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Normal Congruency Sequence Effects in Psychopathology:

A Behavioral and Electrophysiological Examination Using a Confound-Minimized Design

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Abstract

Clinical studies of adaptive control emphasize the role disruptions in control play in psychopathology. However, many studies used confound-laden designs and examined only one type of psychopathology. Recent studies of event-related potentials (ERPs) suggest that robust congruency sequence effects (CSEs) – a popular index of adaptive control – appear in confound-minimized designs. Thus, the present study sought to determine whether a confound-minimized CSE paradigm could identify adaptive control dysfunction in people with major depressive disorder (MDD), generalized anxiety disorder (GAD), and obsessive-compulsive disorder (OCD). We predicted that participants with MDD and GAD would show smaller ERP CSEs and that participants with OCD would show larger ERP CSEs than healthy controls. Data from 44 people with GAD, 51 people with MDD, 31 people with OCD, and 56 healthy comparison participants revealed normal CSEs as indexed by response times (RTs) and ERPs in the psychopathology groups. Moreover, psychiatric symptoms did not moderate these CSEs. Finally, we observed a strong mean-variance relationship in RT CSEs, such that participants with stronger post-recruitment of control in mean RT scores showed the most consistent post-conflict responses (i.e., the least intraindividual variability). These findings suggest that prior findings from confound-laden tasks indicating altered CSEs in psychopathology stem from processes that are unrelated to adaptive control. Future research should employ experimental designs that isolate these processes to advance our understanding of abnormal CSEs in psychopathology.

Keywords: congruency sequence effects (CSEs); event-related potentials (ERPs); psychopathology; multilevel location-scale models; psychiatric symptoms; intraindividual variability

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Adaptive control refers to the coordination of cognitive, emotional, and physiological processes associated with identifying problems or conflict in the environment and optimizing subsequent goal-directed behavior (Miller & Cohen, 2001). Adaptive control is fundamental to everyday living, and includes such processes as overriding pre-potent responses, ignoring irrelevant stimuli, and monitoring performance. Models of adaptive control emphasize these applications and try to account for the remarkable flexibility in decision-making that healthy people demonstrate in the real world, while clinical research emphasizes the role that disruptions in adaptive control play in psychopathology. However, many studies of adaptive control in psychopathology are based on confound-laden designs and analyses that are inconsistent with best practices for examining adaptive control (Braem et al., 2019). In addition, studies of adaptive control in psychopathology tend to compare a single type of pathology with healthy individuals. Few studies directly compare multiple psychopathology types, which limits the inferences and associations with pathological processes that one can make. To overcome these obstacles, we sought to determine whether a confound-free adaptive control paradigm could identify dysfunction in people with psychopathology, including comparisons across multiple common types of psychopathology.

Congruency Sequence Effects

A core behavioral phenomenon that supports the role of conflict or discrepancy detection and subsequent adjustments in adaptive control is the behavioral alterations that occur after high-conflict trials. For example, in studies using the flanker task (Eriksen & Eriksen, 1974), high-conflict or incongruent trials (e.g., <<><<<) are associated with slower response times (RTs) and higher error rates than low-conflict or congruent trials (e.g., <<<<<), and this is typically referred to as the congruency effect or the flanker effect (MacLeod, 1991). According to the conflict

monitoring theory of adaptive control, conflict leads to the recruitment of adaptive control, which serves to shift attention toward the target stimulus and improve subsequent performance (Botvinick et al., 2001b; Botvinick et al., 2004b). Consistent with this view, performance improves after incongruent trials. Specifically, increases of control are associated with faster RTs and lower error rates on incongruent trials preceded by (a) incongruent trials (iI trials) as compared to (b) congruent trials (cI trials). Researchers call the resulting reduction of the congruency effect following incongruent relative to congruent trials "conflict adaptation" (Botvinick et al., 2001a; Botvinick et al., 2004a; Carter & van Veen, 2007a), the "Gratton effect" (Gratton et al., 1992), or the "congruency sequence effect" (CSEs; for review, see Braem et al., 2019).

The conflict monitoring theory posits that CSEs are due to adjustments in control following conflict, and that the anterior cingulate cortex (ACC) is a brain region central to the detection of such conflict (Botvinick et al., 2001b; Botvinick et al., 2004b; Botvinick et al., 1999; Carter et al., 1998; Yeung et al., 2004; Yeung & Nieuwenhuis, 2009). Once conflict is detected, compensatory mechanisms adjust behavior by engaging adaptive control. The dorsolateral prefrontal cortex (dlPFC) exerts control by selectively increasing or decreasing attention to the target stimulus (Botvinick et al., 2001b; Cohen et al., 2000; Corbetta & Shulman, 2002; Kerns et al., 2004). For example, control might serve to increase attention to the target following conflict, which results in reduced conflict related activity. This coupling between ACC and dlPFC is commonly referred to as the conflict-control loop (Carter & van Veen, 2007b), but research has added to this conceptualization by implicating other brain regions, such as the ventrolateral prefrontal cortex (Egner, 2011). Much of the work to date has used fMRI to support a role for the

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ACC in conflict detection, but electrophysiological manifestations of ACC activity also show patterns that support a role for the ACC in detecting conflict (Larson et al., 2014).

Alternative explanations of CSEs during the flanker task refer to feature integration (Hommel et al., 2004; Notebaert et al., 2001; Wendt et al., 2006) and contingency learning (Schmidt & Besner, 2008; Schmidt et al., 2007; Schmidt & De Houwer, 2011). The feature integration account attributes the CSE to uncontrolled stimulus and/or response repetitions whose natures varies across different sequences of congruent and incongruent trials. The contingency learning account attributes the CSE to learning that each distractor is more often associated with the congruent response than with each possible incongruent response when each congruent stimulus appears more frequently than each incongruent stimulus. Consistent with these accounts, the CSE is often absent in tasks without feature integration or contingency learning confounds (Mordkoff, 2012; Schmidt & De Houwer, 2011).

Event-Related Brain Potentials

Scalp-recorded event-related brain potentials (ERPs) associated with conflict monitoring are sensitive to trial-by-trial adjustments in adaptive control. In studies of the flanker task, the conflict N2 is sensitive to CSEs (Clayson & Larson, 2011a, 2011b, 2012, 2013b; Forster et al., 2011; Larson et al., 2012a; Yang et al., 2019), such that the N2 is smaller for iI than for cI trials. The conflict N2 is a negative-going component that peaks approximately 250 to 350 ms after the presentation of a flanker stimulus and is generally considered to index conflict monitoring (Larson et al., 2014). In Stroop-like tasks, N450 and the conflict slow potential (conflict SP) are associated with conflict monitoring and resolution, respectively, and both ERPs are sensitive to CSEs (Larson et al., 2016b; Larson et al., 2009a, 2009b; West & Alain, 1999, 2000; West & Bailey, 2012). Similar to the N2, the N450 and conflict SP are smaller for iI than for cI trials, and

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these amplitude changes are generally interpreted as reflecting reactive adjustments in control following conflict.

Confound-Minimized Designs

Recent behavioral and electrophysiological evidence suggests that robust CSEs can be observed in the absence of feature integration processes and contingency learning. For example, studies using four-choice response tasks during which stimulus and response repetitions are not allowed generally show intact, robust CSEs (Duthoo et al., 2014; Freitas & Clark, 2015; Jimenez & Mendez, 2013, 2014; Kim & Cho, 2014; Schmidt & Weissman, 2014; Weissman et al., 2015; Weissman et al., 2014). In these four-choice response tasks, no stimuli or responses repeat in consecutive trials, which precludes feature integration confounds, and each distractor-response pairing is equally likely to occur, which precludes contingency learning confounds (see Braem et al., 2019 for a review). After removing S-R repetitions during a flanker task, N2 amplitudes showed robust CSEs (Clayson & Larson, 2011a, 2011b) and a linear reduction in amplitude in up to three consecutive incongruent trials (Clayson & Larson, 2011a). These analyses provide evidence that S-R repetitions do not fully account for the post-conflict recruitment of adaptive control. Further evidence comes from a study that employed a confound-minimized design involving a four-choice task. Here, the N450 and conflict slow potential (SP) amplitudes, which are ERPs associated with conflict detection and resolution during Stroop-like tasks (for review, see Larson et al., 2014), showed robust CSEs (Larson et al., 2016b), similar to other ERP studies using the color-word Stroop task (Larson et al., 2012a; Larson et al., 2011; Larson et al., 2009b).

CSEs in Clinical Research

Clinical research has used adaptive control studies to identify subtle cognitive difficulties in neurological and psychiatric disorders. For example, traumatic brain injuries (TBIs) appear to

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have long-term negative effects on CSEs, but these changes are only observed in ERPs, not in behavior (Larson et al., 2011; Larson et al., 2009a). Similar findings of intact behavioral CSEs but reduced ERP CSEs are observed in autism spectrum disorders (Larson et al., 2012b). Although behavioral research has yielded mixed findings on the relationship between CSEs and anxiety or depression (for review, see Dignath et al., 2020), ERP studies of CSEs indicate weak CSEs related to both anxiety (Larson et al., 2013; Osinsky et al., 2010) and depression (Clawson et al., 2013). Finally, unlike anxiety or depression, participants with obsessive-compulsive disorder (OCD) showed exaggerated CSEs indexed by conflict N2 and RTs (Riesel et al., 2017), which is consistent with other behavioral research (Liu et al., 2012; Meiran et al., 2011). A limitation of most studies, however, is the use of confound-laden tasks, wherein confounds can make it difficult to isolate the "control-driven" CSE in neurological or psychiatric illness. To fill this gap in the present study, we employ a confound-minimized task to investigate the CSE.

A second limitation is that most studies focus on comparing the CSE in a single psychopathology group to the CSE healthy controls. Investigations that include multiple psychopathology groups might shed more light on why CSEs are sometimes smaller and sometimes larger in different groups. Consistent with this possibility, distinct patterns of impairment have been observed in psychopathology with smaller CSEs in depression and anxiety (Clawson et al., 2013; Larson et al., 2013; Osinsky et al., 2010) and larger CSEs in OCD (Riesel et al., 2017). However, researchers have not made these distinctions within the same study to allow a quantitative (not just qualitative) comparison of the CSE across groups.

The source of altered CSEs in psychopathology remains unclear. In people with depression and anxiety, relatively small CSEs might be related to increased rumination about past behavior or worry about future actions due to an inability to effectively manage cognitive

control—leading to loop of negative thoughts and abnormalities regulating emotion (e.g., Etkin & Schatzberg, 2011). In OCD, more robust CSEs and error monitoring could be due to a poorly tuned control system that fails to downregulate unambiguous or non-conflicting situations—potentially leading to repetitive behaviors and attempts to resolve perceived conflicts (e.g., Endrass & Ullsperger, 2014; Riesel et al., 2017).

Although distinct patterns of CSEs appear in prior studies of anxiety and OCD, both disorders are associated with heightened worry and overestimation of threat, which might suggest a similar pattern of impairment in CSEs. Consistent with this possibility, related work shows that the error-related negativity (ERN), an index of error monitoring, is heightened in both OCD (Riesel, 2019) and anxiety (Clayson et al., 2020; Moser et al., 2016). Theoretical work suggests ERN and N2 reflect conflict monitoring, and neuroimaging studies implicate the anterior cingulate cortex (ACC) in generating both signals (for review, see Larson et al., 2014). An examination of CSE-symptom relationships across psychopathology groups, especially in the context of a confound-minimized task, may identify unique associations between psychiatric symptoms and CSEs that can guide cross-diagnostic interventions of abnormal cognitive control. Thus, studying both psychopathology groups within the same study may help to determine whether studies of the CSE that relied on traditional diagnostic categories missed key individual-differences relationships that become clear when using a trans-diagnostic approach. To fill this gap, we investigate whether CSEs vary across different psychopathology groups.

Present Study

The purpose of the present study was to determine whether ERP indices of confound-minimized (i.e., "control-driven") CSEs vary across different psychopathology groups and whether ERPs are more sensitive than RTs to disturbances related to psychopathology. Larson et

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al. (2016b) used a confound-minimized design to examine CSEs in the N450 and conflict SP. Both ERPs showed CSEs in the absence of feature integration effects or contingency learning confounds. Therefore, we focused on these ERP components in the present study. Specifically, we sought to determine whether CSEs vary across three psychopathology groups: major depression disorder (MDD), generalized anxiety disorder (GAD), and OCD. We predicted that participants with MDD and GAD would show smaller CSEs, indexed by N450 and conflict SP, than healthy controls while participants with OCD would show larger ERP CSEs than healthy controls. We had no specific predictions related to RT CSEs. We also conducted exploratory analyses to explore relationships between different CSE measures and psychiatric symptoms.

