under the INT or EXT umbrellas – somatoform, detachment, and thought disorders. INT and EXT do account for a meaningful majority of the diagnoses listed in the HiTOP model (Kotov et al., 2017), but in order to have the most well-represented measure of p-factor it would be worthwhile to include the other disorder domains mentioned in different samples.

Future directions

First, it would be critical to replicate this study in a different dataset. Although there are many reasons to consider this within-subjects meta-analytic approach as statistically reliable based on the thorough data and analysis assumption assessments conducted, there are still concerns, as mentioned above. Additionally, to our knowledge, this type of meta-analytic approach has not been attempted before and it would be important to review this across various datasets, both with similar and different variables and theories, as well as similar and different population sets.

Second, there may be a benefit to conducting a more focused review of the data by selecting ERP components that are either meaningfully related to the self-report measures or meaningfully related to the psychopathology and RDoC constructs based on a priori understandings of the transdiagnostic models and theories. Ideally, this would include conducting higher-end statistical analyses to then identify and narrow down the ERP components

included. There are benefits to continuing with the current indiscriminate approach towards neurophysiology measures, but it may be interesting to apply the approach to a priori neurophysiology relationships with psychopathology.

Third, it would be beneficial to expand beyond the current task and ERP data included in this study. If a more focused review (i.e., selecting neurophysiology measures based on higher-end statistical analyses) is desirable, this expansion will be more pertinent to ensure a large dataset model for the meta-analytic review process.

The tasks in this data were selected due to the goal of reviewing CON, PEM and NEM. The go/no-go task is generally considered as related to cognitive control, while the gambling task is generally related to positive and negative feedback or emotion responses. However, other tasks, such as the emotion regulation task, oddball task, or resting task can provide further insight into not only the CON, PEM, and NEM domains, but other domains in RDoC as well. For instance, the emotion regulation task may add to PEM, NEM and CON understandings, in addition to another domain: arousal/regulatory systems (NIMH, About RDoC 2009). Incorporating more tasks could work towards a more complete picture of relationships between RDoC measures and psychopathology.

Another addition would be the inclusion of more ERP components. The current data did include all frequencies, all midline electrodes, and various methods of data reductions, to be inclusive in this novel approach, however, even more computations are possible. For example, ICPS analyses only included delta and theta frequency measures, but could be expanded to include alpha, beta-1, beta-2, and gamma measures, as well. The same expansion could be conducted in the context of ITPS analyses. Although the focus was on midline electrodes in order to create a somewhat even distribution of brain areas from frontal region to occipital

region, it would be helpful to review electrodes or electrode clusters based on brain regions of interest. For instance, the prefrontal cortex and the anterior cingulate cortex, in addition to various interconnected networks, are known to be related to cognitive control processes (Cai et al., 2022). Another area of interest related to positive and negative emotions may be the amygdala (Sergerie et al., 2008). Beyond an extension to these features of ERP, the timeframes and components examined could be expanded, as well. The timeframes of the tasks were during the feedback response portion, but more components could be reviewed to include the stimulus-locked timeframes (i.e., the moment that the participant makes a selection). This could help identify cognitive processes during the selection period. This expansion and subsequent breakdown by ERP features could provide valuable and detailed information about the neural processes related to psychopathology.

This preliminary meta-analytic approach aimed to statistically test the method and models, but is meant to be the first step in what could be a vast number of possible studies given future validation and expansion endeavors.

Appendices

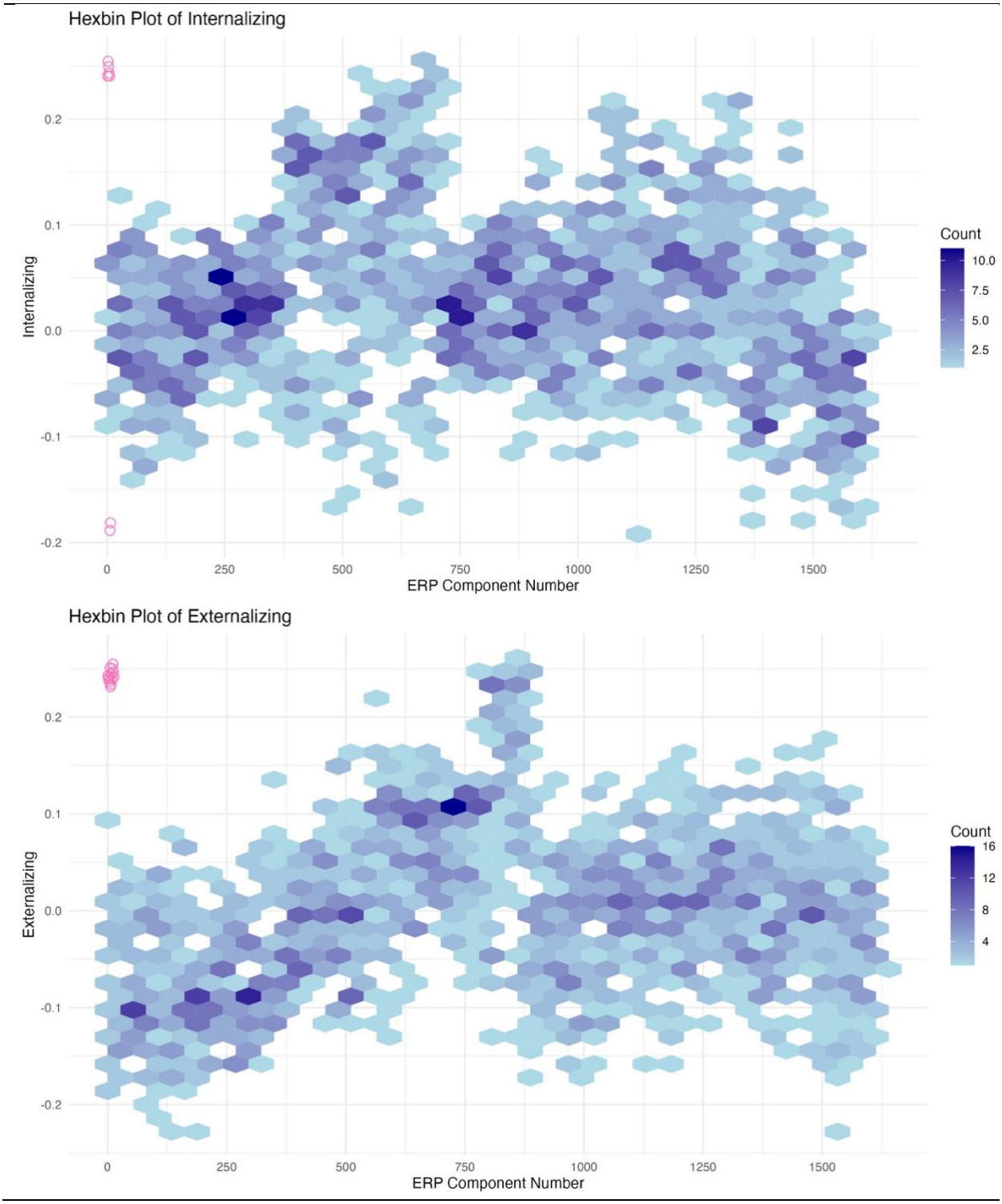
Appendix A: Normality tests

Table A1. Meta-analysis: Statistical normality tests for the dependent variables of interest.

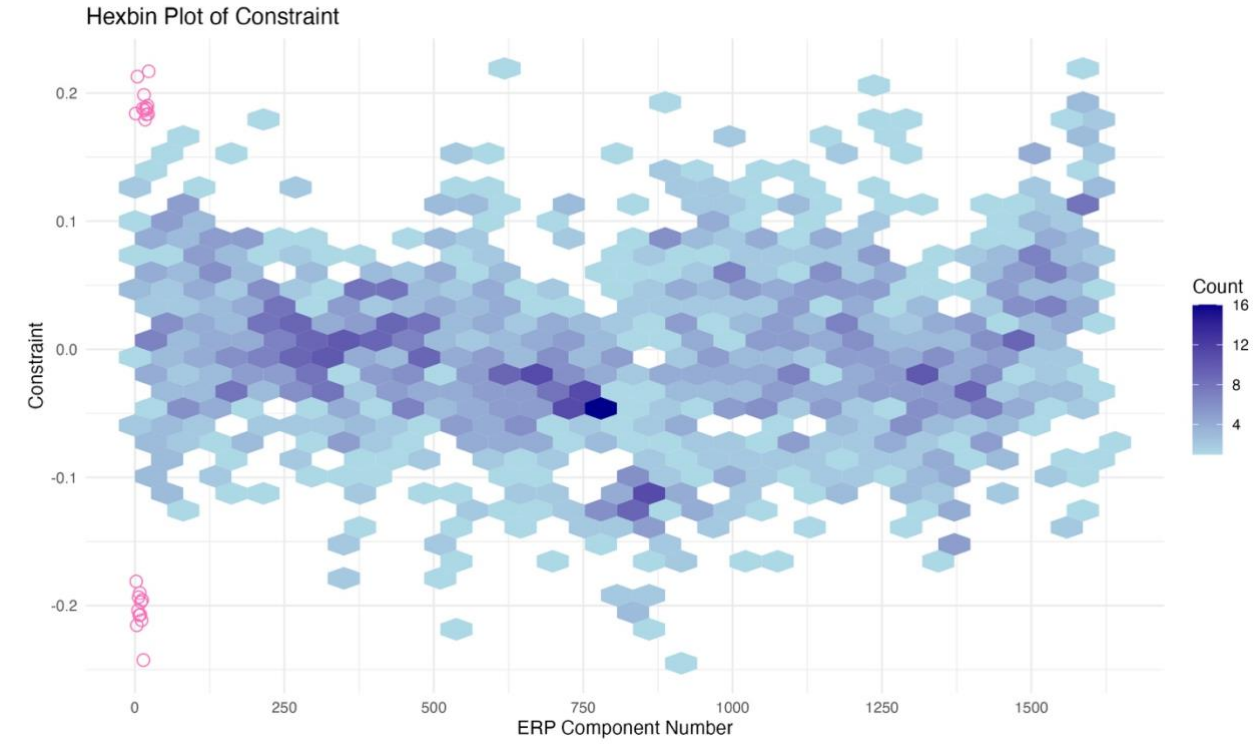
	K-S	p	Lilliefors	p	Skew	Kurtosis	White's	White's p
Internalizing	.03	.25	.03	.02	.19	2.77	1.99	.05
Externalizing	.03	.21	.03	.01	.29	2.91	9.15	.00
p-factor	.04	.02	.04	.00	.20	2.68	7.22	.00

Note: There are mixed reviews based on the table, but as noted for a large sample size, a visual inspection is most pertinent.

