PROJECT SUMMARY

Overview:

We propose a novel neurocognitive modeling technique that allows simultaneous predictions of neural and behavioral data. The technique has as a major advantage that new models can now be applied that were previously impossible due to parameter identification issues. We propose a particular example of such a neurocognitive model and outline a sequence of experiments in which we test the assumptions of the example model. The example extends the classical diffusion decision model with parameters that correspond to visual encoding time, motor response time, and allows for the estimation of within-trial decision noise.

Intellectual Merit :

The joint modeling of neural and behavioral data has been the topic of much recent interest. The primary advantage of joint modeling is that it improves researchers' ability to estimate parameters of neural, cognitive, or behavioral models by using constraints imposed by an additional data mode.

An advantage of joint modeling that has thus far been underexploited is the capacity to construct genuine neurocognitive models that are informed by both behavioral and neural data. Indeed, joint estimation opens up the possibility to construct entirely new models whose parameters are only estimable from more than one type of information. We will develop a multimodal sequential accumulation model that makes predictions about the combination of reaction time, accuracy, and EEG data, and that allows for conclusions not possible from either type of data individually. Specifically, we propose to collect new data sets and apply new models that will allow us to disentangle parameters of a cognitive model that cannot be estimated without the use of both neural and behavioral data.

Our data will also provide a direct test of the classical modeling assumption that visual encoding, decision-making, and executing a motor response are sequential processes.

Broader Impacts :

The success of the proposed project will have broad repercussions for the new field of neurocognitive modeling. A more powerful joint-modeling technique will allow for better aggregation of multiple modes of data and provide a more accurate view on the effect of experimental manipulations, interventions, and treatments.

More broadly, our methods will not be limited to the specific cognitive model (the decision-diffusion model) nor will they be limited to the EEG data we will collect. Our modeling strategy, which borrow heavily from the statistical literature on latent variable modeling, will be generic so that fMRI or single-cell data may be included instead of or in addition to EEG data.

As part of a commitment to allow others to maximally benefit from this project, the new method will be actively disseminated through publications, conferences, workshops, teaching, software, and tutorials. Furthermore, all data and software created will be made freely available online. By making the method accessible with tutorials and freely available software and example data sets, the proposed project will enable all researchers to add joint modeling to their analytic toolkit.

The project will involve junior and senior research associates who will receive training and start a career in neuroscience or cognitive science.

Estimation of unidentified cognitive models with physiological data

Joachim Vandekerckhove and Ramesh Srinivasan
University of California, Irvine

Introduction

Neurocognitive modeling has generated considerable interest as a tool to improve our understanding of brain mechanisms that underlie cognitive functions (Tong & Pratte, 2012; Turner, Forstmann, Love, Palmeri, & Van Maanen, 2016). In most studies, cognitive models are fit to behavioral data (e.g., response time and accuracy) and the parameters of these models are correlated to brain signals in order to associate brain activity with specific cognitive functions (see Turner et al., 2013, for one example). More recently, there has been interest in *joint modeling* of brain signals and behavior (Frank et al., 2015; M. D. Nunez, Vandekerckhove, & Srinivasan, 2016). The primary advantage of joint modeling is that it improves researchers' ability to estimate parameters of neural, cognitive, or behavioral models by using constraints imposed by an additional data mode. For example, M. D. Nunez, Vandekerckhove, and Srinivasan (2016) used single-trial EEG measurements to estimate trial-specific parameters of a diffusion model – a model of speeded human decision making that is only very weakly identified by the behavioral data alone.

Neurocognitive models have already led to a more sophisticated understanding of brain data and brain mechanisms of cognition. In the proposed project we will develop an approach to use brain data to directly constrain the cognitive models to improve our understanding of human cognition. **A major untapped advantage of joint modeling is the capacity to *construct genuine neurocognitive models* that are informed by both behavioral and neural data.** In this approach, we make use of the correlations between neural data and cognitive processes to improve our estimates of the cognitive processes underlying behavior. Indeed, joint estimation opens up the possibility to construct models whose parameters are only estimable from both behavioral and brain data. The added power derives from the fact that *these models simultaneously predict both behavior and brain activity* and thus parameter estimates and statistical inferences can be informed by both types of data. In what follows, we will propose a specific multimodal sequential accumulation model of perceptual decision making that is fit to a combination of reaction time, accuracy, and EEG data, and can be used to predict both behavior and EEG data.

While there exists a multitude of theoretical and experimental work studying *micro*-scale electrophysiological decision making (i.e., single neurons that track the cognitive process and neural computation of decision making; Gold & Shadlen, 2007; Shadlen & Newsome, 2001), there exists little theoretical work to inform experimental results on the *meso*- or *macro*-scale level in electrophysiology. The electroencephalogram (EEG) records cortical synaptic activity that is synchronized across the cortex and is thought to represent higher-level function in electrophysiology (Buzsaki, 2006; P. L. Nunez & Srinivasan, 2006). With cognitive models of decision making that are informed by brain activity—as recorded with EEG—we can improve our understanding of the human decision making process.

The strategy of using physiological data to constrain parameter estimates in cognitive models is not restricted to electrophysiological potentials. Neuroimaging methods also offer different types of information that can potentially be used to identify cognitive models. Inference for cognitive models can be gained using functional magnetic resonance (fMRI) measures of blood-oxygen-level dependent (BOLD) activity and functional connectivity measures. Structural MRI measures (e.g., gray matter thickness), or corticocortical and thalamocortical white-matter tracts using Diffusion Tensor Imaging (DTI) could be used to inform individual differences in cognitive processes. **The methodology we propose could be readily applied to such fMRI, MRI, and DTI measures.**

Joint modeling techniques will lead to the development of new theoretical models of "neurocognition" that predict both neural data and human behavior. While we have already demonstrated a simple joint model that can successfully predict behavioral data from EEG inputs (M. D. Nunez, Vandekerckhove, & Srinivasan, 2016), we also propose more powerful applications that would be to *predict neurophysiological properties from behavioral data alone*. This application of neurocognitive models has the potential to expose decision-making and attention deficits in certain patient populations—such as schizophrenic and stroke populations—by comparing discerned cognitive deficits from neurocognitive models. Future hypotheses of human cognition and deficits in patient cognition can be tested in the proposed frameworks.