Method

Participants

Response-locked ERP data from the present sample of 182 participants were previously reported in a study of the error-related negativity (Clayson et al., 2022). The present analyses focused on CSEs for response times (RTs) and two stimulus-related ERPs: N450 and conflict SP. CSEs in RTs or ERPs were not previously reported. The Brigham Young University Institutional Review Board approved all study procedures, and participants provided written informed consent.

We recruited participants with GAD, MDD, and OCD via flyers and referrals from university and community mental health centers. An inclusion criterion of these participants was a primary self-reported diagnosis of GAD, MDD, or OCD made by a psychiatrist, psychologist, or physician in the community, and we used this selection criterion to improve the ecological validity of the clinical sample (see Clayson et al., 2022). We recruited healthy controls from undergraduate psychology courses and advertisements around campus. Exclusion criteria for all

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participants included medication changes in the last two months, neuroleptic medications, psychotic or bipolar disorder diagnosis, history of any substance use disorder of any severity, self-reported learning disabilities or attention-deficit/hyperactivity disorder, neurological disease, left-handedness, and status as a minor (age < 18 years old). We also excluded healthy controls if they self-reported any psychiatric diagnosis; demographic summary data appear in Table 1.

We primarily aimed to assess an ecologically-valid sample presenting from the community with self-reported diagnoses (see Clayson et al., 2022). Nonetheless, we further aimed to determine whether the pattern of results would be similar in participants with confirmed clinical diagnoses. Therefore, psychiatric diagnoses were confirmed using the Mini-International Neuropsychiatric Inventory (MINI; Sheehan et al., 1998), which was administered to all participants by clinical psychology students trained and supervised by a licensed clinical psychologist (MJL). The MINI was chosen because of its good concordance with the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) and with diagnoses made by physicians and psychiatrists. In addition, the MINI requires less time to administer than the SCID (Amorim, 2000; Amorim et al., 1998; de Azevedo Marques & Zuardi, 2008; Sheehan et al., 1998). Summary information for the subset of participants with MINI-confirmed diagnoses appears in Supplementary Table 1.

Clinical Ratings

Depression symptoms were measured with the Beck Depression Inventory, 2nd Edition (BDI-II; Beck, 1996), trait worry was measured using the Penn State Worry Questionnaire (PSWQ; Meyer et al., 1990), obsessive-compulsive symptoms were measured using the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989a; Goodman et al., 1989b). Coefficient alphas for each measure in the current sample were .94 (95% CI: .93, .95) for BDI-II,

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.95 (95% CI: .94, .96) for PSWQ, and .92 (95% CI: .91, .94) for Y-BOCS. Reliabilities by group are included in Supplementary Table 2, and all point estimates were above .80, except for the BDI-II in controls, which was .78.

Experimental Task

The experimental task was identical to the task used in Larson et al. (2016b). Stimuli appeared on a 17-inch computer monitor located approximately 60 cm from the participant's head. Each block began with a fixation cross (white, Arial 50-point font), which was presented for 2000 ms on a black background. Three horizontally-oriented, identical distractor words ("Left", "Right", "Up", or "Down"; white, Arial 48-point font)—one just above fixation, one at fixation, and one just below fixation—appeared at the center of the screen for 133 ms. After a 33 ms blank screen, one of four possible target words ("Left", "Right", "Up", or "Down"; white, Arial 77-point font) appeared for 133 ms at the center of the screen. After 133 ms, the target word was replaced by a fixation cross. Participants were instructed to identify the target word as quickly and accurately as possible by making a spatially compatible button press on a computer keyboard. Participants were instructed to press the "F" key with the left middle finger to indicate the target word was "Left", and "G" key with the left index finger for the target word "Right", the "J" key with the right middle finger for the target word "Up", or the "N" key with the right index finger for the target word "Down". The word "Error" replaced the fixation cross for 200 ms 1500-1700 ms after target onset, if a participant responded incorrectly or not at all within 1500 ms of target onset. The inter-trial interval (ITI) varied randomly (without replacement) between 1850 ms and 2150 ms in steps of three ms around an average duration of 2000 ms.

The distracter and target stimuli described above were combined to create four congruent distracter-target pairs (left-left, right-right, up-up, down-down) and four incongruent distracter-

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target pairs (left-right, right-left, up-down, down-up). Distracter-target pairs involving the words "Left" and/or "Right" were presented in odd trials while distracter-target pairs involving the words "Up" and/or "Down" were presented in even trials. Each congruent and each incongruent distracter-target pair was randomly presented twelve times within a block. Participants completed a 48-trial practice block and eight 96-trial test blocks.

The order of congruent and incongruent trials within each block was first-order counterbalanced. This ensured that each of the four sequential trial types that are needed to calculate the CSE (i.e., cC trials, iC trials, cI trials, and iI trials) appeared 24 times per block, except for one sequential trial type that appeared only 23 times because the first trial of each block was not preceded by a congruent or an incongruent trial. The underrepresented sequential trial type varied randomly across blocks as trial order was randomized for each block separately. We used the Psychophysics Toolbox (Brainard, 1997) for MATLAB (Natick, MA) running on a Windows PC machine for stimulus presentation and to record each participant's responses.

Electrophysiological Data Recording and Reduction

EEG was recorded from 128 passive scalp sites using a 129-channel hydrocel geodesic sensor net and Magstim Electrical Geodesics, Inc. (Magstim EGI; Eugene, OR) amplifier system (20K nominal gain, delta-sigma sinc lowpass filter with a half-power cutoff of 2,000 Hz). The sensor layout of the hydrocel net is shown in Clayson et al. (2011). EEG was online referenced to the vertex electrode, and a ground sensor was placed at location PCz. EEG was digitized continuously at 250Hz with a 24-bit analog-to-digital converter. Impedances were maintained below 50kΩ.

EEG data were subsequently filtered offline using a sixth-order (36 db/oct) infinite impulse response Butterworth filter with half-amplitude cutoffs at .1 and 30 Hz implemented in

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ERPLab v6.1.4 (Lopez-Calderon & Luck, 2014). Stimulus-locked epochs were then extracted separately for each participant using a temporal window from 300 ms prior to the presentation of the target stimulus to 1,000 ms following presentation of the target stimulus. Consistent with our previous work (Clawson et al., 2017; Clayson & Larson, 2011a, 2011b; Larson et al., 2016a; Larson et al., 2016b), eye blinks and horizontal and vertical saccadic eye movement were removed from the segmented waveforms using independent components analysis (ICA). For the ICA procedure, epoched EEG data from all channels were processed through a binary version of EEGLab's *runica* function called *binica* (Delorme & Makeig, 2004). Any ICA components that correlated at .9 or above with the scalp topography of a blink template and at .8 or above with the scalp topography of vertical and horizontal saccade templates were removed from the data. The templates that we used for artifact correction included those that were automatically generated by the ERP PCA Toolkit and those that were created by the present authors from the present dataset.

Following artifact correction, channels with more than a 100 μV step within 100 ms intervals, a voltage difference of 300 μV through the duration of the epoch, or an absolute correlation with the nearest six neighboring channels that fell below .4 were marked as a bad for the epoch. Channels that were marked bad for more than 20% of epochs were considered globally bad. Bad channels were interpolated using spherical splines (Perrin et al., 1989), but if more than 10% of channels were marked bad for an epoch, the entire epoch was rejected. EEG data were then rereferenced off-line to an average reference. The period from 300 ms to 100 ms prior to presentation of the target stimulus was used for baseline adjustment.

Stimulus-locked ERPs were then analyzed. To reduce the biasing effects of background EEG noise on ERP measurements, ERPs were scored using a time-window mean amplitude approach (Clayson et al., 2013; Luck, 2014). EEG sites for analysis were chosen *a priori* and

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used sensors and scoring windows based on the previous study from our lab that used this paradigm (Larson et al., 2016b). Regions of interest (ROIs), which are created by averaging data across multiple electrodes, were used to provide an improved signal-to-noise ratio over data from a single electrode (Baldwin et al., 2015; Clayson et al., 2021b). The N450 was extracted as the average activity over five sites (5, 6 [FCz], 12, 13, 112; for electrode configuration, see Clayson & Larson, 2013a) and quantified using a time window from 325 to 425 ms following target onset. The conflict SP was extracted as the average activity over nine sites (60, 66, 67, 71, 72, 76, 77, 84, 85) and quantified as the average activity from 550 to 700 ms following target onset.

Internal Consistency

Generalizability theory was used to calculate dependability (ϕ), which is an estimate of internal consistency (Baldwin et al., 2015; Brennan, 2001, 2003, 2010; Carbine et al., 2021; Clayson et al., 2021a; Clayson et al., 2021d; Shavelson & Webb, 1991), but participants were not excluded based on internal consistency estimates due to the emphasis on within-person variance. Generalizability theory is a multifaceted framework for estimating psychometric reliability that is well suited for studies of ERPs. Dependability estimates from generalizability theory are analogous to coefficient alpha from classical test theory (for description of generalizability theory as it relates to studies of ERPs, see Baldwin et al., 2015; Clayson et al., 2021d; Clayson & Miller, 2017a). Dependability was examined using the ERP Reliability Analysis (ERA) Toolbox v 0.5.3 (Clayson et al., 2021c; Clayson et al., 2021d; Clayson & Miller, 2017a, 2017b). The ERA Toolbox used CmdStan v 2.24.1 (Stan Development Team, 2017) to implement the analyses in Stan (Carpenter et al., 2017). To estimate variance components, the Hamiltonian Monte Carlo estimation procedures used 4 chains and 10,000 iterations each. Dependability point estimates across groups ranged from .97 to >.99 for RTs, .91 to .96 for N450, and .89 to .96 for conflict

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SP. Dependability estimates are reported for each group, and all point estimates were above .80 (see Supplementary Table 3).

Data Analysis

Multilevel models are well suited to examining single-trial ERP scores (Brush et al., 2018; Clayson & Larson, 2019; Volpert-Esmond et al., 2018; Volpert-Esmond et al., 2021). We used the location-scale generalization[1] of multilevel models to address our aims (Walters et al., 2018), which we have successfully applied in studies of ERPs (Clayson et al., 2021c; Clayson et al., 2022; for commentary on the use of these models in studies of ERPs; see Volpert-Esmond, 2022). Traditional, location-only multilevel models include fixed and random effects to estimate conditional means but hold residual variance in the model constant across fixed/random effects (i.e., homoskedasticity). Location-only models answer questions about the average participant scores (e.g., Do healthy controls show more negative amplitudes than psychopathology groups? Do incongruent trials show longer RTs than congruent trials?).

Location-scale multilevel models expand the multilevel structure into the scale (i.e., variance) part of the model, which allows for the simultaneous modeling of means (i.e., location) and within-person variances (i.e., scale). In other words, location-scale models relax the assumption of homoskedasticity (equal residual variance across all observations) by introducing a model for the scale. The multilevel location-scale model allows a multilevel structure for the residual variance, including fixed and random effects. Thus, the residual variance (i.e., scale) can be defined by fixed effects capturing the overall variance and random effects that capture the person-specific deviations from the fixed effects. Modeling the scale portion of the model

[1] A discussion of location-scale models, their application to ERPs, and code for their implementation is described in the supplementary material of Clayson et al. (2022).

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answers questions about response variability (e.g., Do healthy controls show less amplitude variability than psychopathology groups? Do incongruent-trial RTs show less response variability than congruent-trial RTs?). By simultaneously modeling mean scores (i.e., location) and within-person variances (i.e., scale), location-scale models can determine whether there are psychopathology group differences in mean scores or in within-person variation.

Although the current manuscript focuses on analyses using location-scale models, the supplementary material includes the summaries of the location-only models for comparison to other work. We fit three multilevel location-scale models using the same predictors on the location and scale portions. *Model 1* included predictors for previous- and current-trial congruency and their interaction. *Model 2* included the same predictors as *Model 1*, diagnostic group (controls, GAD, MDD, OCD), the interaction between GAD and MDD, each interaction between previous- and current-trial congruency and group. Diagnostic group was dummy coded, with psychiatrically-healthy controls as the reference level. *Model 3* used the same predictors as *Model 1* and included psychiatric symptoms (BDI-II, PSWQ, YBOCS) and each interaction. For the location and scale portions of the model, we suppressed the overall intercept and estimated current-trial congruency-specific population intercepts, random intercepts for participants, and random slopes for previous- and current-trial congruencies. *Model 2* evaluated whether psychopathology group status predicted congruency sequence effects, and *Model 3* evaluated whether psychiatric symptoms predicted congruency sequence effects.