Appendix B: Data distribution




Hexbin Plot of p-factor
p-factor
0.2
0.1
0.0
-0.1
-0.2
0
250
500
750
1000
1250
1500
ERP Component Number
Count
12.5
10.0
7.5
5.0
2.5


Hexbin Plot of Constraint
Constraint
0.2
0.1
0.0
-0.1
-0.2
0
250
500
750
1000
1250
1500
ERP Component Number
Count
16
12
8
4

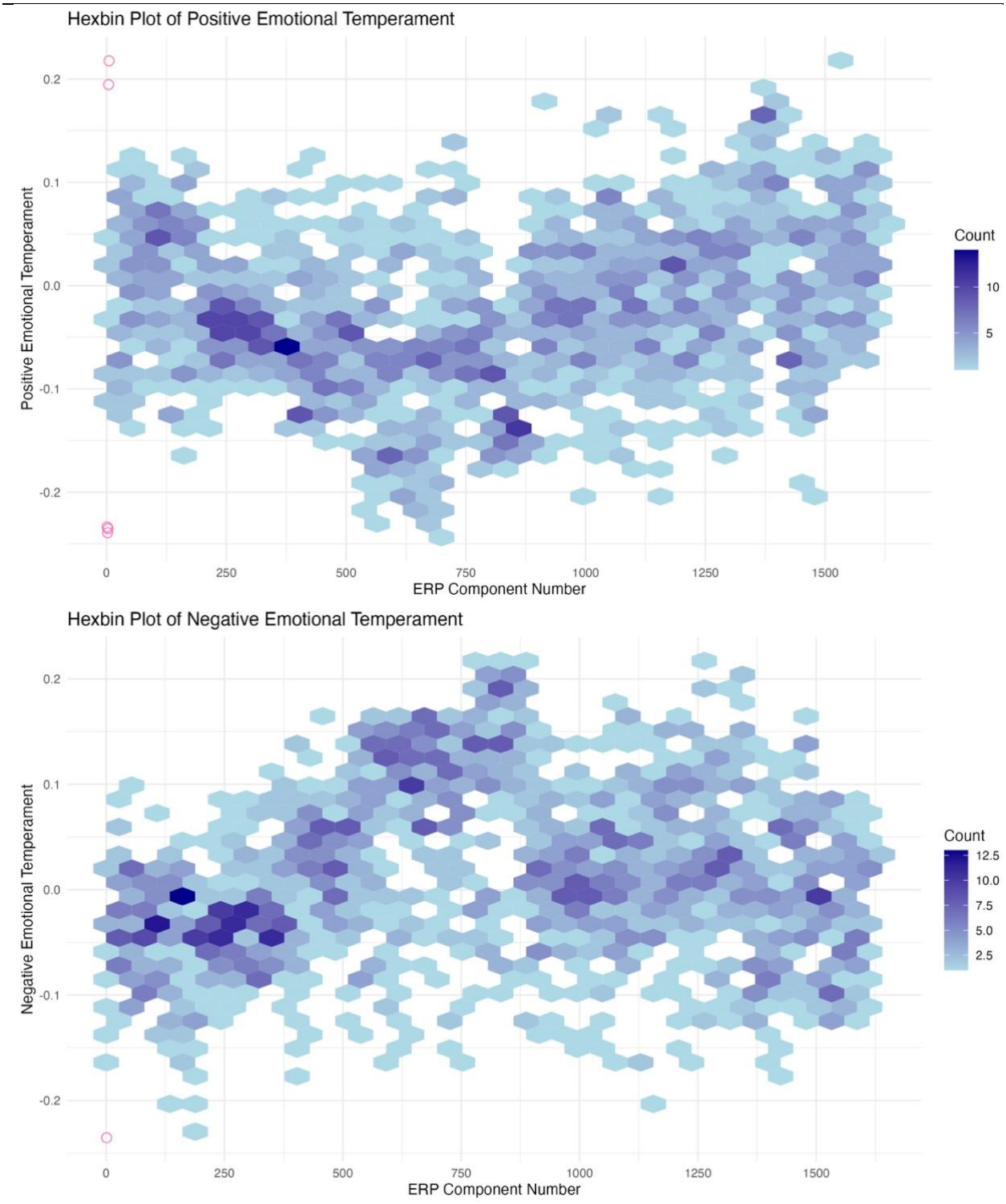


Figure B1. Data distribution hexbin plots of each variables ordered by frequency.
Note. Delta = 1 through 540; Theta = 541 through 942; Alpha = 943 through 1110; Beta-1 = 1111 through 1278; Beta-2 = 1279 through 1446; Gamma = 1447 through 1614.

Appendix C: Influence Analysis - Cook's distance, Leverage, and Studentized Residual plots

Influence analysis

Influence plots visually depicted the relationships among leverage (hat-values), standardized residuals, and Cook's distance for each observation and each regression analysis separately. Robustness checks using influence diagnostics (described in Methods and in the below table) indicated that the regression findings remained consistent after removing potentially influential observations, supporting the stability of results.

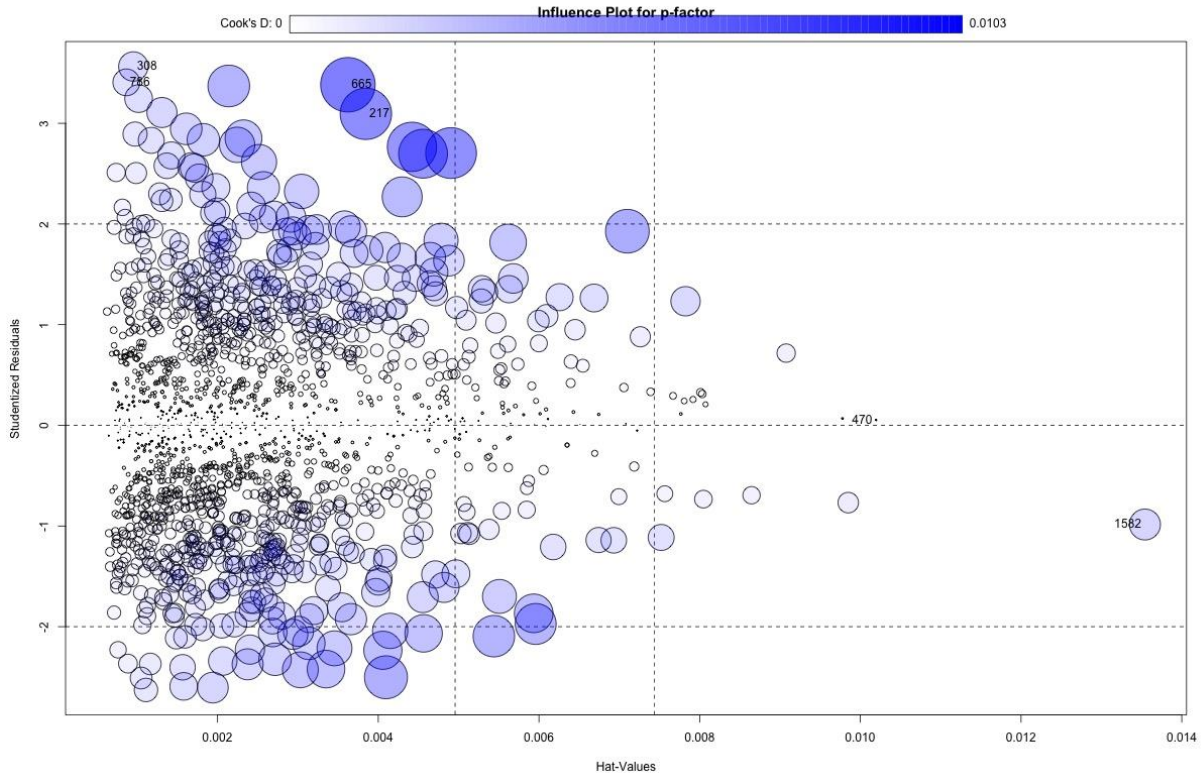
The result column of the table is indicating whether or not the model is consistent with the above multiple regression results in terms of the direction, strength ordering of the predictors, significance of beta coefficients, and significance of the overall model (F-statistic significance). Although, this does not mean that all regression result values are identical, but rather indicates that there was not difference in the overall model results when removing observations in stepwise regression analyses.

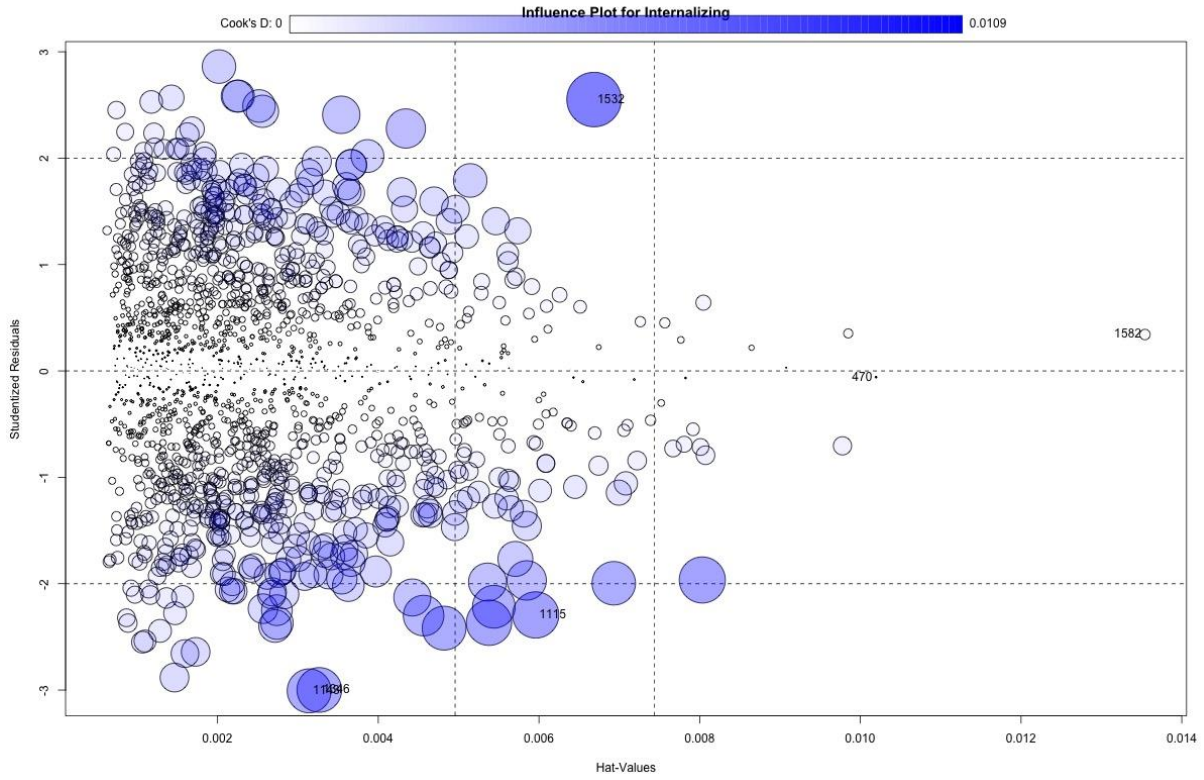
Table C1. Meta-analysis regressions: Influence statistics review.