Separation of encoding, decision, and motor processes during human decision making

A *decision-diffusion model* (DDM; Link, 1975; Ratcliff & McKoon, 2008) states that reaction time and accuracy are explained by a continuous accumulation of evidence towards certain predetermined evidence thresholds. That is, it is assumed that on any given trial a subject accumulates evidence until sufficient evidence is available for one choice over another. Models of this nature have been used widely in the cognitive modeling literature and applied to a broad range of empirical phenomena. Cognitive parameters of the diffusion model have also been empirically validated with tests of selective influence (Voss, Rothermund, & Voss, 2004). Variations on the DDM have been applied to studies in perceptual discrimination (Ratcliff, Thapar, & McKoon, 2006), letter identification (Ratcliff, Gomez, & McKoon, 2004), lexical decision (Wagenmakers, Ratcliff, Gomez, & McKoon, 2008), categorization (Klauer, Voss, Schmitz, & Teige-Mocigemba, 2007), recognition memory (Ratcliff, 1978), and intelligence (Ratcliff, Thapar, & McKoon, 2010). DDMs have also been applied in neurophysiological studies, such as examining the effects on decision making of alcohol intake (van Ravenzwaaij, Dutilh, & Wagenmakers, 2012), sleep deprivation (Ratcliff & van Dongen, 2009), and hypoglycemia (Geddes et al., 2010). The model has also been applied in the neurosciences (Mulder, Wagenmakers, Ratcliff, Boekel, & Forstmann, 2012; Philiastides, Ratcliff, & Sajda, 2006; Ratcliff, Hasegawa, Hasegawa, Smith, & Segraves, 2007).

The identification of the cognitive processes underlying behavior, and the specification of which process is influenced by task manipulation, disease state, or pharmacological agent depends on the statistical identification of the model. A critical issue in making of practical use of DDM is the problem of unidentifiable parameters. A model parameter is said to be *classically identified* when a data set yields a unique maximum-likelihood estimate for that parameter. That is, if (given a data set X) the likelihood of a model $p(X|\theta)$ reaches its maximum value at θ_1, the parameter is identified if for all $p(X|\theta_1) = p(X|\theta_2) \Rightarrow \theta_1 = \theta_2$: no *other* parameter value gives an equally good fit. In this sense, the DDM incorporates two identification problems, where estimation methods based on behavioral data alone require additional assumptions. One identification problem is found in the model of evidence accumulation; the other is in the model of time taken for perceptual and motor processes (see Tuerlinckx, 2004, for more mathematical detail on the DDM).

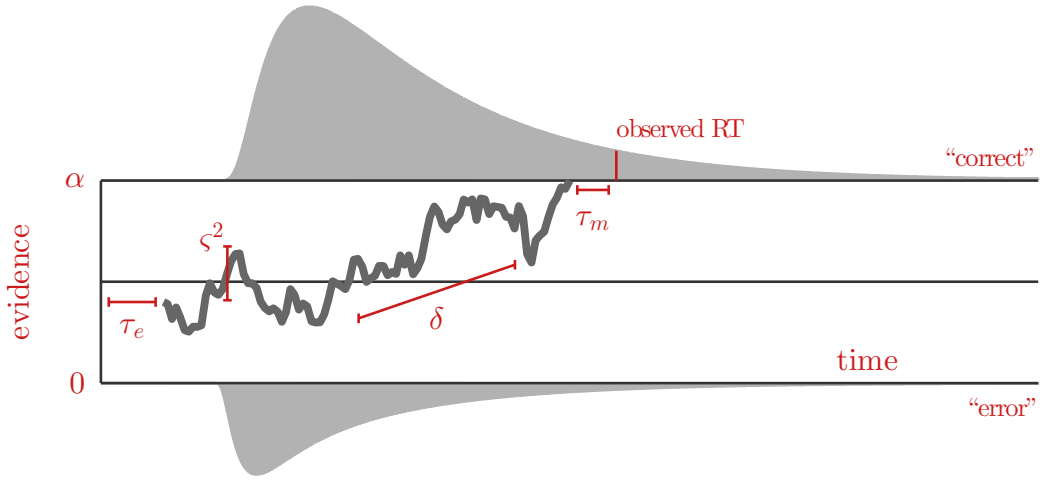

Figure 1. A graphical illustration of the diffusion decision model (DDM) for two-choice response times. The figure emphasizes the dual nature of the nondecision time component τ, which is an aggregate of time needed for encoding the stimulus τ_e and time needed to execute the motor response τ_m. Other central parameters are the distance between the two absorbing boundaries α, the evidence accumulation rate δ, and the variance of the evidence accumulation process ς^2. Together, these parameters give rise to the bivariate choice-latency distribution.

Figure 1 illustrates the process of evidence accumulation and each of the three parameters involved in estimating the DDM. The thick line indicates a cognitive representation of evidence being accumulated over time, following a noisy trajectory that on average trends upward. The mean trend of the cognitive process is captured by the drift rate parameter δ: if δ is large and positive, the process is fast and will rapidly hit the upper evidence threshold, indicating a fast, correct decision. If δ is close to 0, the process will hover around its starting point and reach the upper or lower boundary with approximately equal probability. The drift rate is commonly interpreted as the "ability" parameter. The instantaneous variance of this process is given by the diffusion coefficient ς^2 and is affected by internal noise in the cognitive process. The parameter α captures the distance between the upper and lower boundary of required cognitive evidence, and so quantifies the amount of information needed to make a decision. If it is high, responses will tend to be slow but correspond tightly to the direction of the process drift, emulating a cautious and deliberate process. If α is low, decisions will be fast but the randomness of the accumulation process will cause it to hit the wrong bound with increasing probability. Boundary separation α is typically interpreted as the "caution" parameter.