The models were fit within *R* v4.1.1 (R Development Core Team, 2021) using the package *brms* v2.17 (Bürkner, 2017, 2018), which is a front-end wrapper for *Stan* (Stan Development Team, 2021). For models with RTs as the dependent variable, the priors for the current-trial congruency used a normal distribution with a mean of 400 and a standard deviation

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of 100. The priors for the fixed effects estimates of the RT models used a normal distribution with a mean of 0 and standard deviation of 100. For models with ERP amplitude as the dependent variable, the priors for fixed effect estimates, including the event-type intercepts, used a normal distribution with a mean of 0 and a standard deviation of 3. For all models, all variance components were estimated using a Student's *t* prior distribution with 10 degrees of freedom, a location parameter of 0, and a scale parameter of 2. An LKJ prior distribution with a shape parameter of 2 was used as the prior for the correlation between the varying intercepts and slopes effects of event type (correct, error) within persons (Lewandowski et al., 2009). A shape = 2 parameter can be considered mildly informative, putting more weight on an identity matrix. Leave-one-out cross-validation (LOO-CV) was used to compare model fit (Vehtari et al., 2016) using the *loo* package in *R* (Vehtari et al., 2022).

Simulation studies indicate that the multilevel location-scale models yield sufficient power (> .80), good coverage, and minimal-to-zero bias with as few as 10 observations per person for the following effect sizes (pseudo-R^2): around .09 for $n = 50$, around .08 for $n = 100$, and around .05 for $n = 200$ (Walters et al., 2018). The present study included data from 182 participants with an average of 652 observations per participant. Therefore, the location-scale models should yield robust parameter estimates for the present dataset.

Results

Summary demographic and psychiatric information, RTs, and ERP amplitudes are shown in Table 1. Grand average waveforms for N450 and conflict SP are shown in Figures 1 and 2, respectively. Participants were similar in age and years of education (Fs < 2.1, ps > .10). There was an unequal gender distribution across groups, $X^2(3) = 17.41$, $p < .001$ (see Table 1 for gender distributions).

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Significant group differences were observed for BDI-II, PSWQ, and Y-BOCS (Fs > 32.9, ps < .001), and significant differences were decomposed with pairwise contrasts using the Tukey honest significant difference test to adjust p values for multiple comparisons. Psychopathology groups showed higher BDI-II scores than the control group (ps < .001), and the MDD group showed higher BDI-II scores than the OCD group (p = .04). Group differences were not observed between GAD and MDD (p = .15) or between GAD and OCD (p = .87). All psychopathology groups showed higher PSWQ scores than the control group (ps < .001), but differences were not observed between the psychopathology groups (ps > .15). All psychopathology groups showed higher Y-BOCS scores than the control group (ps < .01). The OCD group showed high Y-BOCS scores than the MDD or GAD groups (ps < .001). Group differences between MDD and GAD were not observed for Y-BOCS (p = .29).

Model Selection

We first determined the best fitting models for RTs, N450, and conflict SP amplitudes. For each dependent variable, *Model 1* included only previous- and current-trial congruencies and their interaction. *Model 2* included previous- and current-trial congruencies, diagnostic group, and their interactions. *Model 3* included previous- and current-trial congruencies, psychiatric symptoms, and their interactions. Predictors were included on both location and scale portions[2] of the models.

Model 1 was the best fitting model for RTs, N450 amplitudes, and conflict SP amplitudes (see Supplementary Table 4). Therefore, the inclusion of predictors for diagnostic group or

[2] Location-scale models showed substantial improvement in fit over location-only models. Differences in expected log predictive density ($\widehat{elpd}_{loo}$) were above 4,597 in favor of location-scale models (SEs < 172; see Supplementary Table 3). Location-only models are summarized in supplementary material Tables 3-5.

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psychiatric symptoms did not substantially improve model fit, suggesting that diagnostic status and psychiatric symptoms did not moderate CSEs (see Psychopathology Effects section). *Model 1* was consequently selected for further interpretation below for each dependent variable, and model parameters are summarized in Table 2.

The same models were fit within participants who had MINI-confirmed diagnoses. The pattern of effects was the same, and *Model 1* was again the best fitting model for RTs, N450 amplitudes, and conflict SP amplitudes (see Supplementary Table 5). Therefore, the present analyses focus on data from all participants.

Because all dependent variables showed best model fit using the predictors in *Model 1*, analyses were rerun using a multivariate location-scale model framework[3], wherein all dependent variables are modelled simultaneously from a single multivariate distribution. The advantage of this approach is the estimation of (co)variance components between dependent variables, such as relationships between RT, N450, and conflict SP conflict adaptation indices.

Response Times

RTs showed the expected congruency effect and were longer for incongruent trials than congruent trials (95% credible interval [95% CrI]: 103 to 113 ms). Regarding the impact of previous-trial congruency, RTs were longer for iC trials than for cC trials (95% CrI: 18 to 22

[3] Multivariate models estimate relationships between between-person, within-person, and residual effects across outcomes. This approach contrasts against the separate analysis of each outcome, which effectively constrains relationships between outcomes to 0. See Baldwin, Imel, Braithwaite, and Atkins (2014) for a thorough discussion of multivariate multilevel models, and see Williams, Martin, Liu, and Rast (2020) for an in-depth discussion of multivariate location-scale models. For the present analyses measurements (RT, N450, conflict SP) from the same trial were analyzed using multivariate location-scale models, and an LKJ prior distribution with a shape parameter of 2 was used as the prior for the correlation between residuals. Some RT measurements were dropped in the multivariate analysis due to EEG data not being available for a given trial due to artifact rejection. Results for RTs from the multivariate and univariate analyses were similar.

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ms), and RTs were faster for iI trials than for cI trials (95% CrI: -14 to -9 ms). A CSE index was calculated using the following equation: (cI – cC) – (iI – iC) (see Clayson & Larson, 2012; Nieuwenhuis et al., 2006; van Steenbergen et al., 2010), and a robust CSE was observed for RTs (95% CrI: 29 to 36 ms)[4].

For the scale portion of the model, parameters were exponentiated to transform them from the log- to a standard deviation scale. Comparisons are expressed as ratios, rather than linear differences, because differences were first computed on the log scale—e.g., exp(incongruent – congruent) = exp(incongruent)/exp(congruent). The standard deviation for incongruent trials was larger than it was for congruent trials (95% CrI Ratio: 1.03 to 1.09), indicating that on average within-person variance was 3% to 9% larger for incongruent trials than for congruent trials. Differences were not observed between iC and cC trials (95% CrI Ratio: 0.98 to 1.02) or between iI and cI trials (95% CrI Ratio: 0.97 to 1.01), and CSEs were not observed (95% CrI: -.002, 0.06; see footnote 4). Therefore, only a congruency effect was observed on the scale portion of the RT model.

The use of location-scale models permits the examination of relationships between means and variances because random effects parameters are jointly estimated from a multivariate normal distribution. Therefore, we examined whether CSEs on the location portion of the model were related to CSEs on the scale portion. On average, larger CSEs on the location portion of the model were strongly related to larger CSEs on the scale portion of the model (95% CrI *r*: .73 to .94). Similarly, larger iI and cI differences on the location portion were closely related to larger iI and cI differences on the scale portion of the model (95% CrI *r*: .53 to .83), and larger cC and iC

[4] Models were reparameterized for the calculation of CSEs (cI – cC) – (iI – iC). CSEs were estimated within the parameterized model, so they are on the log scale and not expressed in terms of ratios.

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differences on the location portion were related to larger cC and iC differences on the scale portion of the model (95% CrI *r*: .31 to .73), and a larger congruency effect[5] on the location portion was related to a larger congruency effect on the scale portion of the model (95% CrI *r*: .30 to .57). These relationships indicate that those participants who responded fastest showed greater consistency (i.e., less variability) in their RTs (see also Williams et al., 2021).

N450 Amplitude

As expected, N450 amplitudes were larger (i.e., more negative) for incongruent trials than for congruent trials (95% CrI: -.20 to -.04 μV). For previous-trial effects, N450 was smaller for iC trials than for cC trials (95% CrI: .14 to .26 μV) and for iI trials than for cI trials (95% CrI: .10 to .24 μV), but the CSE was not present (95% CrI: -.06 to .11 μV).

Examination of the scale portion of the model indicated that within-person variances were similar for congruent and incongruent trials (95% CrI Ratio: 0.98 to 1.01). Differences in within-person variances were not observed between iC and cC trials (95% CrI Ratio: 0.99 to 1.02) or between iI and cI trials (95% CrI Ratio: 0.99 to 1.02), and a CSE was similarly not observed for N450 (95% CrI: -.02 to .02).

CSEs on the location portion of the N450 model were unrelated to CSEs on the scale portion of the model (95% CrI *r*: -.51 to .58). Similarly, cI and iI differences and cC and iC differences on the location portion were not related to cC and iC differences on the scale portion of the model (cI/iI 95% CrI *r*: -.35 to .30; cC/iC 95% CrI *r*: -.33 to .38), and the congruency effect was not related across the location and scale portions of the model (95% CrI *r*: .-.17 to .32).

[5] A comparison of mean-variance relationships was not originally planned, but they are included due to their relevance for CSEs. Models were reparamaterized and rerun to report the congruency effects and CSEs for mean-variance relationships.

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Conflict SP Amplitude

Incongruent trials were associated with larger conflict SP amplitudes than congruent trials (95% CrI: 0.45 to 0.72 μV). Although conflict SP did not differ between iC and cC trials (95% CrI: -.12 to 0.04 μV), conflict SP did show larger cI amplitude than iI amplitude (95% CrI: -.50 to -.33 μV) and robust CSEs (95% CrI: 0.27 to 0.51).

Conflict SP amplitudes on the scale portion of the model showed a similar lack of effects as N450 amplitudes. Within-person variances for conflict SP were similar across congruent and incongruent trials (95% CrI Ratio: 0.98 to 1.00). Differences in within-person variances were not observed between iC and cC trials (95% CrI Ratio: 0.98 to 1.01) or between iI and cI trials (95% CrI Ratio: 0.99 to 1.02) or for CSEs (95% CrI: -.03 to .02).

Regarding mean-variance relationships, conflict SP CSEs (95% CrI *r*: -.20 to .61), iI and cI differences (95% CrI *r*: -.21 to .46), and cC and iC differences (95% CrI *r*: -.29 to .36) were not related across location and scale portions of the model. The congruency effect was also not related across the location and scale portions (95% CrI *r*: -.01 to .36).

RT-ERP Relationships

A motivation for using multivariate models is to allow an examination of potential relationships among location and scale portions the RT, N450, and conflict SP models. Correlations for congruency sequence effects among means and variances of each measurement appear in Tables 5 and 6. Only RT parameters showed mean-variance relationships, and these relationships were reported above (see Figure 3).

Psychopathology Effects

A primary motivation of the current study was to examine psychopathology group differences in CSEs. Although Models 2 and 3 did not improve the predictive accuracy over

Model 1, we examined the interaction effects between CSE and diagnostic group and between CSE and psychiatric symptoms to assess whether CSEs differed across any particular diagnostic group or psychiatric symptom dimension. Neither diagnostic group nor psychiatric symptom dimension moderated any of the CSEs for RTs, N450, or conflict SP (see Table 7).

Discussion

The aim of the present study was to determine whether altered ERP indices of confound-minimized (i.e., "control-driven") CSEs are associated with psychopathology. We replicated the expected RT and conflict SP CSEs (Larson et al., 2016b), and extended these findings by showing a strong mean-variance relationship in RT CSEs. Contrary to our initial expectations, however, RT, N450, and conflict SP CSEs were similar in magnitude across healthy controls and the three psychopathology groups. Moreover, CSEs were not moderated by traditional diagnostic categories or clinical symptom status examined in a trans-diagnostic fashion. These findings suggest that altered CSEs in previous studies of psychopathology may index feature integration or contingency learning confounds rather than differences in the recruitment of adaptive control.