Criterion Name	Cutoff Calculation	Observations Removed	Results
Cook's distance	>.0025 *	Internalizing: 66 Externalizing: 80 p-factor: 83	Overall model is consistent for all three outcome variables based on criteria
Leverage	>.0045	Internalizing: 118 Externalizing: 118 p-factor: 118	Overall model is consistent for all three outcome variables based on criteria
Studentized Residuals	±2 = Moderate ±3 = Extreme	Moderate**: Internalizing: 56 Externalizing: 62 p-factor: 57 Extreme: Internalizing: 2 Externalizing: 1 p-factor: 7	Overall model is consistent for all three outcome variables based on both criteria

*Cook's d cutoff is based on large model computation

**Extreme outliers that would also meet this criteria are not included in the count for moderate outliers.


Influence Plot for p-factor
Cook's D: 0
0.0103
308
786
665
217
470
1582
Studentized Residuals
Hat-Values
0.002
0.004
0.006
0.008
0.010
0.012
0.014


Influence Plot for Internalizing
Cook's D: 0
0.0109
1532
1582
470
1115
Studentized Residuals
Hat-Values
0.002
0.004
0.006
0.008
0.010
0.012
0.014

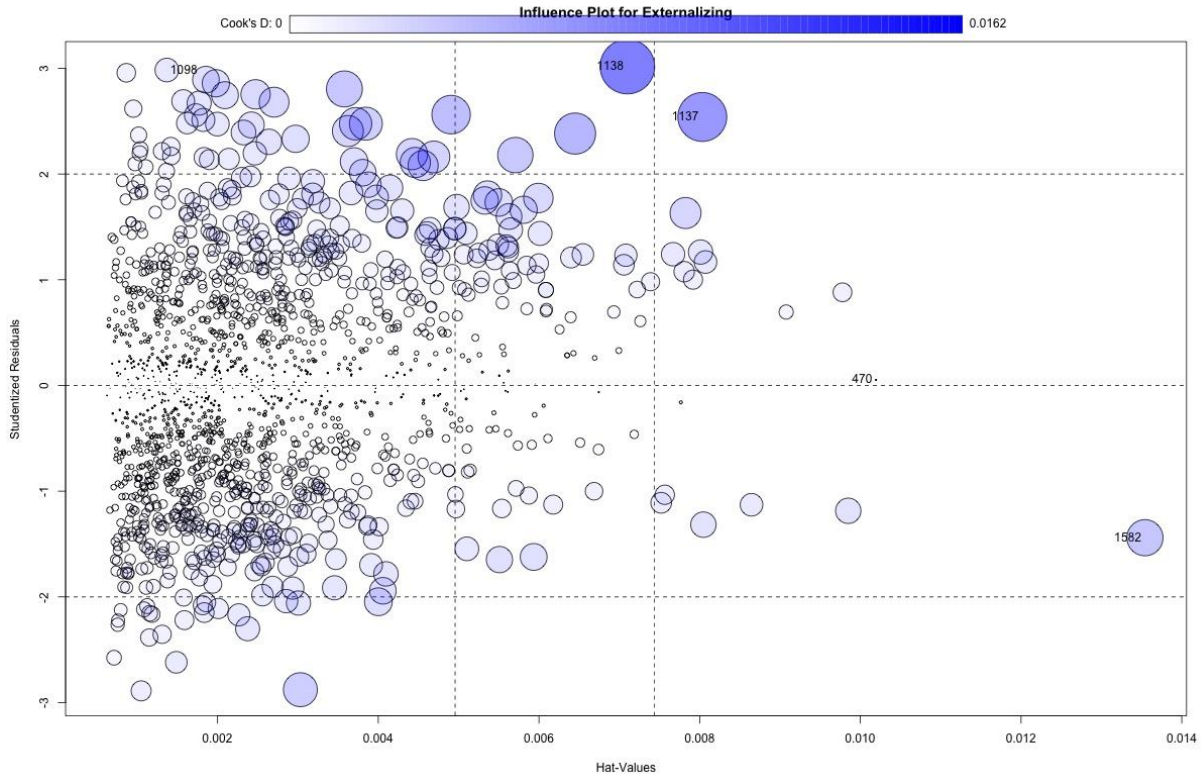
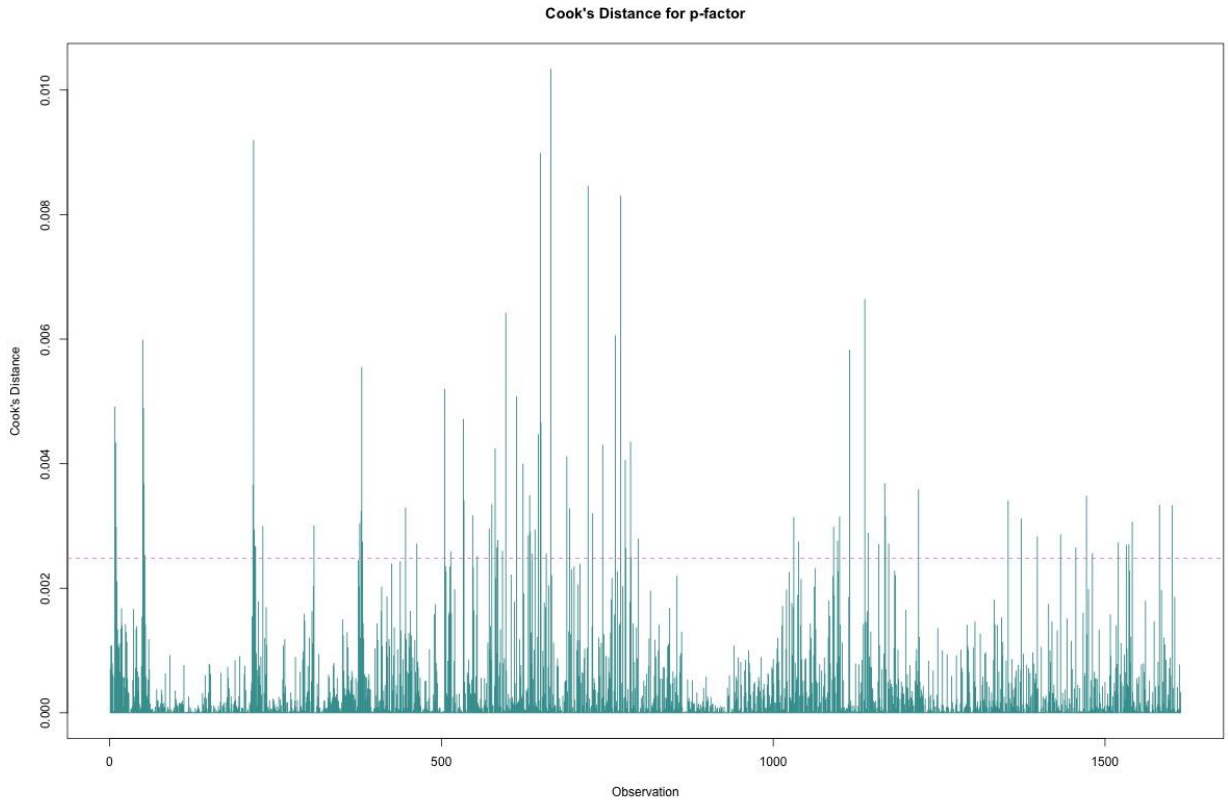
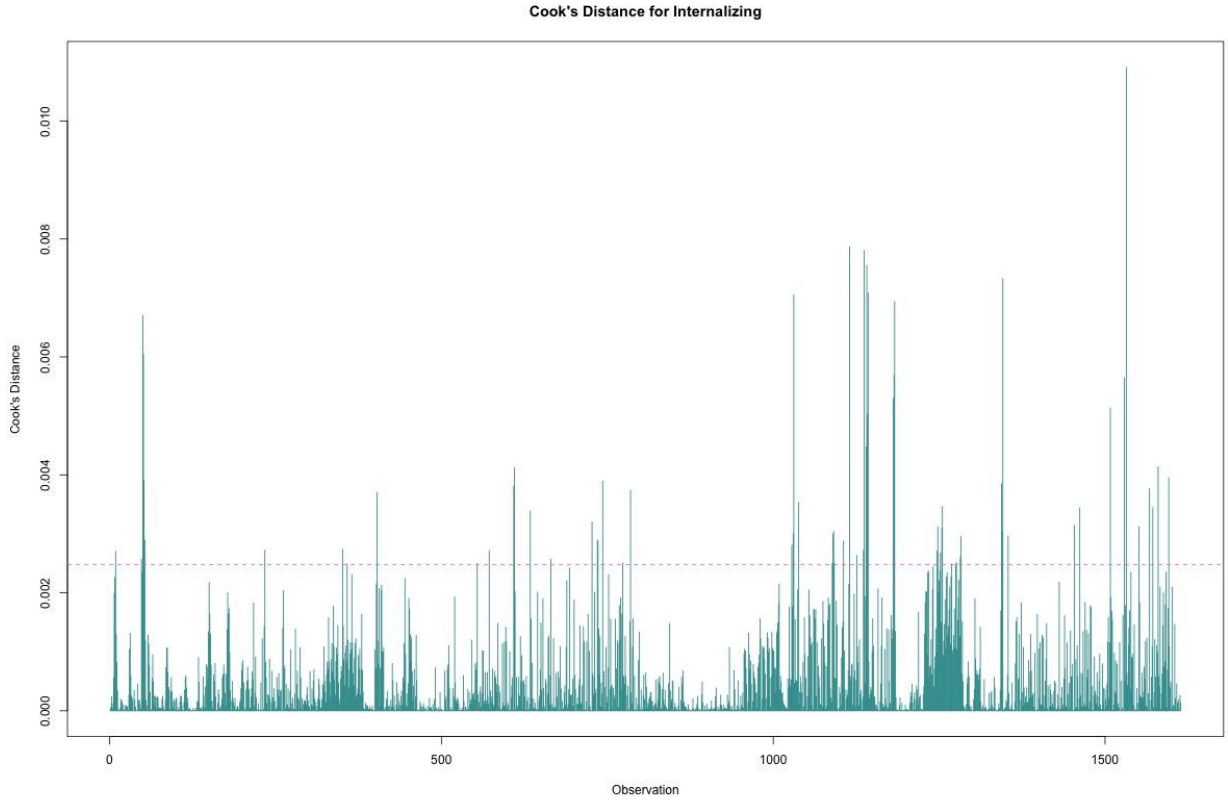


Figure C1. Influence plots of the independent variable measures for each of the dependent variable regression analyses.