In the classical DDM, these three "evidence-related" parameters cannot all be identified from the response time and accuracy data because they exhibit a trade-off relationship such that no $(\delta, \varsigma^2, \alpha)$ triplet uniquely provides the best fit to the data. In most applications of the DDM, the diffusion coefficient ς^2 is chosen as the unidentified parameter that cannot be estimated and is instead given an arbitrary value (often 0.1 or 1), fixed across individuals and experimental conditions. In other words, all experimental effects and natural differences are assumed to arise only due to changes in drift rate ("ability") or boundary separation ("caution"). In our proposed neurocognitive model this assumption is relaxed by taking advantage of relationships between brain signals and the decision process. For example, this cognitive evidence accumulation process is thought to be reflected in certain neurons of the lateral intraparietal areas (LIP) in primates

(Shadlen & Newsome, 2001) and the process has been shown to be observable using EEG in humans (O'Connell, Dockree, & Kelly, 2012; Philiastides, Heekeren, & Sajda, 2014; Twomey, Murphy, Kelly, & O'Connell, 2015). Neural markers of boundary separation have been previously explored by Frank et al. (2015), who found that trial-to-trial BOLD activity in the subthalamic nucleus and trial-to-trial fluctuations in frontal theta band power (4-8 Hz) track trial-to-trial differences in the evidence threshold. Moreover, we will propose neural markers of decreases in internal noise (below; in Phase 2).

The DDM has an additional identification problem in the time domain. The DDM has a "nondecision time" parameter τ that is sometimes referred to as "the time for encoding and responding" or "T_{er}" (e.g., Ratcliff & McKoon, 2008). As the parameter is meant to quantify the total duration of nondecisional processes such as encoding the stimulus (before the decision making process) and executing the motor response (after), it clearly captures two separate components. According to the classical DDM, the total reaction time (RT) is the sum of the decision time (DT) and these two nondecisional components: $RT = \tau_e + DT + \tau_m$. We will refer to this as *the additive assumption* of the DDM. In Figure 1, we have illustrated these two nondecision time components separately. In the classical DDM, the time for encoding τ_e and the motor execution time τ_m are unidentified. It is clear from the additive assumption above that no unique maximum likelihood estimates can exist for τ_e and τ_m, since we can find many alternative pairs $\tau_e^* = \tau_e + \varepsilon$ and $\tau_m^* = \tau_m - \varepsilon$ that lead to the same predicted RT: $RT = \tau_e^* + DT + \tau_m^* = (\tau_e + \varepsilon) + DT + (\tau_m - \varepsilon) = \tau_e + DT + \tau_m$. If, however, either τ_e or τ_m can be informed from some other source of information, the identification issue is resolved: Given some unique estimate of τ_e, a unique estimate of τ_m follows and vice versa. One such additional source of information that can facilitate parameter identification is the N200 latency that was shown by M. D. Nunez, Vandekerckhove, and Srinivasan (2016) to predict trial-specific nondecision times.

In what follows, we will propose a modified DDM that makes use of neural markers such as the N200 latency to estimate otherwise unidentifiable parameters, beginning with encoding time τ_e and motor response time τ_m in Phase 1. Then, we will extend the model again to allow for the simultaneous estimation of drift rate δ, diffusion coefficient ς^2, and boundary separation α (see Fig. 1) in Phase 2. In this study, we will propose and test neural markers that will allow us to estimate *all* the DDM parameters using a combination of behavioral and neural data.

The proposed modeling framework

The joint modeling framework we propose is closely related to the cognitive latent variable models (CLVM) proposed by Vandekerckhove (2014). CLVMs carry a natural capacity for joint analysis: Vandekerckhove provides as an example of such a "hybrid-data" model a scenario where behavioral and survey data are simultaneously available. In terms of model specification, only small changes are required to accommodate neurophysiological data in place of survey data.

We propose a joint neurocognitive model of (1) encoding time, (2) motor response time, and (3) decision time (via a diffusion process). In Phase 1, the respective components of this model are identified with measures of encoding time and motor preparation from stimulus time-locked and response time-locked electrocortical potentials (i.e., as recorded using EEG), and decision processes that are estimated from observed choice and reaction time distributions. In Phase 2, we propose to identify physiological components that correspond to decision making components to solve the identification problem in the decision diffusion process. By constructing a single unifying model—that, crucially, makes simultaneous predictions about behavior and brain data—we will be able to estimate the individual components. Trial-to-trial differences and individual differences will be evaluated using hierarchical models as well as both single-trial and subject-level measures of EEG.

Single-trial measures of EEG are useful markers of the cognitive proces, and have recently been exploited in the field of cognitive neuroscience (Parra, Spence, Gerson, & Sajda, 2005; Pernet, Sajda, & Rousselet, 2011). Additionally, many brain-computer interfaces (BCIs) take advantage of robust single-trial and few-trial EEG correlates of motor-preparation signals and attentional modulation of sensory responses to control physical devices (McFarland, Miner, Vaughan, & Wolpaw, 2000; Krusienski, Sellers, McFarland, Vaughan, & Wolpaw, 2008). Our approach is similar to this literature, but focused on single-trial EEG measures that are related to cognitive processes represented in the DDM. These will include: (1) single-trial onset event-related potential (ERPs; EEG time-locked to the onset of external stimuli) that are thought to reflect processing before the decision is made (e.g., negative potentials around 200ms reflecting visual encoding time; Loughnane et al., 2016; M. D. Nunez, Vandekerckhove, & Srinivasan, 2016) (2) μ-rhythms (10-13Hz) and beta rhythms (13-20 Hz) on single-trials over central electrodes (C3/C4 in the 10/20 electrode system), which are thought to reflect motor system processing and preparation (McFarland et al., 2000), (3) stimulus-locked positive amplitudes after 300ms in ERPs, known as P300s, that are thought to reflect the evidence accumulation process itself (Twomey et al., 2015) (4) α-power (8-12Hz) decreases during attentionally demanding tasks are thought to reflect decreases in the internal noise in evidence accumulation because α-power decreases predict human performance during visual tasks (see, e.g., Ergenoglu et al., 2004).