In confound-laden designs, learning and memory accounts suggest that CSEs index feature integration (Hommel et al., 2004; Notebaert et al., 2001; Wendt et al., 2006) and/or contingency learning confounds (Schmidt & Besner, 2008; Schmidt et al., 2007; Schmidt & De Houwer, 2011). Thus, group differences in such designs may indicate that participants with GAD, MDD, or OCD show abnormalities related to either or both confounds. Interestingly, the processes underlying these confounds are common targets of cognitive bias modification for people with psychopathology (for review, see Martinelli et al., 2022), an intervention that targets maladaptive information processing in a range of psychiatric conditions including depression (Yang et al., 2015), anxiety (Amir et al., 2009), and OCD (Williams & Grisham, 2013).

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Cognitive bias modification facilitates processing stimuli and learning contingencies in a fashion that circumvents or opposes maladaptive processing styles (MacLeod & Clarke, 2015; MacLeod & Mathews, 2012). The present findings indicating intact "control-driven" CSEs in MDD, GAD, and OCD fit with the cognitive bias modification approach in suggesting that some deficits in psychopathology index processes unrelated to adaptive control.

Another contribution of the present study is that we independently replicated findings of conflict SP CSEs using a confound-minimized design (Larson et al., 2016b). Although Larson et al. (2016b) observed CSEs for N450 amplitudes, the present study failed to replicate this particular finding. It is possible that N450 is not as sensitive to CSEs as conflict SP, as findings from Stroop tasks typically support stronger trial-by-trial effects for conflict SP than for N450 (e.g., Larson et al., 2009a; Larson et al., 2009b). The present study used about three times more participants than the Larson et al. study (182 healthy and psychiatric participants compared to 59 healthy participants), and it is possible that the original finding was due to sampling error. Nonetheless, the failure to observe CSEs in N450 can be considered in future studies of ERP CSEs using confound-minimized designs to determine whether N450 is robustly sensitive to trial-by-trial adjustments in control.

We extended the findings of Larson et al. (2016b) by examining intra-individual variability in RT and ERP responses and mean-variance relationships. For RTs, we observed a congruency effect for means and intra-individual variability, such that incongruent trials were associated with longer RTs and greater within-person variability of single-trial responses than congruent trials. Additionally, the mean-variance analysis indicated that a stronger congruency effect for mean scores was related to a stronger congruency effect for within-person variance. This outcome conceptually replicates findings from the flanker and Stroop tasks (Williams et al.,

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2021). Put simply, participants who are better at inhibiting task-irrelevant information (i.e., flankers) also respond more consistently. This pattern of effects extended to CSEs: larger cI/iI differences in mean scores related to larger cI/iI differences in within-person variability. This pattern indicates that stronger post-conflict recruitment of control in mean RT scores relates to more consistent post-conflict responses. To the best of our knowledge, the present results represent the first examination of mean-variance relationships in CSEs. We further note that, although within-person variability in RTs was related to CSEs, these relationships were not observed for N450 or conflict SP.

An examination of RT-ERP relationships did not yield strong effects, which is not consistent with research using confound-laden designs. The only prior study using the present paradigm did not examine RT-ERP relationships (Larson et al., 2016b). RT-ERP correlations have been examined for conflict N2 (Clayson & Larson, 2011a; Larson et al., 2013; Larson et al., 2016a) and for N450 and conflict SP (Larson et al., 2009b), but findings are mixed. For example, larger N2 for cI (Clayson & Larson, 2011b) or iI trials (Clayson & Larson, 2011a; Larson et al., 2016a) sometimes relates to faster RTs, but this is not always the case (Larson et al., 2013). This inconsistent pattern of effects might be expected. While the N450 closely relates to conflict detection and the conflict SP relates to conflict resolution, RT reflects numerous cognitive, affective, and motor processes. Thus, relationships between RT and different ERP indices of CSEs likely reflect at least partially distinct processes, leading to an inconsistent pattern of RT-ERP relationships across studies. Furthermore, the present study analyzed single-trial measurements and showed that RTs and ERPs were not correlated within people. Another possible explanation of the inconsistent RT-ERP correlations across studies is the use of solely between-subjects analyses in other research (e.g., correlations of subject averages across

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participants), which might misrepresent within-subject relationships like those assessed in the present study.

The present study had some limitations. First, the focus was on an ecologically valid sample of participants with diagnoses made by community practitioners, which could have led us to misclassify some participants. However, study findings were consistent when only examining participants with MINI-confirmed diagnoses. Additionally, dimensional analyses between psychiatric symptoms and CSEs did not yield strong effects, indicating that the clinical symptom status of the participants with psychopathology did not contribute to the lack of effects. Second, many participants with clinical diagnoses were receiving medication which might have normalized CSEs. Studies of OCD (Riesel et al., 2017) and MDD (Clawson et al., 2013), however, have not shown that medication status alters CSEs. Therefore, the use of psychoactive medications is unlikely to account for the similarity in ERP CSEs across psychopathology and healthy control groups. Third, the sample sizes of each group (*n*s: 31 to 56) were large relative to the average ERP group sample size of 21 (see Clayson et al., 2019), but still modest and might not have been sufficiently powered to detect group differences. Nonetheless, psychiatric symptoms examined trans-diagnostically in the full sample ($n = 182$) did not yield relationships with CSEs. Despite these limitations, this study used a unique technique to model intra-individual variability and multiple psychopathology groups for comparison and assessment of psychiatric symptoms.

In conclusion, we sought to determine whether participants with GAD, MDD, and OCD show altered CSEs as indexed by ERPs and RTs using a confound-minimized paradigm. The results indicated normal CSEs in the psychopathology groups and suggest that prior findings of altered CSEs in these groups might be due to the use of confound-laden designs and analyses.

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Therefore, future research interested in the specific elements of CSEs that might be normal or impaired in psychopathology groups should consider using different confound-laden and confound-free designs to understand the mechanisms associated with abnormal CSEs in psychopathology. Future studies using different confound-laden and confound-free paradigms within the same participants would further serve to evaluate the psychometric characteristics of these paradigms and help clarify the processes involved in trial-by-trial changes in behavior and ERPs.

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References

Amir, N., Beard, C., Burns, M., & Bomyea, J. (2009). Attention modification program in individuals with generalized anxiety disorder. *Journal of Abnormal Psychology*, *118*, 28-33. https://doi.org/10.1037/a0012589

Amorim, P. (2000). Mini International Neuropsychiatric Interview (MINI): Validation of a short structured diagnostic psychiatric interview. *Revista Brasileira de Psiquiatria*, *22*, 106-115.

Amorim, P., Lecrubier, Y., Weiller, E., Hergueta, T., & Sheehan, D. V. (1998). DSM-III-R Psychotic Disorders: Procedural validity of the Mini International Neuropsychiatric Interview (MINI): Concordance and causes of discordance with the CIDI. *European Psychiatry*, *13*, 26-34. https://doi.org/10.1016/S0924-9338(97)86748-X

Baldwin, S. A., Imel, Z. E., Braithwaite, S. R., & Atkins, D. C. (2014). Analyzing multiple outcomes in clinical research using multivariate multilevel models. *Journal of Consulting and Clinical Psychology*, *82*(5), 920-930. https://doi.org/10.1037/a0035628 (Advances in Data Analytic Methods)

Baldwin, S. A., Larson, M. J., & Clayson, P. E. (2015). The dependability of electrophysiological measurements of performance monitoring in a clinical sample: A generalizability and decision analysis of the ERN and Pe. *Psychophysiology*, *52*(6), 790-800. https://doi.org/10.1111/psyp.12401

Beck, A. T. (1996). *Beck Depression Inventory - Second Edition (BDI-II)*. The Psychological Corporation.

Botvinick, M., Carter, C. S., Braver, T. S., Barch, D. M., & Cohen, J. D. (2001a). Conflict monitoring and cognitive control. *Psychological Review*, *108*, 624-652. https://doi.org/10.1037/0033-295X.108.3.624

Botvinick, M., Cohen, J. D., & Carter, C. S. (2004a). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences*, *8*(12), 539-546. https://doi.org/10.1016/j.tics.2004.10.003

Botvinick, M. W., Carter, C. S., Braver, T. S., Barch, D. M., & Cohen, J. D. (2001b). Conflict monitoring and cognitive control. *Psychological Review*, *108*, 624-652. https://doi.org/10.1037/0033-295X.108.3.624

Botvinick, M. W., Cohen, J. D., & Carter, C. S. (2004b). Conflict monitoring and anterior cingulate cortex: An update. *Trends in Cognitive Sciences*, *8*(12), 539-546. https://doi.org/10.1016/j.tics.2004.10.003

Botvinick, M. W., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict monitoring versus selection-for-action in anterior cingulate cortex. *Nature*, *402*, 179-181. https://doi.org/10.1038/46035

Braem, S., Bugg, J. M., Schmidt, J. R., Crump, M. J. C., Weissman, D. H., Notebaert, W., & Cohen, M. X. (2019). Measuring adaptive control in conflict tasks [Review]. *Trends in Cognitive Sciences*, *23*(9), 769-783. https://doi.org/10.1016/j.tics.2019.07.002

Brainard, D. H. (1997). The Psychophysics toolbox. *Spatial Vision*, *10*, 433-436. https://doi.org/10.1163/156856897x00357

Brennan, R. L. (2001). *Generalizability theory: Statistics for social science and public policy*. Springer-Verlag.

Brennan, R. L. (2003). Coefficients and indices in generalizability theory. In *CASMA Research Report No. 1* (pp. 1-48). Center for Advanced Studies in Measurement and Assessment.

Author Manuscript

https://www.education.uiowa.edu/docs/default-source/casma---research/01casmareport.pdf?sfvrsn=2

Brennan, R. L. (2010). Generalizability theory and classical test theory. *Applied Measurement in Education*, *24*(1), 1-21. https://doi.org/10.1080/08957347.2011.532417

Brush, C. J., Ehmann, P. J., Hajcak, G., Selby, E. A., & Alderman, B. L. (2018). Using multilevel modeling to examine blunted neural responses to reward in major depression. *Biol Psychiatry Cogn Neurosci Neuroimaging*, *3*(12), 1032-1039. https://doi.org/10.1016/j.bpsc.2018.04.003

Bürkner, P.-C. (2017). brms: An R package for Bayesian multilevel models using Stan. *Journal of Statistical Software*, *80*. https://doi.org/10.18637/jss.v080.i01

Bürkner, P.-C. (2018). Advanced Bayesian multilevel modeling with the R Package brms. *R Journal*, *10*(1), 395-411. https://doi.org/10.32614/RJ-2018-017

Carbine, K. A., Clayson, P. E., Baldwin, S. A., LeCheminant, J., & Larson, M. J. (2021). Using generalizability theory and the ERP Reliability Analysis (ERA) Toolbox for assessing test-retest reliability of ERP scores Part 2: Application to food-based tasks and stimuli. *International Journal of Psychophysiology*, *166*, 188-198. https://doi.org/10.31234/osf.io/zuc8g

Carpenter, B., Gelman, A., Hoffman, M., Lee, D., Goodrich, B., Betancourt, M., Brubaker, M. A., Guo, J., Li, P., & Riddell, A. (2017). Stan: A probabilistic programming language. *Journal of Statistical Software*, *76*(1), 1-32. https://doi.org/https://doi.org/10.18637/jss.v076.i01

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M., Ross, L. L., Stenger, V. A., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, *280*, 747-749. DOI: 10.1126/science.280.5364.747

Carter, C. S., & van Veen, V. (2007a). Anterior cingulate cortex and conflict detection: An update of theory and data. *Cognitive Affective and Behavioral Neuroscience*, *7*, 367-379. https://doi.org/10.3758/CABN.7.4.367

Carter, C. S., & van Veen, V. (2007b). Anterior cingulate cortex and conflict detection: An update of theory and data. *Cogn. Affect. Behav. Neurosci.*, *7*, 367-379. https://doi.org/10.3758/CABN.7.4.367

Clawson, A., Clayson, P. E., Keith, C. M., Catron, C., & Larson, M. J. (2017). Conflict and performance monitoring throughout the lifespan: An event-related potential (ERP) and temporospatial component analysis. *Biological Psychology*, *124*, 87-99. https://doi.org/10.1016/j.biopsycho.2017.01.012

Clawson, A., Clayson, P. E., & Larson, M. J. (2013). Cognitive control adjustments and conflict adaptation in major depressive disorder. *Psychophysiology*, *50*, 711-721. https://doi.org/10.1111/psyp.12066