Cook's Distance for p-factor
Cook's Distance
0.000
0.002
0.004
0.006
0.008
0.010
0
500
1000
1500
Observation


Cook's Distance for Internalizing
Cook's Distance
0.000
0.002
0.004
0.006
0.008
0.010
0
500
1000
1500
Observation

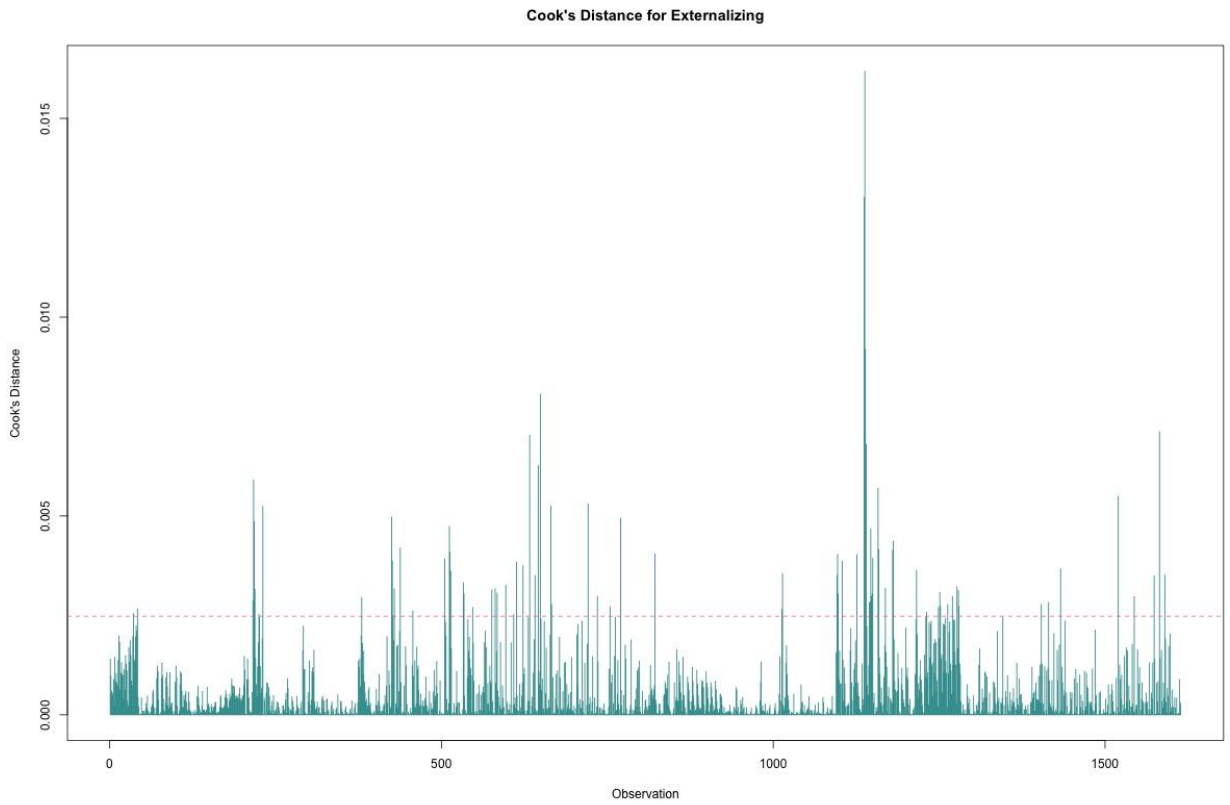
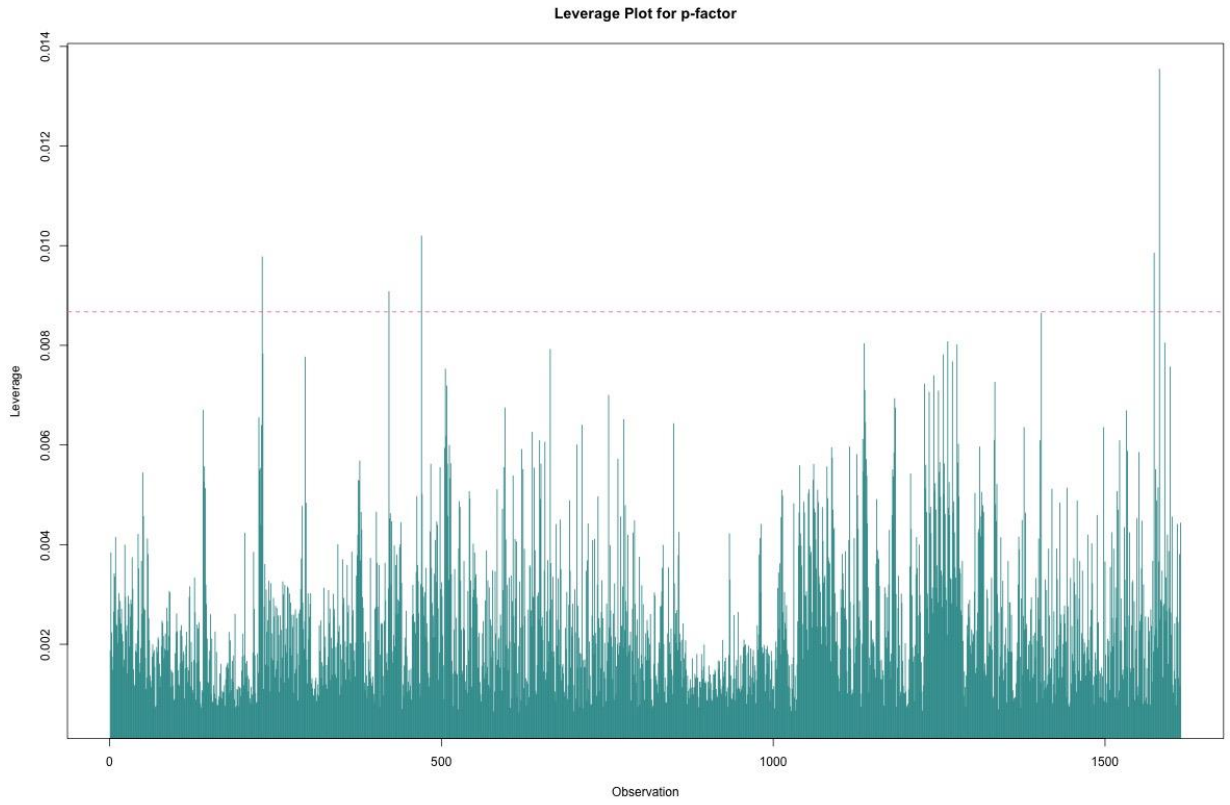
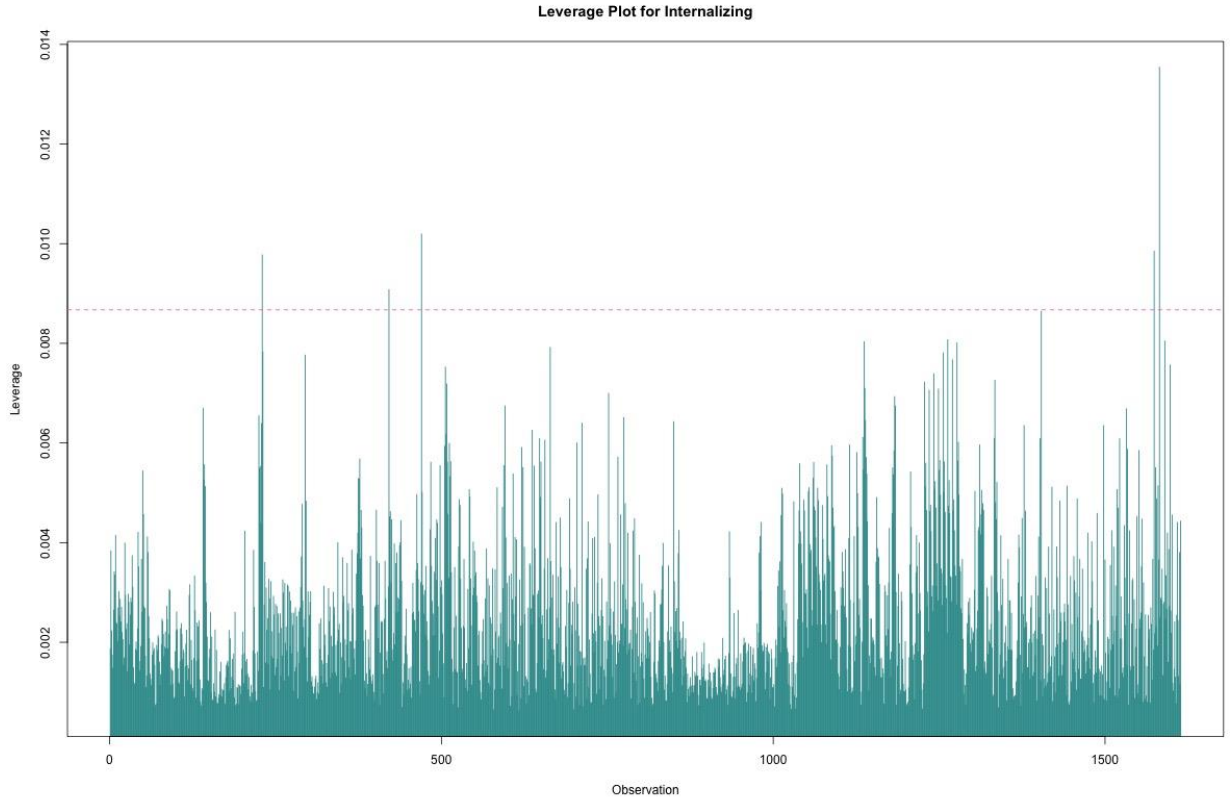


Figure C2. Cook's distance plots of the independent variable measures for each of the dependent variable regression analyses.