The various components of the model will be linked through a hierarchical structure as in Vandekerckhove (2014). The hierarchical structure will involve a (very) low-dimensional set of latent variables, Φ, and multiple sets of weights (loadings) Λ and Ψ. From the latent variables we can then obtain trial-specific predictions for the EEG data (x^e; e.g., N200 data) using one set of weights (Λ^e) and predictions for the trial-specific reaction time (t) and accuracy (a) using a different set of weights.

In Phase 1, we will focus on the unidentified parameters in the time dimension, specifically the encoding time τ_e and motor response time τ_m. A typical set of model assumptions for person p on trial i would be the following:

- Neural correlate for encoding time x^e_{pi} follows a normal distribution centered on the prediction, which is a linear combination of the latent factors: $x^e_{pi} \sim N\left(\Lambda^e_p \Phi_{pi}, \Sigma^e_p\right)$
- Encoding time $\tau_{e(pi)}$ is a linear combination of the *same* latent factors: $\tau_{e(pi)} = \Psi^e_p \Phi_{pi}$
- Neural correlate for motor response time x^m_{pi} follows a normal distribution centered on the prediction, which is a linear combination of the latent factors: $x^m_{pi} \sim N\left(\Lambda^m_p \Phi_{pi}, \Sigma^m_p\right)$
- Motor response time $\tau_{m(pi)}$ is a linear combination of the *same* latent factors: $\tau_{m(pi)} = \Psi^m_p \Phi_{pi}$
- Choice response time (t_{pi}, a_{pi}) follows a DDM with nondecision time fixed to $(t_{pi}, a_{pi}) \sim DDM\left(\alpha_p, \tau_{e(pi)} + \tau_{m(pi)}, \delta_{pi}, \varsigma^2_{pi}\right)$
- The dimensionality d of the latent variables (i.e., the number of rows in the matrix Φ) is low

With these assumptions, the classically unidentified parameters τ_e and τ_m are simultaneously informed by the brain data and the behavioral data, and unique estimates become available. Furthermore, the formulation of the joint structure as a set of latent variables that is projected onto different data modes with specific loadings matrices Λ will allow us to conduct confirmatory tests of the relationship between the neural correlates and cognitive model parameters – something not possible with the various approaches discussed in Turner et al. (2016). As an extreme example, constraining the loadings matrices Λ^e_p and Ψ^e_p to be equal would be a test of the hypothesis that the neural correlate x^e represents the (mean) encoding time τ_e *exactly*. This is one of the specific

models that we will employ in Phase 1. The technical aspect of applying such models is discussed in Vandekerckhove (2014).

In Phase 2, we will focus on the unidentified parameters of the evidence accumulation process. The modeling assumptions in this phase will be largely identical to those in the first phase, with the exception that different neural markers (see above) will be used to allow for simultaneous inference of the drift rate, diffusion coefficient, and boundary separation parameters.

Experimental methods

The data required to fit the joint model will be obtained through a series of four experiments—combined with exploratory and confirmatory data analysis—designed to systematically manipulate cognitive processes (and DDM parameters) during decision making. Our approach will be to carry out exploratory analyses in order to select the optimally tuned EEG feature to resolve the identification of parameters in the DDM. We then perform a confirmatory experiment wherein the process is systematically modulated. In Phase 0, we have already analyzed existing data to identify EEG features (N200 latency and mu-rhythm desynchronization) that inform on encoding time and response time. In Phase 1, we will perform two experiments to test ether we can resolve the unidentified parameters in the time domain. In Experiment 1a we will manipulate which response hand is to be used (dominant vs. nondominant) to vary motor response time and in Experiment 1b we will implement a noise manipulation to vary encoding time. These data will be also be analyzed to generate predictions of EEG features that can resolve the identification of decision model parameters, specifically the boundary separation and diffusion parameters. In addition, we will test and refine the use of the P300 to identify the drift rate. We will then perform two experiments to test the identification of the three decision parameters: Experiment 2a will involve a discrimination difficulty manipulation to vary the drift rate and Experiment 2b will require a speed/accuracy manipulation by instruction to vary the boundary separation.

Visual stimuli

To both test hypotheses of neurocognition as well as explore new avenues of neurocognitive research, in each of four experiments 25 subjects will be asked to complete two sessions of a two-alternative forced choice task while EEG is collected simultaneously. In these experiments subjects will be asked to determine whether a Gabor stimulus contained energy at high or low spatial frequencies. Stimuli for each experiment will be built and displayed using the MATLAB Psychophysics toolbox (Psychtoolbox-3). An example stimulus is given in Figure 2. A typical session will have 480 trials.

The general time course of each trial in each experiment is as follows: subjects will be asked to fixate on a fixation spot in the center of the screen throughout the experiment; visual noise is displayed for 500 ms to 1000 ms during the *cue* interval; then a Gabor signal stimulus embedded in the visual noise is displayed for 1200 ms to 2000 ms during the *response* interval. The signal to noise ratio of the visual noise and Gabor signal will change depending upon the difficulty / encoding condition (see Phase 0 and Phase 2, below). During the response interval, subjects are asked to respond as accurately or as fast as possible in the time allowed, depending upon the experimental condition. Subjects will respond using a button box with either their dominant or non-dominant hand depending upon the experimental condition (see Phase 1). Visual or auditory feedback will occur in order to keep the subject engaged and in order to explore feedback-dependent cognitive effects.