Clayson, P. E., Baldwin, S. A., & Larson, M. J. (2013). How does noise affect amplitude and latency measurement of event-related potentials (ERPs)? A methodological critique and simulation study. *Psychophysiology*, *50*, 174-186. https://doi.org/10.1111/psyp.12001

Clayson, P. E., Baldwin, S. A., & Larson, M. J. (2021a). Evaluating the internal consistency of subtraction-based and residualized difference scores: Considerations for psychometric reliability analyses of event-related potentials. *Psychophysiology*, *58*(4), e13762. https://doi.org/10.1111/psyp.13762

Clayson, P. E., Baldwin, S. A., Rocha, H. A., & Larson, M. J. (2021b). The data-processing multiverse of event-related potentials (ERPs): A roadmap for the optimization and

Author Manuscript

standardization of ERP processing and reduction pipelines. *NeuroImage*, *245*, 118712. https://doi.org/10.1016/j.neuroimage.2021.118712

Clayson, P. E., Brush, C. J., & Hajcak, G. (2021c). Data quality and reliability metrics for event-related potentials (ERPs): The utility of subject-level reliability. *International Journal of Psychophysiology*, *165*, 121-136. https://doi.org/10.1016/j.ijpsycho.2021.04.004

Clayson, P. E., Carbine, K. A., Baldwin, S. A., & Larson, M. J. (2019). Methodological reporting behavior, sample sizes, and statistical power in studies of event-related potentials: Barriers to reproducibility and replicability. *Psychophysiology*, *111*(6), 5-17. https://doi.org/https://doi.org/10.1111/psyp.13437

Clayson, P. E., Carbine, K. A., Baldwin, S. A., Olsen, J. A., & Larson, M. J. (2021d). Using generalizability theory and the ERP Reliability Analysis (ERA) Toolbox for assessing test-retest reliability of ERP scores Part 1: Algorithms, framework, and implementation. *International Journal of Psychophysiology*, *166*, 174-187. https://doi.org/10.1016/j.ijpsycho.2021.01.006

Clayson, P. E., Carbine, K. A., & Larson, M. J. (2020). A registered report of error-related negativity and reward positivity as biomarkers of depression: P-curving the evidence. *International Journal of Psychophysiology*, *150*, 50-72. https://doi.org/10.1016/j.ijpsycho.2020.01.005

Clayson, P. E., Clawson, A., & Larson, M. J. (2011). Sex differences in electrophysiological indices of conflict monitoring. *Biological Psychology*, *87*(2), 282-289. https://doi.org/10.1016/j.biopsycho.2011.03.011

Clayson, P. E., & Larson, M. J. (2011a). Conflict adaptation and sequential trial effects: Support for the conflict monitoring theory. *Neuropsychologia*, *49*, 1953-1961. https://doi.org/10.1016/j.neuropsychologia.2011.03.023

Clayson, P. E., & Larson, M. J. (2011b). Effects of repetition priming on neurophysiological and behavioral indices of conflict adaptation and cognitive control. *Psychophysiology*, *48*(12), 1621-1630. https://doi.org/10.1111/j.1469-8986.2011.01265.x

Clayson, P. E., & Larson, M. J. (2012). Cognitive performance and electrophysiological indices of cognitive control: A validation study of conflict adaptation. *Psychophysiology*, *49*(5), 627-637. https://doi.org/10.1111/j.1469-8986.2011.01345.x

Clayson, P. E., & Larson, M. J. (2013a). Adaptation to emotional conflict: Evidence from a novel face emotion paradigm. *PLoS ONE*, *8*(9), e75776. https://doi.org/10.1371/journal.pone.0075776

Clayson, P. E., & Larson, M. J. (2013b). Psychometric properties of conflict monitoring and conflict adaptation indices: Response time and conflict N2 event-related potentials. *Psychophysiology*, *50*(12), 1209-1219. https://doi.org/10.1111/psyp.12138

Clayson, P. E., & Larson, M. J. (2019). The impact of recent and concurrent affective context on cognitive control: An ERP study of performance monitoring. *International Journal of Psychophysiology*, *143*, 44-56. https://doi.org/10.1016/j.ijpsycho.2019.06.007

Clayson, P. E., & Miller, G. A. (2017a). ERP Reliability Analysis (ERA) Toolbox: An open-source toolbox for analyzing the reliability of event-related potentials. *International Journal of Psychophysiology*, *111*, 68-79. https://doi.org/10.1016/j.ijpsycho.2016.10.012

Clayson, P. E., & Miller, G. A. (2017b). Psychometric considerations in the measurement of event-related brain potentials: Guidelines for measurement and reporting. *International Journal of Psychophysiology*, *111*, 57-67. https://doi.org/10.1016/j.ijpsycho.2016.09.005

Author Manuscript

Clayson, P. E., Rocha, H. A., Baldwin, S. A., Rast, P., & Larson, M. J. (2022). Understanding the error in psychopathology: Notable intraindividual differences in neural variability of performance monitoring. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *7*(6), 555-565. https://doi.org/10.1016/j.bpsc.2021.10.016

Cohen, J. D., Botvinick, M., & Carter, C. S. (2000). Anterior cingulate and prefrontal cortex: Who's in control? [10.1038/74783]. *Nature Neuroscience*, *3*(5), 421-423. http://dx.doi.org/10.1038/74783

Corbetta, M., & Shulman, G. L. (2002). Control of goal-directed and stimulus-driven attention in the brain [Article]. *Nature Reviews Neuroscience*, *3*(3), 215-229. https://doi.org/10.1038/nrn755

de Azevedo Marques, J. M., & Zuardi, A. W. (2008). Validity and applicability of the Mini International Neuropsychiatric Interview administered by family medicine residents in primary health care in Brazil [Comparative Study]. *General Hospital Psychiatry*, *30*(4), 303-310. https://doi.org/10.1016/j.genhosppsych.2008.02.001

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

Dignath, D., Eder, A. B., Steinhauser, M., & Kiesel, A. (2020). Conflict monitoring and the affective-signaling hypothesis—An integrative review. *Psychonomic Bulletin & Review*, *27*(2), 193-216. https://doi.org/10.3758/s13423-019-01668-9

Duthoo, W., Abrahamse, E. L., Braem, S., Boehler, C. N., & Notebaert, W. (2014). The congruency sequence effect 3.0: A critical test of conflict adaptation. *PLoS ONE*, *9*(10), e110462. https://doi.org/10.1371/journal.pone.0110462

Egner, T. (2011). Right ventrolateral prefrontal cortex mediates individual differences in conflict-driven cognitive control. *Journal of Cognitive Neuroscience*, *23*(12), 3903-3913. https://doi.org/10.1162/jocn_a_00064

Endrass, T., & Ullsperger, M. (2014). Specificity of performance monitoring changes in obsessive-compulsive disorder. *Neuroscience & Biobehavioral Reviews*, *46*, 124-138. https://doi.org/https://doi.org/10.1016/j.neubiorev.2014.03.024

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a non-search task. *Perception & Psychophysics*, *16*, 143-149. https://doi.org/10.3758/BF03203267

Etkin, A., & Schatzberg, A. F. (2011). Common abnormalities and disorder-specific compensation during implicit regulation of emotional processing in generalized anxiety and major depressive disorders. *American Journal of Psychiatry*, *168*(9), 968-978. https://doi.org/10.1176/appi.ajp.2011.10091290

Forster, S. E., Carter, C. S., Cohen, J. D., & Cho, R. Y. (2011). Parametric manipulation of the conflict signal and control-state adaptation. *Journal of Cognitive Neuroscience*, *23*(4), 923-935. https://doi.org/10.1162/jocn.2010.21458

Freitas, A. L., & Clark, S. L. (2015). Generality and specificity in cognitive control: conflict adaptation within and across selective-attention tasks but not across selective-attention and Simon tasks. *Psychological Research*, *79*, 143-162. https://doi.org/10.1007/s00426-014-0540-1

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Delgado, P., Heninger, G. R., & Charney, D. S. (1989a). The Yale-Brown Obsessive Compulsive Scale: II. Validity.

Author Manuscript

Archives of General Psychiatry, *46*(11), 1012-1016 doi:10.1001/archpsyc.1989.01810110054008

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., Heninger, G. R., & Charney, D. S. (1989b). The Yale-Brown Obsessive Compulsive Scale: I. Development, use, and reliability. *Archives of General Psychiatry*, *46*(11), 1006-1011. DOI: 10.1001/archpsyc.1989.01810110048007

Gratton, G., Coles, M. G., & Donchin, E. (1992). Optimizing the use of information: Strategic control of activation of responses. *Journal of Experimental Psychology: General*, *121*(4), 480-506. https://doi.org/10.1037/0096-3445.121.4.480

Hommel, B., Proctor, R. W., & Vu, K. P. (2004). A feature-integration account of sequential effects in the Simon task. *Psychol. Res.*, *68*, 1-17. https://doi.org/10.1007/s00426-003-0132-y

Jimenez, L., & Mendez, A. (2013). It is not what you expect: Dissociating conflict adaptation from expectancies in a Stroop task. *Journal of Experimental Psychology: Human Perception and Performance*, *39*, 271-284. https://doi.org/10.1037/a0027734

Jimenez, L., & Mendez, A. (2014). Even with time, conflict adaptation is not made of expectancies. *Frontiers in Psychology*, *5*, 1042. https://doi.org/10.3389/fpsyg.2014.01042

Kerns, J. G., Cohen, J. D., MacDonald, A. W., Cho, R. Y., Stenger, V. A., & Carter, C. S. (2004). Anterior cingulate conflict monitoring and adjustments in control. *Science*, *303*, 1023-1026. https://doi.org/10.1126/science.1089910

Kim, S., & Cho, Y. S. (2014). Congruency sequence effect without feature integration and contingency learning. *Acta Psychologica*, *149*, 60-68. https://doi.org/10.1371/journal.pone.0102337

Larson, M. J., Clawson, A., Clayson, P. E., & Baldwin, S. A. (2013). Cognitive conflict adaptation in generalized anxiety disorder. *Biological Psychology*, *94*(2), 408-418. https://doi.org/10.1016/j.biopsycho.2013.08.006

Larson, M. J., Clawson, A., Clayson, P. E., & South, M. (2012a). Cognitive control and conflict adaptation similarities in children and adults. *Developmental Neuropsychology*, *37*(4), 343-357. https://doi.org/10.1080/87565641.2011.650337

Larson, M. J., Clayson, P. E., & Clawson, A. (2014). Making sense of all the conflict: A theoretical review and critique of conflict-related ERPs [Review]. *International Journal of Psychophysiology*, *93*(3), 283-297. https://doi.org/10.1016/j.ijpsycho.2014.06.007

Larson, M. J., Clayson, P. E., Keith, C. M., Hunt, I. J., Hedges, D. W., Nielsen, B. L., & Call, V. R. A. (2016a). Cognitive control adjustments in healthy older and younger adults: Conflict adaptation, the error-related negativity (ERN), and evidence of generalized decline with age. *Biological Psychology*, *115*, 50-63. https://doi.org/10.1016/j.biopsycho.2016.01.008

Larson, M. J., Clayson, P. E., Kirwan, C. B., & Weissman, D. H. (2016b). Event-related potential indices of congruency sequence effects without feature integration or contingency learning confounds. *Psychophysiology*, *53*(6), 814-822. https://doi.org/10.1111/psyp.12625

Larson, M. J., Farrer, T. J., & Clayson, P. E. (2011). Cognitive control in mild traumatic brain injury: Conflict monitoring and conflict adaptation. *International Journal of Psychophysiology*, *82*(1), 69-78. https://doi.org/10.1016/j.ijpsycho.2011.02.018

Larson, M. J., Kaufman, D. A., & Perlstein, W. M. (2009a). Conflict adaptation and cognitive control adjustments following traumatic brain injury. *Journal of the International*

Author Manuscript

Neuropsychological Society, *15*(6), 927-937. https://doi.org/10.1017/S1355617709990701

Larson, M. J., Kaufman, D. A., & Perlstein, W. M. (2009b). Neural time course of conflict adaptation effects on the Stroop task. *Neuropsychologia*, *47*(3), 663-670. https://doi.org/10.1016/j.neuropsychologia.2008.11.013

Larson, M. J., South, M., Clayson, P. E., & Clawson, A. (2012b). Cognitive control and conflict adaptation in youth with high-functioning autism. *Journal of Child Psychology and Psychiatry*, *53*(4), 440-448. https://doi.org/10.1111/j.1469-7610.2011.02498.x

Lewandowski, D., Kurowicka, D., & Joe, H. (2009). Generating random correlation matrices based on vines and extended onion method. *Journal of Multivariate Analysis*, *100*(9), 1989-2001. https://doi.org/10.1016/j.jmva.2009.04.008

Liu, Y., Gehring, W., Weissman, D., Taylor, S., & Fitzgerald, K. (2012). Trial-by-trial adjustments of cognitive control following errors and response conflict are altered in pediatric obsessive compulsive disorder [Original Research]. *Frontiers in Psychiatry*, *3*. https://doi.org/10.3389/fpsyt.2012.00041

Lopez-Calderon, J., & Luck, S. J. (2014). ERPLAB: An open-source toolbox for the analysis of event-related potentials. *Frontiers In Human Neuroscience*, *8*(e3004), 213. https://doi.org/10.3389/fnhum.2014.00213

Luck, S. J. (2014). *An introduction to the event-related potential technique* (2nd ed.). The MIT Press.