Leverage Plot for p-factor
Leverage
0.002
0.004
0.006
0.008
0.010
0.012
0.014
0
500
1000
1500
Observation


Leverage Plot for Internalizing
Leverage
0.002
0.004
0.006
0.008
0.010
0.012
0.014
0
500
1000
1500
Observation

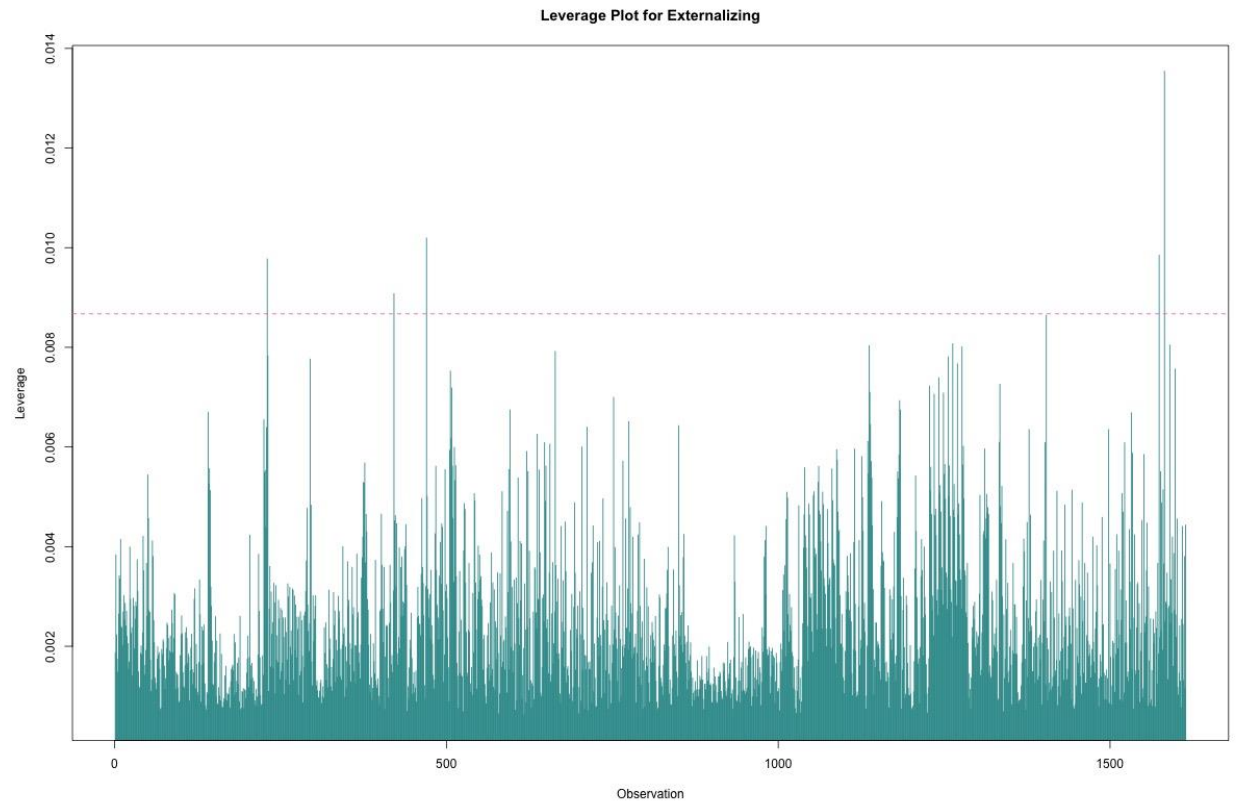
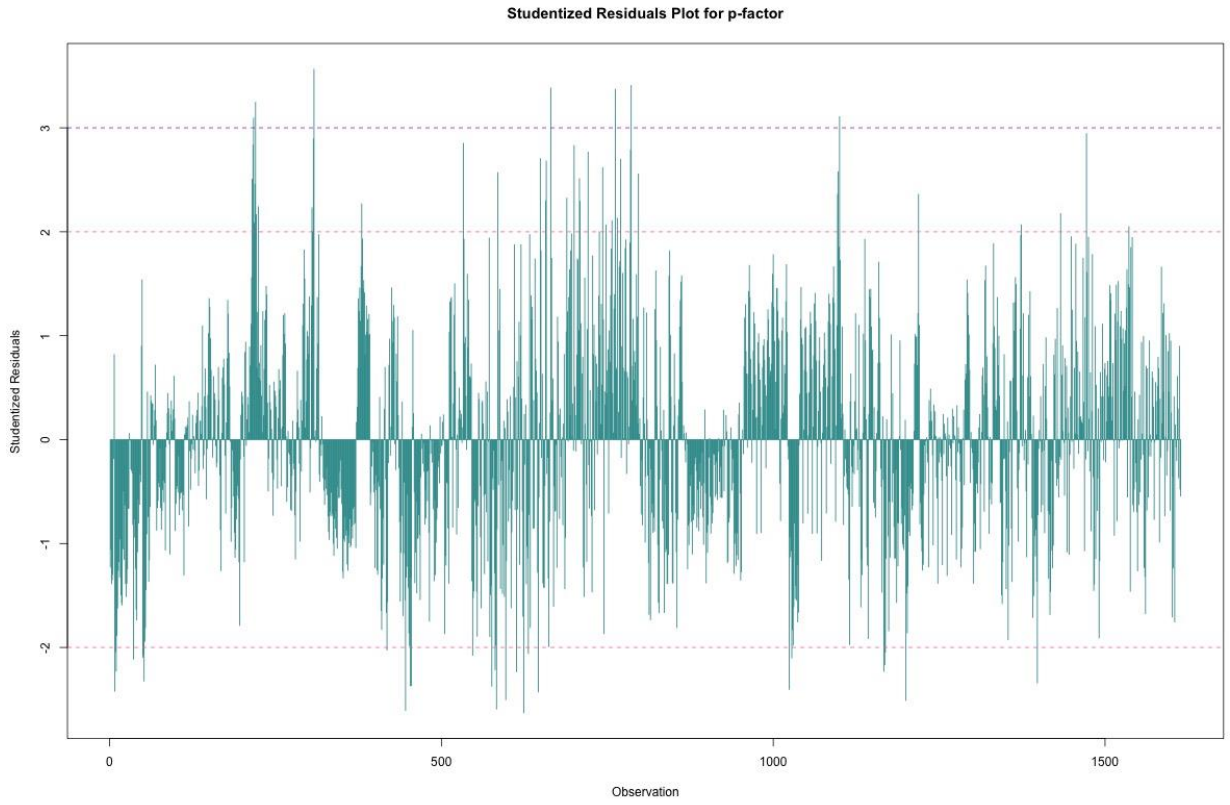
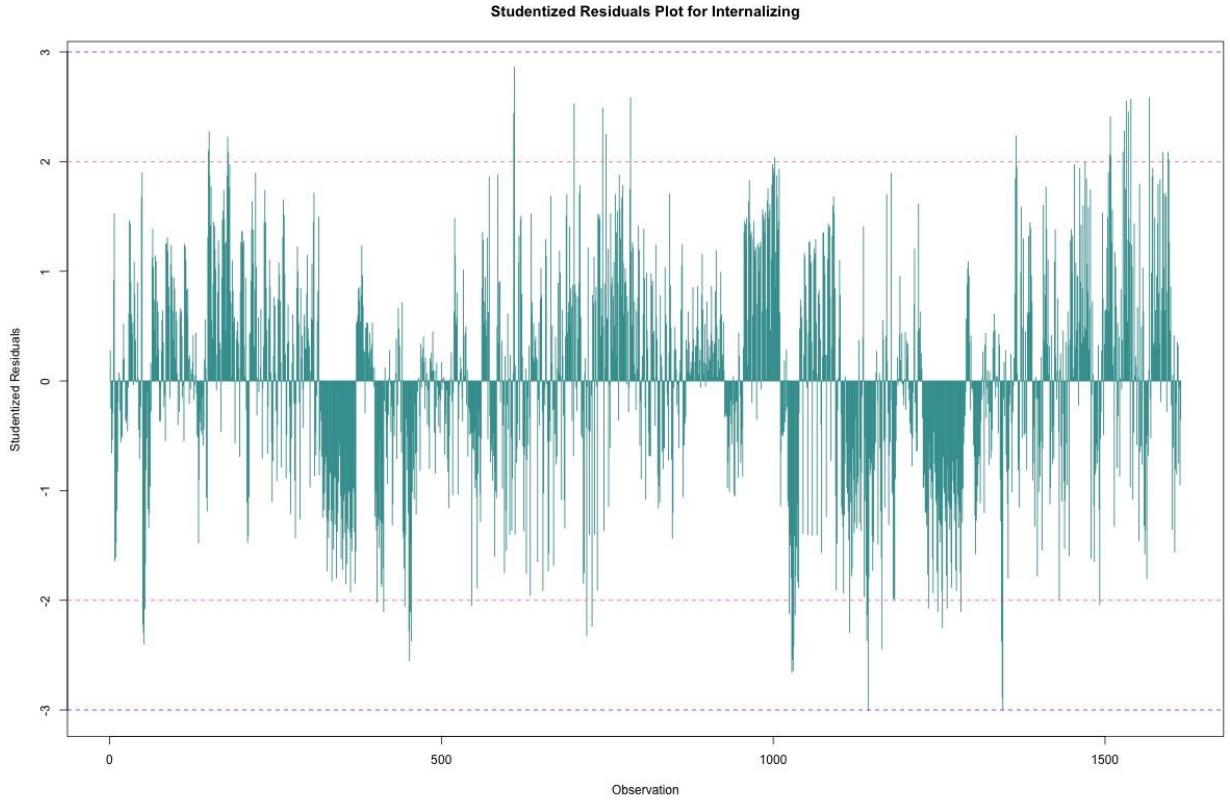


Figure C3. Leverage plots of the independent variable measures for each of the dependent variable regression analyses.