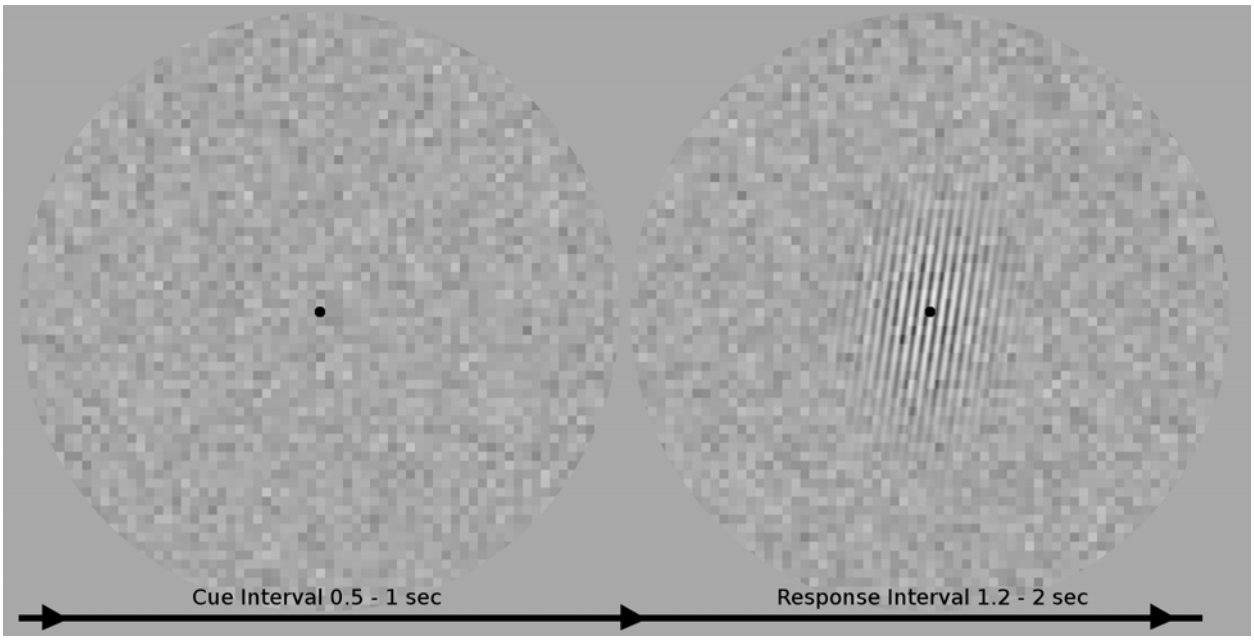

Figure 2. Example stimuli of the *cue* and *response* intervals of a high noise condition. Similar stimuli will be used for *Phases 0-2*. Subjects are first cued to the contrast of external noise during the *cue* interval. The subjects are asked to make a decision and response after the stimulus appears during the *response* interval. The onset of the visual noise and visual signal are offset in two different time intervals in order to estimate evoked potentials in the EEG to both the onset of the noise and onset of the signal. Similar methods are detailed in both M. D. Nunez et al. (2015) and M. D. Nunez, Vandekerckhove, & Srinivasan (2016)

EEG collection

EEG will be collected simultaneously using Electrical Geodesic, Inc.'s 128 electrode Hydrocell sensor nets and a Net Amps 200 series amplifier in order to record EEG from the human brain during decision making. Electrical activity from the scalp will be recorded at a sampling rate of 1000 samples per second and hardware bandpass filtered to a 1 to 100 Hz window. This hardware bandpass is chosen purposely to maintain high frequencies so that broadband noise is submitted to an Independent Component Analysis (ICA) in order to aid artifact correction (i.e., removing electrical activity due to muscle, movement and environmental electrical influence; Makeig, Bell, Jung, Sejnowski, et al., 1996; M. D. Nunez, Nunez, & Srinivasan, 2016).

Phases of the proposed work

Phase 0: Previous results

Prediction of encoding time from neural markers: N200 latency. While some cognitive parameters of the Decision Diffusion Model are classicaly unidentifiable with behavior alone, identifiability issues can resolved by including electrophysiological measures as correlates of cognition directly as observed data in hierarchical models. Using trial-level measurements of negative evoked potential peak latencies in response to visual stimuli (i.e., "N200s") in preliminary exploratory research we found evidence that trial-to-trial residual response time τ is related to the N200 latency with almost 1-to-1 correspondence in low-noise conditions. Posterior distributions of effects of single-trial N200 latency on single-trial residual response times τ for seven subjects from a hierarchical Bayesian analysis are provided in Figure 3. Taken with past results, this provides further evidence that