MacLeod, C., & Clarke, P. J. F. (2015). The Attentional Bias Modification approach to anxiety intervention. *Clinical psychological science*, *3*(1), 58-78. https://doi.org/10.1177/2167702614560749

MacLeod, C., & Mathews, A. (2012). Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology*, *8*(1), 189-217. https://doi.org/10.1146/annurev-clinpsy-032511-143052

MacLeod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review [Review]. *Psychological Bulletin*, *109*(2), 163-203. http://eutils.ncbi.nlm.nih.gov/entrez/eutils/elink.fcgi?dbfrom=pubmed&id=2034749&retmode=ref&cmd=prlinks

Martinelli, A., Grüll, J., & Baum, C. (2022). Attention and interpretation cognitive bias change: A systematic review and meta-analysis of bias modification paradigms. *Behaviour Research and Therapy*, *157*, 104180. https://doi.org/https://doi.org/10.1016/j.brat.2022.104180

Meiran, N., Diamond, G. M., Toder, D., & Nemets, B. (2011). Cognitive rigidity in unipolar depression and obsessive compulsive disorder: Examination of task switching, Stroop, working memory updating and post-conflict adaptation. *Psychiatry Research*, *185*(1), 149-156. https://doi.org/https://doi.org/10.1016/j.psychres.2010.04.044

Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, *28*(6), 487-495. https://doi.org/https://doi.org/10.1016/0005-7967(90)90135-6

Miller, E. K., & Cohen, J. D. (2001). An integrative theory of prefrontal cortex function. *Annual Review of Neuroscience*, *24*, 167-202. https://doi.org/10.1146/annurev.neuro.24.1.167

Mordkoff, J. T. (2012). Observation: Three reasons to avoid having half of the trials be congruent in a four-alternative forced-choice experiment on sequential modulation. *Psychonomic Bulletin & Review*, *19*, 750-757. https://doi.org/10.3758/s13423-012-0257-3

Author Manuscript

Moser, J. S., Moran, T. P., Kneip, C., Schroder, H. S., & Larson, M. J. (2016). Sex moderates the association between symptoms of anxiety, but not obsessive compulsive disorder, and error-monitoring brain activity: A meta-analytic review. *Psychophysiology*, *53*(1), 21-29. https://doi.org/https://doi.org/10.1111/psyp.12509

Nieuwenhuis, S., Stins, J. F., Posthuma, D., Polderman, T. J. C., Boomsma, D. I., & de Geus, E. J. (2006). Accounting for sequential trial effects in the flanker task: Conflict adaptation or associative priming? *Memory & Cognition*, *34*(6), 1260-1272. https://doi.org/10.3758/BF03193270

Notebaert, W., Soetens, E., & Melis, A. (2001). Sequential analysis of a Simon task: Evidence for an attention-shift account. *Psychol. Res.*, *65*(3), 170-184. https://doi.org/10.1007/s004260000054

Osinsky, R., Alexander, N., Gebhardt, H., & Hennig, J. (2010). Trait anxiety and dynamic adjustments in conflict processing. *Cognitive Affective and Behavioral Neuroscience*, *10*(3), 372-381. https://doi.org/10.3758/CABN.10.3.372

Perrin, F., Pernier, J., Bertrand, O., & Echallier, J. F. (1989). Spherical splines for scalp potential and current-density mapping. *Electroencephalography and clinical neurophysiology*, *72*(2), 184-187. https://doi.org/10.1016/0013-4694(89)90180-6

R Development Core Team. (2021). *R: A language and environment for statistical computing*. In *R Foundation for Statistical Computing* http://www.R-project.org/

Riesel, A. (2019). The erring brain: Error-related negativity as an endophenotype for OCD-A review and meta-analysis. *Psychophysiology*, *56*(4), e13348. https://doi.org/10.1111/psyp.13348

Riesel, A., Klawohn, J., Kathmann, N., & Endrass, T. (2017). Conflict monitoring and adaptation as reflected by N2 amplitude in obsessive–compulsive disorder. *Psychological Medicine*, *47*(8), 1379-1388. https://doi.org/10.1017/S0033291716003597

Schmidt, J. R., & Besner, D. (2008). The Stroop effect: Why proportion congruent has nothing to do with congruency and everything to do with contingency. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *34*(3), 514-523. https://doi.org/10.1037/0278-7393.34.3.514

Schmidt, J. R., Crump, M. J. C., Cheesman, J., & Besner, D. (2007). Contingency learning without awareness: Evidence for implicit control [Controlled Clinical Trial]. *Consciousness and Cognition*, *16*(2), 421-435. https://doi.org/10.1016/j.concog.2006.06.010

Schmidt, J. R., & De Houwer, J. (2011). Now you see it, now you don't: Controlling for contingencies and stimulus repetitions eliminates the Gratton effect. *Acta Psychologica*, *138*, 176-186. https://doi.org/10.1016/j.actpsy.2011.06.002

Schmidt, J. R., & Weissman, D. H. (2014). Congruency sequence effects without feature integration or contingency learning confounds. *PLoS ONE*, *9*(7), e102337. https://doi.org/10.1371/journal.pone.0102337

Shavelson, R. J., & Webb, N. M. (1991). *Generalizability theory: A primer*. SAGE Publications, Inc.

Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., Hergueta, T., Baker, R., & Dunbar, G. C. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): The development and validation of a structured diagnostic psychiatric intervew for DSM-IV adn ICD-10. *Journal of Clinical Psychiatry*, *59*, 22-33.

Author Manuscript

Stan Development Team. (2017). *CmdStan: The command-line interface to Stan, version 2.14.0*. In http://mc-stan.org

Stan Development Team. (2021). Stan Modeling Language Users Guide and Reference Manual v2.26.1. http://mc-stan.org/

van Steenbergen, H., Band, G. P. H., & Hommel, B. (2010). In the mood for adaptation: How affect regulates conflict-driven control. *Psychological Science*, *21*(11), 1629-1634. https://doi.org/10.1177/0956797610385951

Vehtari, A., Gabry, J., Magnusson, M., Yao, Y., Bürkner, P., Paananen, T., & Gelman, A. (2022). *loo: Efficient leave-one-out cross-validation and WAIC for Bayesian models*. In (Version R package version 2.5.1) https://mc-stan.org/loo

Vehtari, A., Gelman, A., & Gabry, J. (2016). Practical Bayesian model evaluation using leave-one-out cross-validation and WAIC. *Statistics and Computing*, *27*(5), 1413-1432. https://doi.org/10.1007/s11222-016-9696-4

Volpert-Esmond, H. I. (2022). Looking at change: Examining meaningful variability in psychophysiological measurements. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*, *7*(6), 530-531. https://doi.org/10.1016/j.bpsc.2022.02.006

Volpert-Esmond, H. I., Merkle, E. C., Levsen, M. P., Ito, T. A., & Bartholow, B. D. (2018). Using trial-level data and multilevel modeling to investigate within-task change in event-related potentials. *Psychophysiology*, *55*(5), e13044. https://doi.org/https://doi.org/10.1111/psyp.13044

Volpert-Esmond, H. I., Page-Gould, E., & Bartholow, B. D. (2021). Using multilevel models for the analysis of event-related potentials. *International Journal of Psychophysiology*, *162*, 145-156. https://doi.org/10.1016/j.ijpsycho.2021.02.006

Walters, R. W., Hoffman, L., & Templin, J. (2018). The power to detect and predict individual differences in intra-individual variability using the mixed-effects location-scale model. *Multivariate Behavioral Research*, *53*(3), 1-15. https://doi.org/10.1080/00273171.2018.1449628

Weissman, D. H., Egner, T., Hawks, Z., & Link, J. (2015). The congruency sequence effect emerges when the distracter precedes the target. *Acta Psychologica*, *156C*, 8-21. https://doi.org/10.1016/j.actpsy.2015.01.003

Weissman, D. H., Jiang, J., & Egner, T. (2014). Determinants of congruency sequence effects without learning and memory confounds. *Journal of Experimental Psychology*, *40*(5), 2022-2037. https://doi.org/10.1037/a0037454

Wendt, M., Kluwe, R. H., & Peters, A. (2006). Sequential modulations of interference evoked by processing task-irrelevant stimulus features. *J. Exp. Psychol. Hum. Percept*, *32*(3), 644-667. https://doi.org/10.1037/0096-1523.32.3.644

West, R., & Alain, C. (1999). Event-related neural activity associated with the Stroop task. *Cognitive Brain Research*, *8*(2), 157-164. https://doi.org/10.1016/s0926-6410(99)00017-8

West, R., & Alain, C. (2000). Effects of task context and fluctuations of attention on neural activity supporting performance of the Stroop task. *Brain Research*, *873*(1), 102-111. https://doi.org/10.1016/s0006-8993(00)02530-0

West, R., & Bailey, K. (2012). ERP correlates of dual mechanisms of control in the counting Stroop task. *Psychophysiology*, *49*(10), 1309-1318. https://doi.org/10.1111/j.1469-8986.2012.01464.x

Author Manuscript

Williams, A. D., & Grisham, J. R. (2013). Cognitive Bias Modification (CBM) of obsessive compulsive beliefs. *BMC Psychiatry*, *13*(1), 256. https://doi.org/10.1186/1471-244X-13-256

Williams, D. R., Martin, S. R., Liu, S., & Rast, P. (2020). Bayesian multivariate mixed-effects location scale modeling of longitudinal relations among affective traits, states, and physical activity. *European Journal of Psychological Assessment*, *36*(6), 981-997. https://doi.org/10.1027/1015-5759/a000624

Williams, D. R., Mulder, J., Rouder, J. N., & Rast, P. (2021). Beneath the surface: Unearthing within-person variability and mean relations with Bayesian mixed models. *Psychological Methods*, *26*(1), 74-89. https://doi.org/10.1037/met0000270

Yang, Q., Paul, K., & Pourtois, G. (2019). Defensive motivation increases conflict adaptation through local changes in cognitive control: Evidence from ERPs and mid-frontal theta. *Biological Psychology*, *148*, 107738. https://doi.org/https://doi.org/10.1016/j.biopsycho.2019.107738

Yang, W., Ding, Z., Dai, T., Peng, F., & Zhang, J. X. (2015). Attention Bias Modification training in individuals with depressive symptoms: A randomized controlled trial. *Journal of Behavior Therapy and Experimental Psychiatry*, *49*, 101-111. https://doi.org/https://doi.org/10.1016/j.jbtep.2014.08.005

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review*, *111*(4), 931-959. https://doi.org/10.1037/0033-295x.111.4.931

Yeung, N., & Nieuwenhuis, S. (2009). Dissociating response conflict and error likelihood in anterior cingulate cortex [Article]. *Journal of Neuroscience*, *29*(46), 14506-14510. https://doi.org/10.1523/jneurosci.3615-09.2009

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Table 1

Summary Demographic, Diagnostic Information, Response Times, And Error Rate Data for Behavioral Analyses