Studentized Residuals Plot for p-factor
Studentized Residuals
Observation
3
2
1
0
-1
-2
0
500
1000
1500


Studentized Residuals Plot for Internalizing
Studentized Residuals
Observation
3
2
1
0
-1
-2
-3
0
500
1000
1500

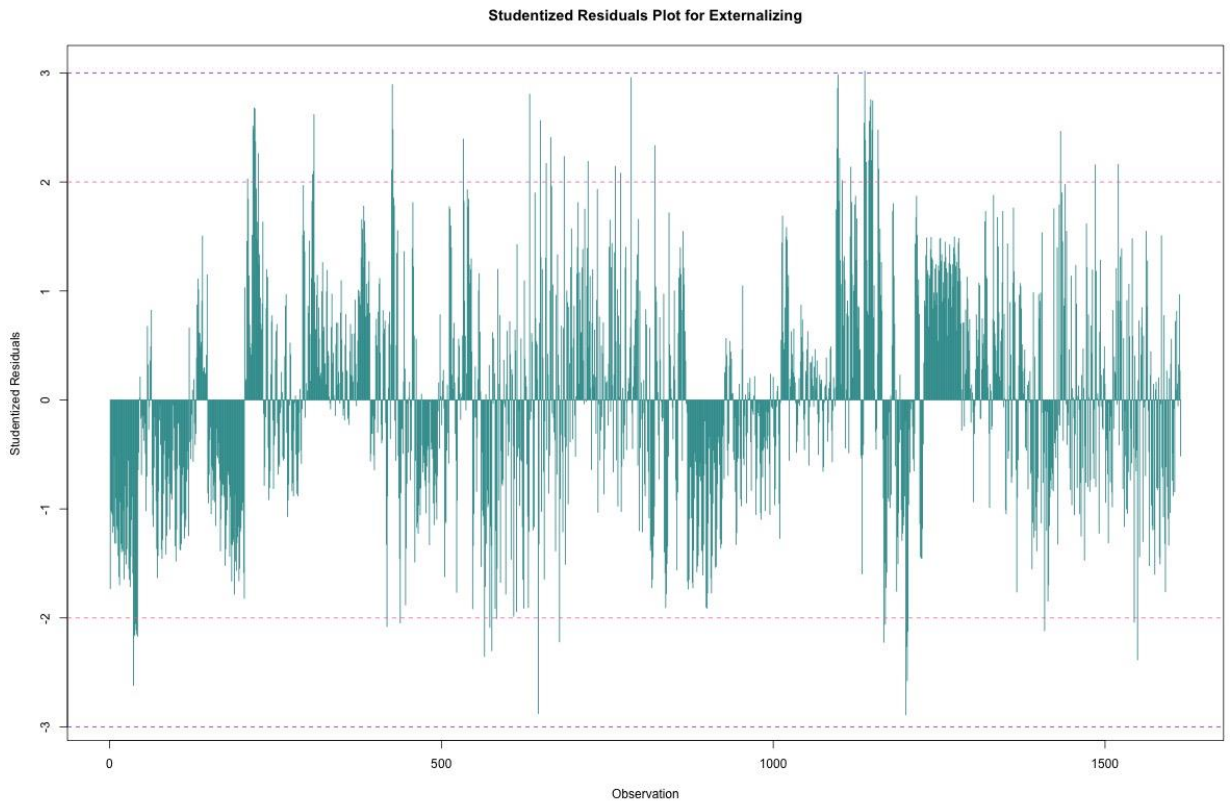


Figure C4. Studentized residuals plots of the independent variable measures for each of the dependent variable regression analyses.

Appendix D: Main effect post-hoc analyses

Internalizing: Frequency

Table D1. Meta-analysis general linear models: Frequency post-hoc results predicting INT.

(I) Frequency	(J) Frequency	Mean Difference (J-I)	Std. Error	p-value	CI
Delta	Theta	-.02	.00	<.001	[-.03, -.01]
	Alpha	-.03	.01	<.001	[-.05, -.01]
	Beta1	-.12	.01	<.001	[-.14, -.10]
	Beta2	-.13	.01	<.001	[-.14, -.11]
	Gamma	-.01	.01	.243	[-.03, .00]
Theta	Delta	.02	.00	<.001	[.01, .03]
	Alpha	-.01	.01	.551	[-.03, .01]
	Beta1	-.10	.01	<.001	[-.12, -.08]
	Beta2	-.10	.01	<.001	[-.12, -.09]
	Gamma	.01	.01	.805	[-.01, .03]
Alpha	Delta	.03	.01	<.001	[.01, .05]
	Theta	.01	.01	.551	[-.01, .03]
	Beta1	-.09	.01	<.001	[-.11, -.07]
	Beta2	-.09	.01	<.001	[-.11, -.07]
	Gamma	.02	.01	.133	[-.00, .04]
Beta1	Delta	.12	.01	<.001	[.10, .14]
	Theta	.10	.01	<.001	[.08, .12]
	Alpha	.09	.01	<.001	[.07, .11]
	Beta2	-.01	.01	.977	[-.03, .02]
	Gamma	.11	.01	<.001	[.09, .13]
Beta2	Delta	.13	.01	<.001	[.11, .14]
	Theta	.10	.01	<.001	[.09, .12]
	Alpha	.09	.01	<.001	[.07, .11]
	Beta1	.01	.01	.977	[-.02, .03]
	Gamma	.11	.01	<.001	[.09, .13]
Gamma	Delta	.01	.01	.243	[-.00, .03]
	Theta	-.01	.01	.805	[-.03, .01]
	Alpha	-.02	.01	.133	[-.04, .00]
	Beta1	-.11	.01	<.001	[-.13, -.09]
	Beta2	-.11	.01	<.001	[-.13, -.09]

Internalizing: Electrode

Table D2. Meta-analysis general linear models: Electrode post-hoc results predicting INT.

(I) Frequency	(J) Frequency	Mean Difference (J-I)	Std. Error	p-value	CI
FZ	FCZ no ICPS	-.03	.01	.002	[-.05, -.01]
	FCZ ICPS	-.04	.01	<.001	[-.06, -.02]
	F6 ICPS	-.01	.01	.966	[-.03, .01]
	CZ	-.03	.01	.005	[-.05, -.01]
	CPZ	-.01	.01	.872	[-.03, .01]
	PZ	-.01	.01	.69	[-.04, .01]
	POZ	.01	.01	.966	[-.01, .03]
	OZ	.03	.01	.022	[.00, .05]
FCZ no ICPS	FZ	.03	.01	.002	[.01, .05]
	FCZ ICPS	-.01	.01	.955	[-.03, .01]
	F6 ICPS	.02	.01	.035	[.00, .04]
	CZ	.00	.01	1	[-.02, .03]
	CPZ	.02	.01	.215	[-.00, .04]
	PZ	.02	.01	.397	[-.01, .04]
	POZ	.04	.01	<.001	[.02, .06]
	OZ	.06	.01	<.001	[.03, .08]
FCZ ICPS	FZ	.04	.01	<.001	[.02, .06]
	FCZ no ICPS	.01	.01	.955	[-.01, .03]
	F6 ICPS	.03	.01	<.001	[.01, .05]
	CZ	.01	.01	.876	[-.01, .03]
	CPZ	.03	.01	.002	[.01, .05]
	PZ	.03	.01	.009	[.00, .05]
	POZ	.05	.01	<.001	[.03, .07]
	OZ	.06	.01	<.001	[.04, .09]
F6 ICPS	FZ	.01	.01	.966	[-.01, .03]
	FCZ no ICPS	-.02	.01	.035	[-.04, -.00]
	FCZ ICPS	-.03	.01	<.001	[-.05, -.01]
	CZ	-.02	.01	.073	[-.04, .00]
	CPZ	.00	.01	1	[-.02, .02]
	PZ	-.01	.01	.997	[-.03, .02]
	POZ	.02	.01	.281	[-.00, .04]
	OZ	.03	.01	<.001	[.01, .06]
CZ	FZ	.03	.01	.005	[.01, .05]
	FCZ no ICPS	.00	.01	1	[-.03, .02]
	FCZ ICPS	-.01	.01	.876	[-.03, .01]
	F6 ICPS	.02	.01	.073	[-.00, .04]
	CPZ	.02	.01	.336	[-.01, .04]
	PZ	.02	.01	.552	[-.01, .04]
	POZ	.04	.01	<.001	[.01, .06]
	OZ	.05	.01	<.001	[.03, .08]

CPZ	FZ	.01	.01	.872	[-.01, .03]
	FCZ no ICPS	-.02	.01	.215	[-.04, .00]
	FCZ ICPS	-.03	.01	.002	[-.05, -.01]
	F6 ICPS	.00	.01	1	[-.02, .02]
	CZ	-.02	.01	.336	[-.04, .01]
	PZ	.00	.01	1	[-.03, .02]
	POZ	.02	.01	.181	[-.00, .04]
	OZ	.04	.01	<.001	[.01, .06]
PZ	FZ	.01	.01	.69	[-.01, .04]
	FCZ no ICPS	-.02	.01	.397	[-.04, .01]
	FCZ ICPS	-.03	.01	.009	[-.05, -.00]
	F6 ICPS	.01	.01	.997	[-.02, .03]
	CZ	-.02	.01	.552	[-.04, .01]
	CPZ	.00	.01	1	[-.02, .03]
	POZ	.02	.01	.081	[-.00, .05]
	OZ	.04	.01	<.001	[.02, .06]
POZ	FZ	-.01	.01	.966	[-.03, .01]
	FCZ no ICPS	-.04	.01	<.001	[-.06, -.02]
	FCZ ICPS	-.05	.01	<.001	[-.07, -.03]
	F6 ICPS	-.02	.01	.281	[-.04, .00]
	CZ	-.04	.01	<.001	[-.06, -.01]
	CPZ	-.02	.01	.181	[-.04, .00]
	PZ	-.02	.01	.081	[-.05, .00]
	OZ	.02	.01	.395	[-.01, .04]
OZ	FZ	-.03	.01	.022	[-.05, -.00]
	FCZ no ICPS	-.06	.01	<.001	[-.08, -.03]
	FCZ ICPS	-.06	.01	<.001	[-.09, -.04]
	F6 ICPS	-.03	.01	<.001	[-.06, -.01]
	CZ	-.05	.01	<.001	[-.08, -.03]
	CPZ	-.04	.01	<.001	[-.06, -.01]
	PZ	-.04	.01	<.001	[-.06, -.02]
	POZ	-.02	.01	.395	[-.04, .01]

Internalizing: Condition

Table D3. Meta-analysis general linear models: Condition post-hoc results predicting INT.