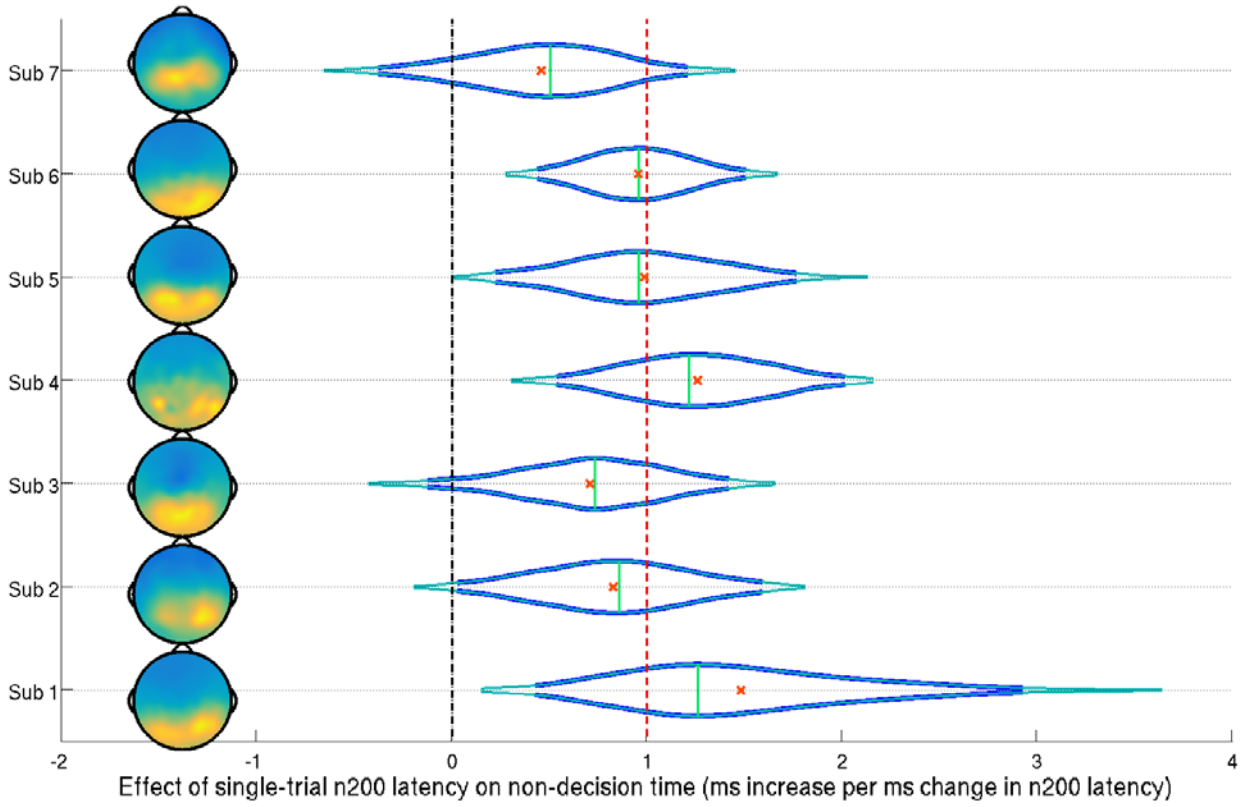

Figure 3. The posterior distributions of linear effect (i.e. "slope") parameters of a trial's N200 latency during the *response* interval on trial-specific non-decision times τ for each subject in a low noise condition. Thick lines forming the distribution functions represent 95% credible intervals while thinner lines represent 99% credible intervals. Crosses and vertical lines represent posterior means and modes respectively. Also shown are the topographic representations of the channel weights of the first SVD component of each subject's response interval ERP, indicating the location of single-trial N200s over occipital and parietal electrodes. Evidence suggests that N200 latencies have a near 1-to-1 correspondence with nondecision times as indicated by the posterior distributions of effect parameters centered on 1ms represented by the vertical dashed red line.

single-trial encoding times τ_e (e.g., visual encoding time before the decision begins in experiments with solely visual information) are reflected in N200 latencies in the parietal cortex. Further evidence in support of this theory is (1) exhibited by other studies that find evidence that visual N200 latencies reflect initial visual preprocessing (Loughnane et al., 2016), (2) that single-neuron recordings of evidence accumulation from lateral intraparietal areas (LIP) in primates typically begin at similar time periods after an experimental stimulus is displayed (Shadlen & Kiani, 2013), and (3) that visual evoked potentials that are observed less than 200ms after stimulus onset, which is assumed to be too early to affect motor response time τ_m. Neurocognitive hierarchical models of encoding, decision, and motor response time thus become identifiable when treating single-trial EEG measures of encoding (N200 latencies) as observations from a normal distribution with a mean being the true encoding time τ_e.

Motor preparation markers from the Brain-Computer Interface literature: μ-rhythm desynchronization. Our neurocognitive modeling framework will be further evaluated by (1) evaluating in-sample and out-of-sample prediction of both behavior and neural data in a previously collected

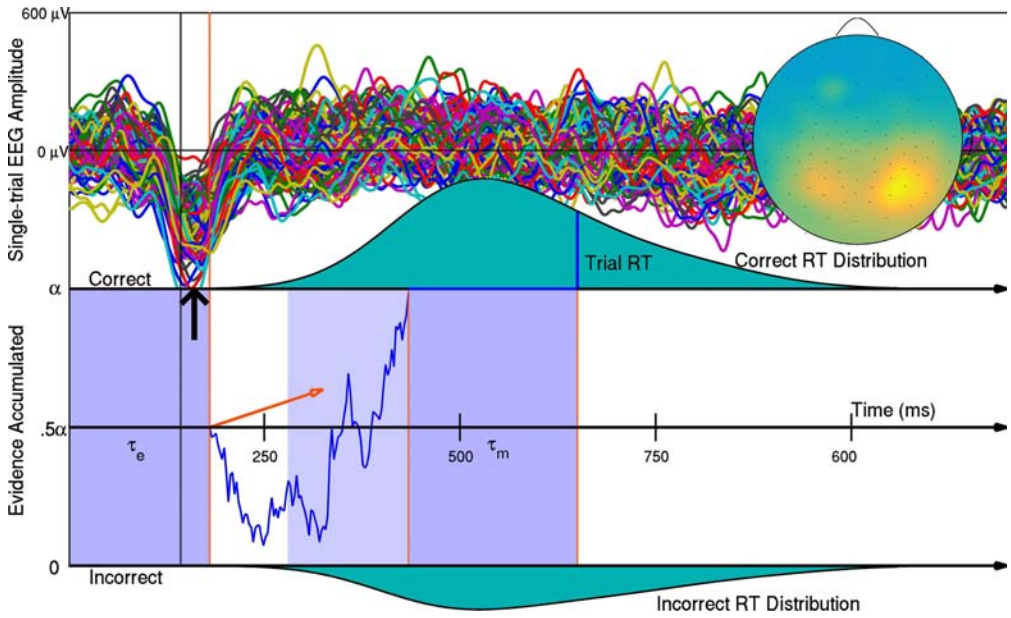

Figure 4. A graphical illustration of a Neural Decision Diffusion model in which the encoding time τ_e on single-trials describes the latency of the negative peaks of the EEG on 146 single-trials in parietal locations. Single-trial observations of the N200 latency are found by using a decomposition of the average ERP response at each electrode and then biasing the raw EEG by the resulting channel weights; this algorithm is detailed by M. D. Nunez, Vandekerckhove, & Srinivasan (2016). Located in the top-right corner of the figure, a spline-interpolated topographic representation of the channel weights on the scalp reflects the parietal location of the single-trial N200 latencies that are predicted by the Neural Decision Diffusion model. Total nondecision time τ reflects both stimulus encoding time τ_e as well as residual motor response τ_m. Motor response is thought to begin during the decision process but ends after the decision is made, as reflected by the onset and termination of the μ-rhythm desynchronization over central electrodes.

data set (the data set used in M. D. Nunez, Vandekerckhove, & Srinivasan, 2016; a strictly confirmatory analysis) and (2) evaluating the differential effects of motor preparation as exhibited by the μ-rhythm on estimated encoding, decision and motor response time. It is hypothesized that the μ-rhythm should only affect estimated motor response time and not the time in which evidence is accumulated for the decision nor the time for visual/auditory encoding of stimuli.