	Controls $n = 56$		GAD $n = 44$		MDD $n = 51$		OCD $n = 31$	
	n	%	*n*	%	*n*	%	*n*	%
Women	35	63	37	84	39	77	13	42
Medicated	0	0	28	64	36	71	17	55
	Mean	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
Age (years)	21.4	4.4	21.6	5.2	22.2	4.4	21.2	2.3
Years of Education	13.6	1.4	14.1	1.5	14.3	1.5	14.0	1.4
BDI-II	2.9	3.2	15.2	8.7	19.1	11.1	13.6	11.8
PSWQ	37.8	10.6	60.8	9.9	56.4	12.9	55.1	12.4
Y-BOCS	1.7	2.9	9.1	8.3	6.6	6.9	18.4	9.3
Response Times (ms)								
cC	542	82	551	82	548	70	556	71
iC	562	84	569	81	567	71	578	75
cI	661	94	670	93	680	77	686	80
iI	650	93	660	93	672	75	666	78
N450 Amplitude (μV)								
cC	-0.5	1.6	-1.0	1.4	-0.6	1.3	-0.6	1.3
iC	-0.3	1.5	-0.9	1.3	-0.4	1.2	-0.3	1.3
cI	-0.7	1.4	-1.1	1.4	-0.7	1.2	-0.7	1.3
iI	-0.6	1.4	-0.9	1.3	-0.5	1.2	-0.4	1.3
Conflict SP (μV)								
cC	0.8	1.6	0.5	1.7	0.9	1.1	0.6	1.9
iC	0.8	1.5	0.5	1.7	0.9	1.3	0.6	2.1
cI	2.0	1.8	1.1	2.0	1.6	1.3	1.2	2.0
iI	1.4	1.6	0.9	1.9	1.1	1.2	0.8	2.0

Note: A total of 12 participants with a primary diagnosis of GAD also had a comorbid MDD diagnosis, and a total of 19 participants with a primary diagnosis of MDD also had a comorbid diagnosis of GAD. Participants with OCD did not have any identified comorbidities. BDI-II = Beck Depression Inventory-II; PSWQ = Penn State Worry Questionnaire; Y-BOCS = Yale-Brown Obsessive Compulsive Scale; cC = congruent trial preceded by a congruent trial; cI = incongruent trial preceded by a congruent trial; iC = congruent trial preceded by an incongruent trial; iI = incongruent trial preceded by an incongruent trial; conflict SP = conflict slow potential

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Table 2

Estimates from Location-Scale Multilevel Models Predicting Response Times

Predictor	Estimate	*SE*	95% CrI
Location Portion			
Current Congruent			
Intercept (Previous Congruent)	548.66	4.93	539.37, 558.42
Previous Incongruent	19.97	1.25	17.52, 22.46
Current Incongruent			
Intercept (Previous Congruent)	672.8	5.54	662.10, 683.53
Previous Incongruent	-11.57	1.45	-14.43, -8.72
Scale Portion (*SD*)			
Current Congruent			
Intercept (Previous Congruent)	4.93	0.02	4.90, 4.97
Previous Incongruent	0.003	0.01	-0.02, 0.02
Current Incongruent			
Intercept (Previous Congruent)	5.00	0.02	4.96, 5.04
Previous Incongruent	-0.01	0.01	-0.03, 0.01
Random Effects			
Mean Structure			
Current Congruent			
Intercept (Previous Congruent)	68.76	2.98	62.86, 74.62
Previous Incongruent	7.11	1.34	4.52, 9.79
Current Incongruent			
Intercept (Previous Congruent)	76.25	3.44	69.71, 83.31
Previous Incongruent	9.66	1.47	6.86, 12.63
Variance Structure (*SD*)			
Current Congruent			
Intercept (Previous Congruent)	0.25	0.01	0.23, 0.27
Previous Incongruent	0.12	0.01	0.10, 0.14
Current Incongruent			
Intercept (Previous Congruent)	0.25	0.01	0.22, 0.27
Previous Incongruent	0.12	0.01	0.10, 0.14

Note: Parameters in standard deviations (*SD*) units are shown on a log scale. *SE* = standard error; 95% CrI = 95% credible interval

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Table 3

Estimates from Location-Scale Multilevel Models Predicting N450 Amplitudes

Predictor	Estimate	*SE*	95% CrI
Location Portion			
Current Congruent			
Intercept (Previous Congruent)	-0.68	0.09	-0.87, -0.50
Previous Incongruent	0.20	0.03	0.14, 0.26
Current Incongruent			
Intercept (Previous Congruent)	-0.78	0.09	-0.97, -0.60
Previous Incongruent	0.17	0.03	0.10, 0.24
Scale Portion (*SD*)			
Current Congruent			
Intercept (Previous Congruent)	1.37	0.02	1.33, 1.40
Previous Incongruent	0.004	0.01	-0.01, 0.02
Current Incongruent			
Intercept (Previous Congruent)	1.37	0.02	1.33, 1.40
Previous Incongruent	0.003	0.01	-0.01, 0.02
Random Effects			
Mean Structure			
Current Congruent			
Intercept (Previous Congruent)	1.27	0.07	1.15, 1.41
Previous Incongruent	0.09	0.05	0.01, 0.20
Current Incongruent			
Intercept (Previous Congruent)	1.22	0.06	1.10, 1.35
Previous Incongruent	0.12	0.06	0.01, 0.23
Variance Structure (*SD*)			
Current Congruent			
Intercept (Previous Congruent)	0.24	0.01	0.22, 0.27
Previous Incongruent	0.09	0.01	0.08, 0.11
Current Incongruent			
Intercept (Previous Congruent)	0.24	0.01	0.22, 0.27
Previous Incongruent	0.08	0.01	0.06, 0.09

Note: Parameters in standard deviations (*SD*) units are shown on a log scale. *SE* = standard error; 95% CrI = 95% credible interval

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Table 4

Estimates from Location-Scale Multilevel Models Predicting Conflict SP Amplitudes

Predictor	Estimate	*SE*	95% CrI
Location Portion			
Current Congruent			
Intercept (Previous Congruent)	0.74	0.11	0.52, 0.96
Previous Incongruent	-0.04	0.04	-0.12, 0.04
Current Incongruent			
Intercept (Previous Congruent)	1.51	0.12	1.28, 1.75
Previous Incongruent	-0.42	0.04	-0.50, -0.33
Scale Portion (*SD*)			
Current Congruent			
Intercept (Previous Congruent)	1.60	0.01	1.57, 1.63
Previous Incongruent	-0.01	0.01	-0.02, 0.01
Current Incongruent			
Intercept (Previous Congruent)	1.58	0.02	1.55, 1.61
Previous Incongruent	0.004	0.01	-0.01, 0.02
Random Effects			
Mean Structure			
Current Congruent			
Intercept (Previous Congruent)	1.47	0.08	1.33, 1.63
Previous Incongruent	0.16	0.07	0.02, 0.30
Current Incongruent			
Intercept (Previous Congruent)	1.62	0.08	1.46, 1.78
Previous Incongruent	0.17	0.07	0.02, 0.30
Variance Structure (*SD*)			
Current Congruent			
Intercept (Previous Congruent)	0.19	0.01	0.17, 0.21
Previous Incongruent	0.09	0.01	0.07, 0.10
Current Incongruent			
Intercept (Previous Congruent)	0.20	0.01	0.18, 0.22
Previous Incongruent	0.09	0.01	0.08, 0.11

Note: Parameters in standard deviations (*SD*) units are shown on a log scale. *SE* = standard error; 95% CrI = 95% credible interval

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Table 5

Correlations among RT, N450, and Conflict SP for the Differences for Congruent and Incongruent Trials as a Function of Previous-Trial Congruency

			Scale					
			RT		N450		SP	
			cC - iC	cI - iI	cC - iC	cI - iI	cC - iC	cI - iI
Location	RT	cC - iC	0.54 [0.31, 0.73]	-0.03 [-0.27, 0.22]	-0.09 [-0.35, 0.18]	-0.06 [-0.34, 0.21]	-0.13 [-0.40, 0.16]	0.15 [-0.11, 0.41]
		cI - iI	0.01 [-0.22, 0.24]	0.70 [0.53, 0.83]	0.06 [-0.18, 0.31]	-0.10 [-0.36, 0.16]	-0.09 [-0.34, 0.16]	0.04 [-0.21, 0.29]
	N450	cC - iC	0.06 [-0.30, 0.40]	0.03 [-0.32, 0.37]	0.03 [-0.33, 0.38]	-0.04 [-0.37, 0.29]	0.05 [-0.32, 0.40]	-0.11 [-0.43, 0.25]
		cI - iI	0.04 [-0.30, 0.36]	-0.08 [-0.40, 0.25]	-0.03 [-0.35, 0.30]	-0.04 [-0.35, 0.30]	0.09 [-0.25, 0.41]	-0.06 [-0.39, 0.28]
	SP	cC - iC	-0.09 [-0.39, 0.24]	-0.12 [-0.42, 0.21]	0.09 [-0.24, 0.41]	0.07 [-0.27, 0.41]	0.03 [-0.29, 0.36]	0.06 [-0.27, 0.39]
		cI - iI	0.12 [-0.21, 0.42]	0.04 [-0.28, 0.35]	0.00 [-0.33, 0.32]	0.03 [-0.30, 0.35]	-0.08 [-0.40, 0.26]	0.14 [-0.21, 0.46]

Note: cC minus iC refers to the difference between a congruent trial preceded by a congruent trial (cC) and a congruent trial preceded by an incongruent trial (iC). cI minus iI refers to the difference between a incongruent trial preceded by a congruent trial (cI) and an incongruent trial preceded by a congruent trial (cI). RT = response time; SP = conflict slow potential

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Table 6

Correlations among Congruency Effects and Congruency Sequence Effect for RT, N450, and Conflict SP

		Scale		
Congruency Effects		RT	N450	SP
Location	RT	0.44 [0.30, 0.57]	0.15 [-0.06, 0.36]	-0.08 [-0.26, 0.09]
	N450	0.02 [-0.17, 0.20]	0.08 [-0.17, 0.32]	0.05 [-0.17, 0.26]
	SP	-0.01 [-0.18, 0.14]	0.12 [-0.11, 0.34]	0.18 [-0.01, 0.36]
Congruency Sequence Effects		RT	N450	SP
Location	RT	0.59 [0.36, 0.77]	-0.14 [-0.41, 0.15]	0.00 [-0.27, 0.27]
	N450	-0.04 [-0.39, 0.33]	0.01 [-0.35, 0.37]	-0.03 [-0.38, 0.34]
	SP	-0.04 [-0.36, 0.29]	0.03 [-0.32, 0.37]	0.13 [-0.23, 0.46]

Note: Congruency effects (incongruent minus congruent trials) in the top panel and congruency sequence effects ((cI – cC) – (iI – iC)) in the bottom panel. cC = congruent trial preceded by a congruent trial; cI = incongruent trial preceded by a congruent trial; iC = congruent trial preceded by an incongruent trial; iI = incongruent trial preceded by an incongruent trial; RT = response time; conflict SP = conflict slow potential

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Table 7

Interaction Effects for Congruency Sequence Effects (CSEs) and Psychopathology Groups and Psychiatric Symptoms

	RT	N450	Conflict SP
GAD	[-5.88, 18.30]	[-.21, 0.41]	[-.27, 0.54]
MDD	[-8.17, 12.10]	[-.23, 0.30]	[-.44, 0.25]
OCD	[-18.03, 3.10]	[-.16, 0.37]	[-.18, 0.51]
PSWQ	[-6.12, 3.66]	[-.21, 0.04]	[-.09, .24]
BDI-II	[-4.75, 4.53]	[-.08, 0.14]	[-.11, 0.19]
YBOCS	[-2.17, 6.20]	[-.02, 0.19]	[-.17, 0.10]

Note: 95% credible intervals for the CSE x Group interaction contrasts. RT = response time; Conflict SP = conflict slow potential; GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive-compulsive disorder

Figure Captions

Figure 1. Grand average N450 waveforms stimulus-locked to target onset as a function of group. The N450 waveforms reflect activity from 325 to 425 ms (shaded) following target onset across multiple fronto-medial sites (5, 6 [FCz], 12, 13, 112). Topographical maps of N450 difference activity (incongruent minus congruent trials) for each group are shown in the top-right corner of each plot, and the maps represent the average N450 difference activity from 325 to 425 ms. GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive-compulsive disorder; cC = congruent trial preceded by a congruent trial; cI = incongruent trial preceded by a congruent trial; iC = congruent trial preceded by an incongruent trial; iI = incongruent trial preceded by an incongruent trial

Figure 2. Grand average conflict slow potential (conflict SP) waveforms stimulus-locked to target onset as a function of group. The conflict SP waveforms reflect activity from 550 to 700 ms (shaded) following target onset across multiple parietal sites (60, 66, 67, 71, 72, 76, 77, 84, 85). Topographical maps of conflict SP difference activity (incongruent minus congruent trials) for each group are shown in the top-right corner of each plot, and the maps represent the average conflict SP difference activity from 550 to 700 ms. GAD = generalized anxiety disorder; MDD = major depressive disorder; OCD = obsessive-compulsive disorder; cC = congruent trial preceded by a congruent trial; cI = incongruent trial preceded by a congruent trial; iC = congruent trial preceded by an incongruent trial; iI = incongruent trial preceded by an incongruent trial

Figure 3. Scatterplots for mean-variance relationships with congruency effects (incongruent minus congruent trials) in the left column and congruency sequence effects ((cI – cC) – (iI – iC)) in the right column. The intraindividual variability (IIV) is show on the y axes and the mean effects are shown on the x axes. cC = congruent trial preceded by a congruent trial; cI = incongruent trial preceded by a congruent trial; iC = congruent trial preceded by an incongruent trial; iI = incongruent trial preceded by an incongruent trial; conflict SP = conflict slow potential

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Data Code and Availability

Data and code supporting findings of this study are available upon request.