(I) Condition	**(J) Condition**	**Mean Difference (I-J)**	**Std. Error**	**p-value**	**CI**
Loss	Gain	.00	.00	.964	[-.01, .01]
	Nogo	.02	.00	<.001	[.01, .03]
	Go	.02	.00	<.001	[.01, .03]
Gain	Loss	.00	.00	.964	[-.01, .01]
	Nogo	.02	.00	.001	[.01, .03]

	Go	.02	.00	.002	[.01, .03]
Nogo	Loss	-.02	.00	<.001	[-.03, -.01]
	Gain	-.02	.00	.001	[-.03, -.01]
	Go	.00	.00	1	[-.01, .01]
Go	Loss	-.02	.00	<.001	[-.03, -.01]
	Gain	-.02	.00	.002	[-.03, -.01]
	Nogo	.00	.00	1	[-.01, .01]

Externalizing: Frequency

Table D4. Meta-analysis general linear models: Frequency post-hoc results predicting EXT.

(I) Frequency	(J) Frequency	Mean Difference (J-I)	Std. Error	p-value	CI
Delta	Theta	-.02	.00	.014	[-.03, -.00]
	Alpha	.07	.01	<.001	[.06, .09]
	Beta1	.00	.01	.974	[-.01, .02]
	Beta2	-.10	.01	<.001	[-.12, -.08]
	Gamma	-.17	.01	<.001	[-.19, -.16]
Theta	Delta	.02	.00	.014	[.00, .03]
	Alpha	.09	.01	<.001	[.07, .11]
	Beta1	.02	.01	.026	[.00, .04]
	Beta2	-.08	.01	<.001	[-.10, -.07]
	Gamma	-.16	.01	<.001	[-.18, -.14]
Alpha	Delta	-.07	.01	<.001	[-.09, -.06]
	Theta	-.09	.01	<.001	[-.11, -.07]
	Beta1	-.07	.01	<.001	[-.09, -.05]
	Beta2	-.17	.01	<.001	[-.20, -.15]
	Gamma	-.25	.01	<.001	[-.27, -.23]
Beta1	Delta	.00	.01	.974	[-.02, .01]
	Theta	-.02	.01	.026	[-.04, -.00]
	Alpha	.07	.01	<.001	[.05, .09]
	Beta2	-.11	.01	<.001	[-.13, -.08]
	Gamma	-.18	.01	<.001	[-.20, -.16]
Beta2	Delta	.10	.01	<.001	[.08, .12]
	Theta	.08	.01	<.001	[.07, .10]
	Alpha	.17	.01	<.001	[.15, .20]
	Beta1	.11	.01	<.001	[.08, .13]
	Gamma	-.07	.01	<.001	[-.10, -.05]
Gamma	Delta	.17	.01	<.001	[.16, .19]
	Theta	.16	.01	<.001	[.14, .18]
	Alpha	.25	.01	<.001	[.23, .27]

	Beta1	.18	.01	<.001	[.16, .20]
	Beta2	.07	.01	<.001	[.05, .10]

Externalizing: Electrode

Table D5. Meta-analysis general linear models: Electrode post-hoc results predicting EXT.

(I) Frequency	(J) Frequency	Mean Difference (J-I)	Std. Error	p-value	CI
FZ	FCZ no ICPS	.01	.01	.77	[-.01, .04]
	FCZ ICPS	-.02	.01	.141	[-.04, .00]
	F6 ICPS	.01	.01	.729	[-.01, .04]
	CZ	.02	.01	.48	[-.01, .04]
	CPZ	.02	.01	.096	[-.00, .05]
	PZ	.01	.01	.983	[-.02, .03]
	POZ	.00	.01	1	[-.02, .03]
	OZ	-.01	.01	.899	[-.04, .01]
FCZ no ICPS	FZ	-.01	.01	.77	[-.04, .01]
	FCZ ICPS	-.03	.01	<.001	[-.06, -.01]
	F6 ICPS	.00	.01	1	[-.02, .02]
	CZ	.00	.01	1	[-.02, .03]
	CPZ	.01	.01	.953	[-.02, .04]
	PZ	-.01	.01	.999	[-.03, .02]
	POZ	-.01	.01	.81	[-.04, .01]
	OZ	-.02	.01	.058	[-.05, .00]
FCZ ICPS	FZ	.02	.01	.141	[-.00, .04]
	FCZ no ICPS	.03	.01	<.001	[.01, .06]
	F6 ICPS	.03	.01	<.001	[.01, .05]
	CZ	.04	.01	<.001	[.01, .06]
	CPZ	.04	.01	<.001	[.02, .07]
	PZ	.03	.01	.004	[.01, .05]
	POZ	.02	.01	.117	[-.00, .04]
	OZ	.01	.01	.957	[-.01, .03]
F6 ICPS	FZ	-.01	.01	.729	[-.04, .01]
	FCZ no ICPS	.00	.01	1	[-.02, .02]
	FCZ ICPS	-.03	.01	<.001	[-.05, -.01]
	CZ	.00	.01	1	[-.02, .03]
	CPZ	.01	.01	.893	[-.01, .03]
	PZ	.00	.01	1	[-.03, .02]
	POZ	-.01	.01	.776	[-.04, .01]
	OZ	-.02	.01	.032	[-.05, -.00]
CZ	FZ	-.02	.01	.48	[-.04, .01]
	FCZ no ICPS	.00	.01	1	[-.03, .02]
	FCZ ICPS	-.04	.01	<.001	[-.06, -.01]
	F6 ICPS	.00	.01	1	[-.03, .02]

	CPZ	.01	.01	.997	[-.02, .03]
	PZ	-.01	.01	.98	[-.03, .02]
	POZ	-.02	.01	.529	[-.04, .01]
	OZ	-.03	.01	.015	[-.05, -.00]
CPZ	FZ	-.02	.01	.096	[-.05, .00]
	FCZ no ICPS	-.01	.01	.953	[-.04, .02]
	FCZ ICPS	-.04	.01	<.001	[-.07, -.02]
	F6 ICPS	-.01	.01	.893	[-.03, .01]
	CZ	-.01	.01	.997	[-.03, .02]
	PZ	-.01	.01	.654	[-.04, .01]
	POZ	-.02	.01	.115	[-.05, .00]
	OZ	-.03	.01	<.001	[-.06, -.01]
PZ	FZ	-.01	.01	.983	[-.03, .02]
	FCZ no ICPS	.01	.01	.999	[-.02, .03]
	FCZ ICPS	-.03	.01	.004	[-.05, -.01]
	F6 ICPS	.00	.01	1	[-.02, .03]
	CZ	.01	.01	.98	[-.02, .03]
	CPZ	.01	.01	.654	[-.01, .04]
	POZ	-.01	.01	.989	[-.03, .02]
	OZ	-.02	.01	.269	[-.04, .01]
POZ	FZ	.00	.01	1	[-.03, .02]
	FCZ no ICPS	.01	.01	.81	[-.01, .04]
	FCZ ICPS	-.02	.01	.117	[-.04, .00]
	F6 ICPS	.01	.01	.776	[-.01, .04]
	CZ	.02	.01	.529	[-.01, .04]
	CPZ	.02	.01	.115	[-.00, .05]
	PZ	.01	.01	.989	[-.02, .03]
	OZ	-.01	.01	.871	[-.04, .01]
OZ	FZ	.01	.01	.899	[-.01, .04]
	FCZ no ICPS	.02	.01	.058	[-.00, .05]
	FCZ ICPS	-.01	.01	.957	[-.03, .01]
	F6 ICPS	.02	.01	.032	[.00, .05]
	CZ	.03	.01	.015	[.00, .05]
	CPZ	.03	.01	<.001	[.01, .06]
	PZ	.02	.01	.269	[-.01, .04]
	POZ	.01	.01	.871	[-.01, .04]

Externalizing: Condition

Table D6. Meta-analysis general linear models: Condition post-hoc results predicting EXT.

(I) Condition	(J) Condition	Mean Difference (I-J)	Std. Error	p-value	CI
Loss	Gain	.00	.01	.937	[-.02, .01]
	Nogo	.03	.01	<.001	[.02, .05]
	Go	.02	.01	<.001	[.01, .03]
Gain	Loss	.00	.01	.937	[-.01, .02]
	Nogo	.04	.01	<.001	[.02, .05]
	Go	.02	.01	<.001	[.01, .04]
Nogo	Loss	-.03	.01	<.001	[-.05, -.02]
	Gain	-.04	.01	<.001	[-.05, -.02]
	Go	-.01	.01	.026	[-.03, -.00]
Go	Loss	-.02	.01	<.001	[-.03, -.01]
	Gain	-.02	.01	<.001	[-.04, -.01]
	Nogo	.01	.01	.026	[.00, .03]

p-factor: Frequency

Table D7. Meta-analysis general linear models: Frequency post-hoc results predicting p-factor.

(I) Frequency	(J) Frequency	Mean Difference (J-I)	Std. Error	p-value	CI
Delta	Theta	-.02	.00	<.001	[-.04, -.01]
	Alpha	.03	.01	<.001	[.01, .04]
	Beta1	-.07	.01	<.001	[-.09, -.06]
	Beta2	-.15	.01	<.001	[-.16, -.13]
	Gamma	-.12	.01	<.001	[-.14, -.10]
Theta	Delta	.02	.00	<.001	[.01, .04]
	Alpha	.05	.01	<.001	[.03, .07]
	Beta1	-.05	.01	<.001	[-.07, -.03]
	Beta2	-.12	.01	<.001	[-.14, -.11]
	Gamma	-.10	.01	<.001	[-.11, -.08]
Alpha	Delta	-.03	.01	<.001	[-.04, -.01]
	Theta	-.05	.01	<.001	[-.07, -.03]
	Beta1	-.10	.01	<.001	[-.12, -.08]
	Beta2	-.17	.01	<.001	[-.19, -.15]
	Gamma	-.15	.01	<.001	[-.17, -.12]
Beta1	Delta	.07	.01	<.001	[.06, .09]
	Theta	.05	.01	<.001	[.03, .07]

	Alpha	.10	.01	<.001	[.08, .12]
	Beta2	-.07	.01	<.001	[-.09, -.05]
	Gamma	-.05	.01	<.001	[-.07, -.03]
Beta2	Delta	.15	.01	<.001	[.13, .16]
	Theta	.12	.01	<.001	[.11, .14]
	Alpha	.17	.01	<.001	[.15, .19]
	Beta1	.07	.01	<.001	[.05, .09]
	Gamma	.03	.01	.002	[.01, .05]
Gamma	Delta	.12	.01	<.001	[.10, .14]
	Theta	.10	.01	<.001	[.08, .11]
	Alpha	.15	.01	<.001	[.12, .17]
	Beta1	.05	.01	<.001	[.03, .07]
	Beta2	-.03	.01	.002	[-.05, -.01]

p-factor: Electrode

Table D8. Meta-analysis general linear models: Electrode post-hoc results predicting p-factor.