To further understand the decision-making process on particular trials, EEG measures of motor response preparation will inform residual motor response time (after the decision is made) in the cognitive model of decision making. The μ-rhythm (10-13Hz) in EEG recorded over the motor cortex (central electrodes) typically decreases in magnitude during motor preparation (Pfurtscheller, Brunner, Schlögl, & Lopes da Silva, 2006). This neural motor measure has also become a reliable predictor in Brain-Computer Interfaces (McFarland et al., 2000). For this reason it is thought that the time course of "desynchronization" decrease in power in the μ-band and magnitude of the power decrease will be predictive of residual motor response time (i.e. ongoing motor preparation after the decision occurs) on single-trials. We will call this marker the "μ latency." As an alternative we will also test the beta band (13-20 Hz) which has also been shown to desyncrhonize prior to movement onset, but is less frequently used in BCI's because of smaller magnitudes and greater susceptibility to artifact. The (potential) advantage of beta rhythms for this research is greater specificity of beta to motor processing, in contrast to the mu-rhythm which overlaps in frequency with the alpha rhythm which is desynchronized by a number of factors.

Predictions from previous work. **Using our previous results, we propose a specific neural marker for encoding time τ_e, namely the N200 latency, and a specific neural marker for residual motor response time τ_m, namely μ latency.**

Phase 1: Analyses in the time domain

In order to independently estimate the contribution of *encoding*, *deciding*, and *motor execution* to the total response time, we use electrophysiological markers for the first and last phases of the decision process. In doing so, we can parse the total response time into the three constituent processes as in the classical subtractive method of Donders (1868). In Phase 1, we will perform a confirmatory test of the predictions that follow from our previous results and from the BCI literature.

Because we have found in previous research (M. D. Nunez et al., 2015) and in exploratory data from Phase 0 that increased contrast of visual noise increase both nondecision time (thought to be driven by encoding time *te* differences) as well as single-trial N200 latencies, we will use a *continuous* manipulation of perceptual noise in order to test the relationship between single-trial encoding time and single-trial N200 latencies. That is, a random draw of noise contrast will be displayed to the subject during the *cue* interval on each trial of the experiment which maintains through the *response* interval, while the Gabor signal contrast remains the same on each trial. An experiment in which perceptual noise is manipulated continuously will be performed with 25 subjects as a confirmatory study.

We also will test the prediction that motor response time can be measured independently of decision time. To do so, we will experimentally manipulate the requirements of the motor response (see below), which we predict will result in an increase in the estimate of the nondecision time parameter τ of the DDM. Then, we will repeat the diffusion model analysis, but we will replace the dependent variable: rather than using the observed response time t, we will use the μ latency. If the μ-rhythm time-course indeed indicates the onset of motor response execution—as we hypothesize—the effect of motor manipulation should disappear in this condition.

To explore the separation of encoding and residual motor response time, 25 subjects will be recruited to perform two sessions of the two-alternative forced choice task described above. In each session, each subject will be given 160 trials in three different experimental blocks. The instruction during Block 1 will be to respond using a button-box using only their dominant hand. The instruction during Block 2 will be to use their non-dominant hand. And the instruction in Block 3 will be to use both hands, with the either hand responding dependent upon the subject's choice. We will obtain EEG data alongside choice response time data. It is expected that when subjects prepare to respond with their dominant hand, motor preparation, and thus residual motor response time τ_m and μ-rhythm, should occur with short latencies. The experimental manipulation should not affect either the encoding time τ_e nor the decision making period. Figure 5 illustrates the expected results if we apply a standard DDM once to the observed reaction times, and once to the observed μ-latency: under the additive assumption, the motor response manipulation should affect the total nondecision time but not the time until the onset of the motor response.

Phase 2: Analyses in the evidence domain

In Phase 2, we will attempt to find neural correlates in the evidence domain that track the evidence accumulation process. Any findings in initial exploratory research (using data collected from Phase 0 and Phase 1) will be evaluated using preregistered confirmatory research.

Accounting for the within-trial evidence accumulation path itself. Recently, macroscopic recordings of the cortex have shown that increasing EEG potentials ramping up to P300 am-