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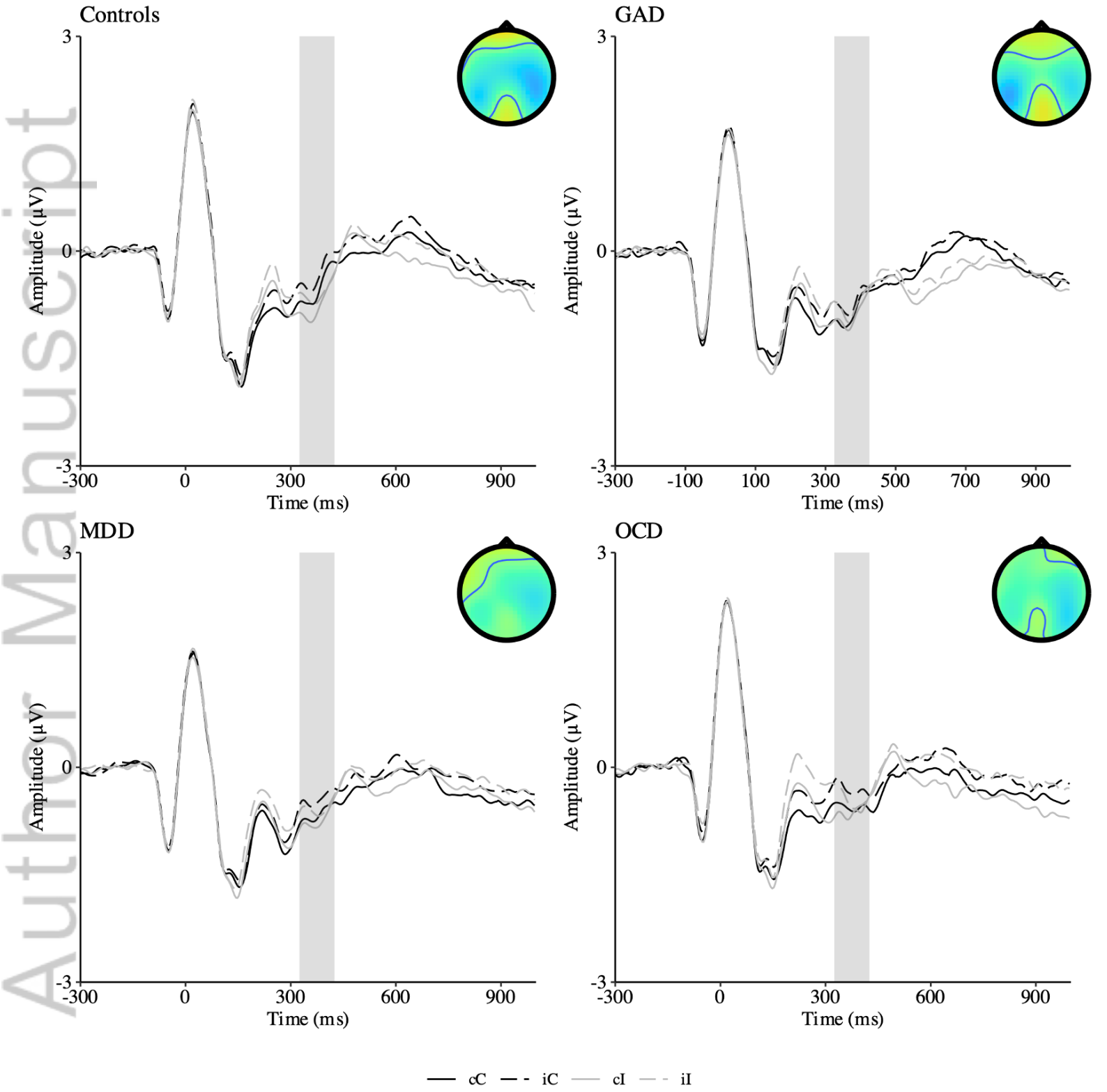


Figure1.png

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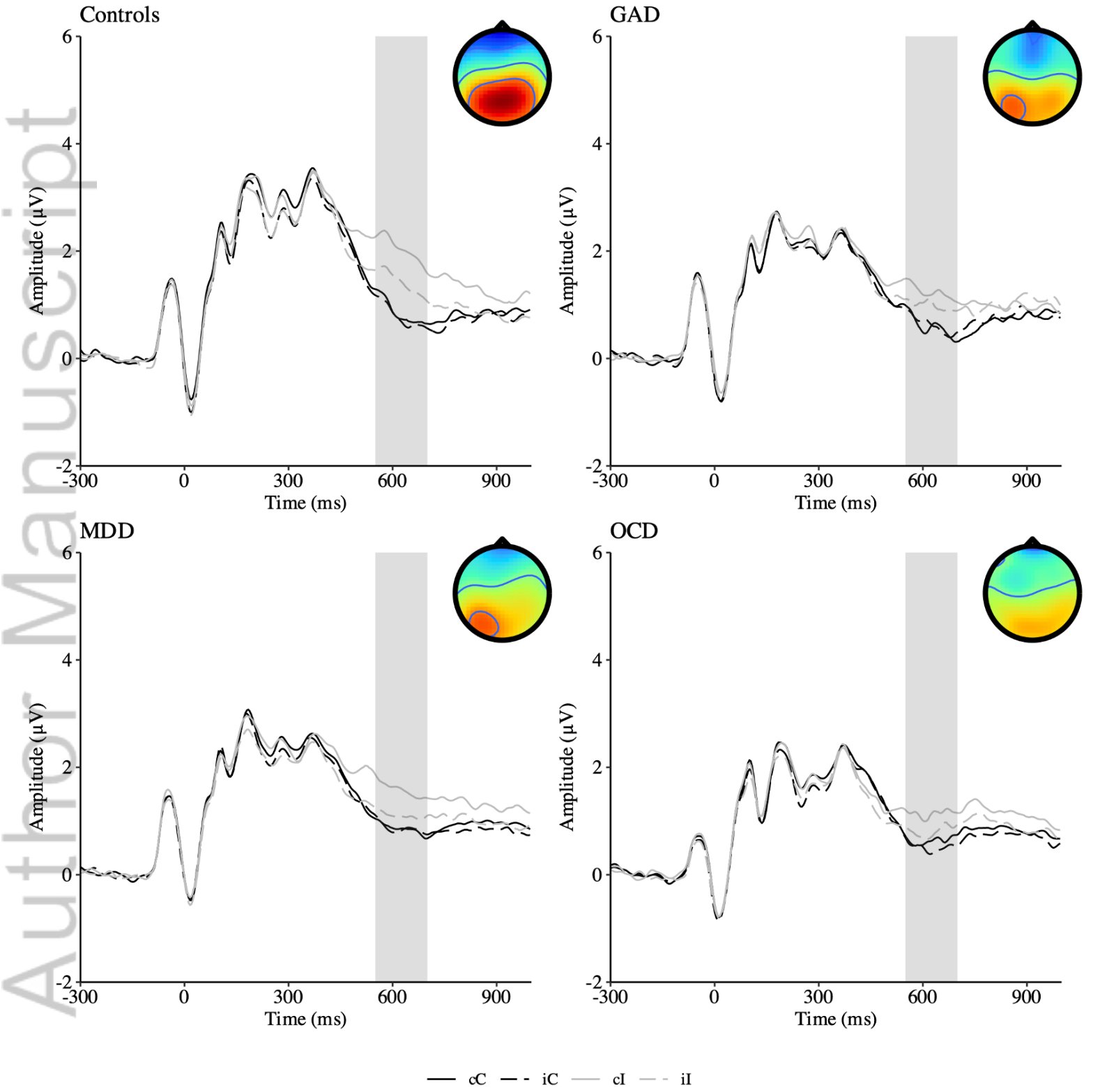


Figure2.png

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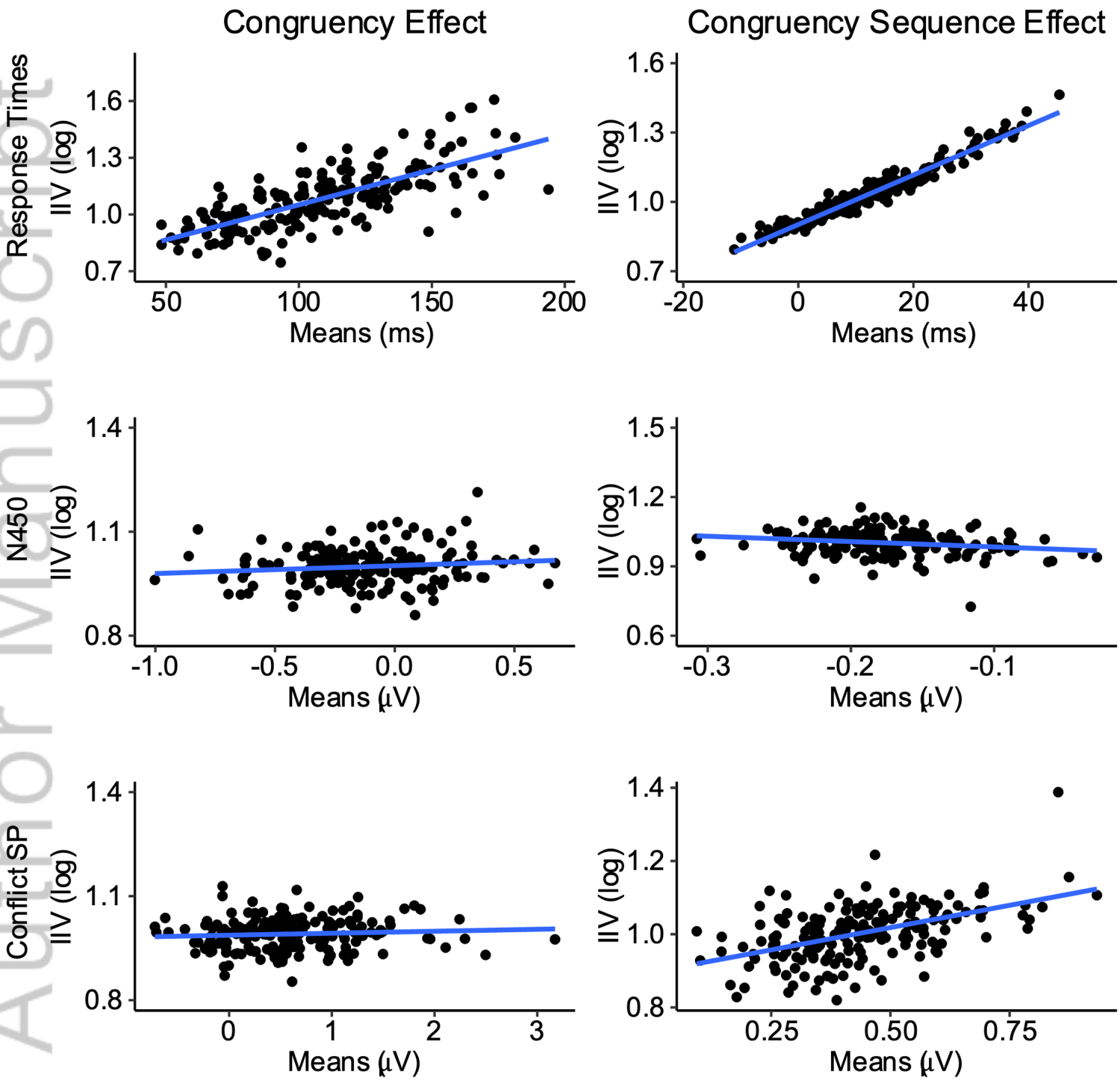


Figure3.png