(I) Frequency	**(J) Frequency**	**Mean Difference (J-I)**	**Std. Error**	**p-value**	**CI**
FZ	FCZ no ICPS	-.01	.01	.866	[-.03, .01]
	FCZ ICPS	-.04	.01	<.001	[-.06, -.01]
	F6 ICPS	.00	.01	.998	[-.02, .03]
	CZ	-.01	.01	.956	[-.03, .01]
	CPZ	.01	.01	.99	[-.02, .03]
	PZ	.00	.01	1	[-.03, .02]
	POZ	.00	.01	.999	[-.02, .03]
	OZ	.01	.01	.944	[-.01, .03]
FCZ no ICPS	FZ	.01	.01	.866	[-.01, .03]
	FCZ ICPS	-.02	.01	.011	[-.05, -.00]
	F6 ICPS	.02	.01	.317	[-.01, .04]
	CZ	.00	.01	1	[-.02, .03]
	CPZ	.02	.01	.273	[-.01, .04]
	PZ	.01	.01	.975	[-.01, .03]
	POZ	.02	.01	.455	[-.01, .04]
	OZ	.02	.01	.137	[-.00, .04]
FCZ ICPS	FZ	.04	.01	<.001	[.01, .06]
	FCZ no ICPS	.02	.01	.011	[.00, .05]
	F6 ICPS	.04	.01	<.001	[.02, .06]
	CZ	.03	.01	.004	[.01, .05]
	CPZ	.04	.01	<.001	[.02, .06]
	PZ	.03	.01	<.001	[.01, .05]
	POZ	.04	.01	<.001	[.02, .06]
	OZ	.04	.01	<.001	[.02, .07]

F6 ICPS	FZ	.00	.01	.998	[-.03, .02]
	FCZ no ICPS	-.02	.01	.317	[-.04, .01]
	FCZ ICPS	-.04	.01	<.001	[-.06, -.02]
	CZ	-.01	.01	.514	[-.04, .01]
	CPZ	.00	.01	1	[-.02, .02]
	PZ	-.01	.01	.967	[-.03, .01]
	POZ	.00	.01	1	[-.02, .02]
	OZ	.00	.01	.999	[-.02, .03]
CZ	FZ	.01	.01	.956	[-.01, .03]
	FCZ no ICPS	.00	.01	1	[-.03, .02]
	FCZ ICPS	-.03	.01	.004	[-.05, -.01]
	F6 ICPS	.01	.01	.514	[-.01, .04]
	CPZ	.02	.01	.442	[-.01, .04]
	PZ	.01	.01	.996	[-.02, .03]
	POZ	.01	.01	.647	[-.01, .04]
	OZ	.02	.01	.254	[-.00, .04]
CPZ	FZ	-.01	.01	.99	[-.03, .02]
	FCZ no ICPS	-.02	.01	.273	[-.04, .01]
	FCZ ICPS	-.04	.01	<.001	[-.06, -.02]
	F6 ICPS	.00	.01	1	[-.02, .02]
	CZ	-.02	.01	.442	[-.04, .01]
	PZ	-.01	.01	.923	[-.03, .01]
	POZ	.00	.01	1	[-.03, .02]
	OZ	.00	.01	1	[-.02, .03]
PZ	FZ	.00	.01	1	[-.02, .03]
	FCZ no ICPS	-.01	.01	.975	[-.03, .01]
	FCZ ICPS	-.03	.01	<.001	[-.05, -.01]
	F6 ICPS	.01	.01	.967	[-.01, .03]
	CZ	-.01	.01	.996	[-.03, .02]
	CPZ	.01	.01	.923	[-.01, .03]
	POZ	.01	.01	.983	[-.02, .03]
	OZ	.01	.01	.783	[-.01, .04]
POZ	FZ	.00	.01	.999	[-.03, .02]
	FCZ no ICPS	-.02	.01	.455	[-.04, .01]
	FCZ ICPS	-.04	.01	<.001	[-.06, -.02]
	F6 ICPS	.00	.01	1	[-.02, .02]
	CZ	-.01	.01	.647	[-.04, .01]
	CPZ	.00	.01	1	[-.02, .03]
	PZ	-.01	.01	.983	[-.03, .02]
	OZ	.00	.01	1	[-.02, .03]
OZ	FZ	-.01	.01	.944	[-.03, .01]
	FCZ no ICPS	-.02	.01	.137	[-.04, .00]
	FCZ ICPS	-.04	.01	<.001	[-.07, -.02]
	F6 ICPS	.00	.01	.999	[-.03, .02]
	CZ	-.02	.01	.254	[-.04, .00]
	CPZ	.00	.01	1	[-.03, .02]

	PZ	-.01	.01	.783	[-.04, .01]
	POZ	.00	.01	1	[-.03, .02]

p-factor: Condition

Table D9. Meta-analysis general linear models: Condition post-hoc results predicting p-factor.

(I) Condition	**(J) Condition**	**Mean Difference (I-J)**	**Std. Error**	**p-value**	**CI**
Loss	Gain	.00	.00	1	[-.01, .01]
	Nogo	.03	.00	<.001	[.02, .04]
	Go	.02	.00	<.001	[.01, .03]
Gain	Loss	.00	.00	1	[-.01, .01]
	Nogo	.03	.00	<.001	[.02, .04]
	Go	.02	.00	<.001	[.01, .03]
Nogo	Loss	-.03	.00	<.001	[-.04, -.02]
	Gain	-.03	.00	<.001	[-.04, -.02]
	Go	-.01	.00	.149	[-.02, .00]
Go	Loss	-.02	.00	<.001	[-.03, -.01]
	Gain	-.02	.00	<.001	[-.03, -.01]
	Nogo	.01	.00	.149	[-.00, .02]

Electrode paired samples t-tests

Table D10. Meta-analysis paired samples t-test: Comparison between psychopathology measures for FZ electrode data.

	FZ Group 1		FZ Group 2		t(161)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.01	.08	-.01	.11	2.61	.01	.21
INT vs p-factor	.01	.08	.00	.09	2.35	.02	.19
EXT vs p-factor	-.01	.11	.00	.09	-2.59	.01	-.20

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with FZ Group 1 and FZ Group 2 column headers.

Table D11. Meta-analysis paired samples t-test: Comparison between psychopathology measures for FCZ electrode data.

	FCZ Group 1		FCZ Group 2		t(161)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.04	.09	-.01	.10	5.46	<.001	.43
INT vs p-factor	.04	.09	.02	.09	4.41	<.001	.35
EXT vs p-factor	-.01	.10	.02	.09	-6.00	<.001	-.47

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with FCZ Group 1 and FCZ Group 2 column headers.

Table D12. Meta-analysis paired samples t-test: Comparison between psychopathology measures for CZ electrode data.

	CZ Group 1		CZ Group 2		t(161)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.04	.09	-.01	.10	5.58	<.001	.44
INT vs p-factor	.04	.09	.02	.10	4.32	<.001	.34
EXT vs p-factor	-.01	.10	.02	.10	-6.10	<.001	-.48

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with CZ Group 1 and CZ Group 2 column headers.

Table D13. Meta-analysis paired samples t-test: Comparison between psychopathology measures for CPZ electrode data.

	CPZ Group 1		CPZ Group 2		t(161)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.03	.09	-.01	.10	4.92	<.001	.39
INT vs p-factor	.03	.09	.01	.09	4.14	<.001	.33
EXT vs p-factor	-.01	.10	.01	.09	-5.08	<.001	-.40

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with CPZ Group 1 and CPZ Group 2 column headers.

Table D14. Meta-analysis paired samples t-test: Comparison between psychopathology measures for PZ electrode data.

	PZ Group 1		PZ Group 2		t(161)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.03	.09	-.01	.09	4.70	<.001	.37
INT vs p-factor	.03	.09	.01	.09	3.81	<.001	.30
EXT vs p-factor	-.01	.09	.01	.09	-4.97	<.001	-.39

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with PZ Group 1 and PZ Group 2 column headers.

Table D15. Meta-analysis paired samples t-test: Comparison between psychopathology measures for POZ electrode data.

	POZ Group 1		POZ Group 2		t(161)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.02	.07	.01	.08	1.73	.09	.14
INT vs p-factor	.02	.07	.02	.08	.77	.44	.06
EXT vs p-factor	.01	.08	.02	.08	-2.58	.01	-.20

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with POZ Group 1 and POZ Group 2 column headers.

Table D16. Meta-analysis paired samples t-test: Comparison between psychopathology measures for OZ electrode data.

	OZ Group 1		OZ Group 2		t(161)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.01	.06	.01	.08	-.65	.52	-.05
INT vs p-factor	.01	.06	.01	.06	-1.00	.32	-.08
EXT vs p-factor	.01	.08	.01	.06	.21	.83	.02

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with OZ Group 1 and OZ Group 2 column headers.

Table D17. Meta-analysis paired samples t-test: Comparison between psychopathology measures for FCZ seed ICPS electrode data.

	FCZ ICPS Group 1		FCZ ICPS Group 2		t(239)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.06	.08	.01	.06	8.28	<.001	.53
INT vs p-factor	.06	.08	.04	.07	5.31	<.001	.34
EXT vs p-factor	.01	.06	.04	.07	-10.09	<.001	-.65

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with FCZ ICPS Group 1 and FCZ ICPS Group 2 column headers.

Table D18. Meta-analysis paired samples t-test: Comparison between psychopathology measures for F6 seed ICPS electrode data.

	F6 ICPS Group 1		F6 ICPS Group 2		t(239)	p	Cohen's d
	Mean	SD	Mean	SD			
INT vs EXT	.03	.06	-.01	.07	7.65	<.001	.49
INT vs p-factor	.03	.06	.01	.06	6.90	<.001	.45
EXT vs p-factor	-.01	.07	.01	.06	-7.76	<.001	-.50

Note. Variables are listed as Group 1 vs Group 2 in the first column and are in line with F6 ICPS Group 1 and F6 ICPS Group 2 column headers.

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