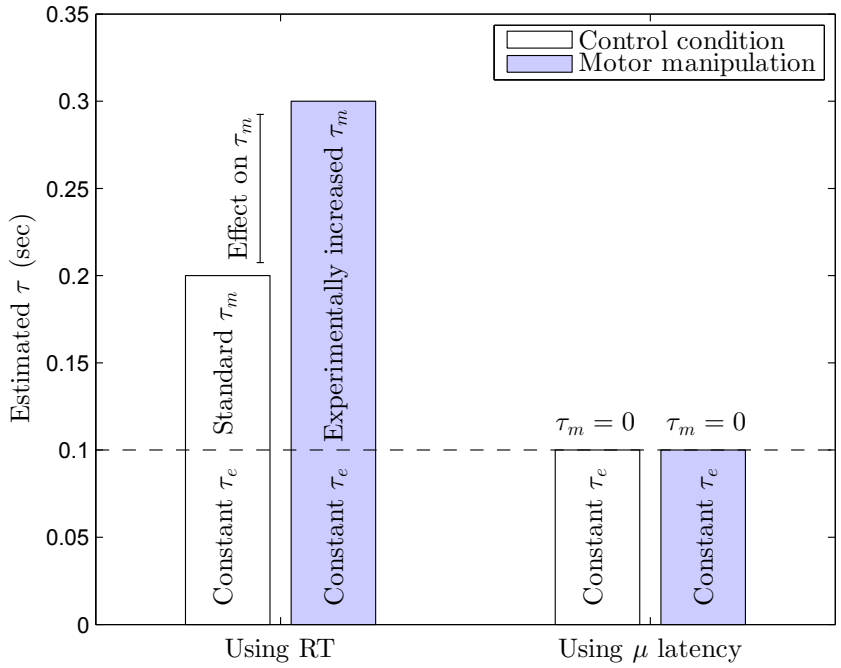

Figure 5. A depiction of predicted results from a diffusion model fit to the data from Experiment 1. The left bars indicate the estimated value nondecision time parameter τ when τ is estimated from the observed response time (RT) data. As depicted in Figure 1, the nondecision time τ is the sum of encoding time τ_e and residual motor response time τ_m. In the classical diffusion model, only the sum $\tau = \tau_e + \tau_m$ can be estimated. It is expected that our motor manipulation affects τ_m, and therefore the estimate of τ. The right bars indicate the estimate of the same parameter τ when the μ latency is used instead of RT. In this case, no effect on τ is expected since τ should then be an unbiased estimate of the encoding time τ_e, which should be unaffected by any motor manipulation.

plitudes are correlates of the stochastic accumulation of evidence during human decision making (O'Connell et al., 2012; Philiastides et al., 2014; Twomey et al., 2015). It has been hypothesized that this evoked EEG data reflects the evidence accumulation process itself – or a mixture of this process with other decision-making correlates. This hypothesis leads to the natural prediction that single-trial drift rates are explained by single-trial P300 slopes. This prediction will initially be explored with data collected from Phase 0 and Phase 1 as well as both in-sample prediction and cross-validation.

However, within a small region of the cortex, neurons will have diverse firing patterns during the decision making process, only some of which are observed to have increasing spiking-rate behavior indicative of stochastic evidence accumulation (Meister, Hennig, & Huk, 2013). The properties of volume conduction through the cortex, skull, and skin only allow for synchronous post-synaptic potentials to be observed at the scalp (P. L. Nunez & Srinivasan, 2006; Buzsaki, 2006). Therefore P300 amplitudes may be predictive of the evidence accumulation process due to the synchronization of many evidence-related neurons rather than the increasing firing rates of neurons performing evidence accumulation. In our view, the P300 is unlikely to reflect the stochastic evidence accumulation process itself, but is still a useful measure evidence accumulation at the population level. If the stochastic evidence accumulation process was truly reflected as a ramp of EEG, a testable

prediction would be that the variance around the mean rate of the P300 ramp on each trial would be linearly related to the diffusion coefficient ς^2, in addition to single-trial P300 slopes being linearly related to the drift rate δ. This prediction will be initially explored with data from 50 participants collected from Phase 1 as well as both in-sample prediction and cross-validation. We will then evaluate any exploratory findings on single-trials in a preregistered confirmatory study with 25 new subjects. Three difficulty conditions will be given to the subjects in the form of decreasing single-to-noise ratios of the visual stimuli. The experimental hypothesis is that the difficulty manipulation should affect evidence accumulation rates δ as indexed by single-trial P300 amplitudes.

Accounting for changing evidence threshold

Feedback stimuli are thought to influence perceptual learning and human decision making (e.g. Seitz, Kim, & Watanabe, 2009). That is, the brain's future computations in response to external stimuli are thought to be influenced by past-decisions regarding similar external stimuli, internal monitoring of accuracy, external feedback associated with correct responses and errors, and external feedback regarding time constraints. For this reason, measurable trial-to-trial variation in neural response to feedback stimuli may reflect variation in behavior on the following trials. ERPs measuring both internal monitoring of accuracy (dubbed "Feedback Related Negativity" Hajcak, Moser, Holroyd, & Simons, 2006) and neural encoding of external feedback (Norcia, Appelbaum, Ales, Cottereau, & Rossion, 2015) may be predictive of future decision related changes.

It is predicted that the evidence required to make a decision (i.e., the boundary separation α) will change trial-to-trial based on both external feedback-type (positive or negative feedback) and the subject's level of attention to that visual or auditory feedback on specific trials. Trial-to-trial variability in evoked frontal theta band EEG oscillatory amplitude (4-8 Hz) in response to visual stimuli has already been shown to modulate the amount of evidence required for a decision (Frank et al., 2015). Because evoked EEG responses (ERPs) typically contain their first positive and negative peaks around 100 and 175 ms respectively corresponding to a less-than 10 Hz frequency band, and the fact that later ERP components such as the P3 may reflect even slower evoked oscillations (Luck, Woodman, & Vogel, 2000), theta band activity related to the changing evidence requirement may actually reflect the attentional processing of visual input in the form of an ERP. Adding to the understanding of encoding, decision, and motor execution, the understanding of the effect of feedback in a computational model of decision making will further knowledge individual variation and deficits in perceptual decision making.

The relationship between (1) event-related EEG in response to auditory and visual feedback and (2) endogenous EEG such as frontal theta band power and trial-to-trial variability in the boundary separation will be explored in existing data from Phase 0 and Phase 1. Any findings will then be evaluated in a confirmatory study of 25 subjects with an experimental manipulation such that subjects will be given (1) directions to be as accurate as possible in 1/3 of the trials, (2) directions to be as fast as possible in 1/3 of the trials, and (3) directions to be as accurate as possible in a fixed amount of time in 1/3 of the trials. Because the speed/accuracy trade-off has previously been shown to manipulate boundary separation (Voss et al., 2004), endogenous neural responses that reflect evaluation of evidence should also be affected by this experimental condition.

Accounting for internal noise in evidence accumulation. The three evidence accumulation variables of the Decision Diffusion model can become identifiable when predicting both macro-scale electrocortical potentials as well as human behavior. For instance, the diffusion coefficient ς^2 describes the variance in the relative evidence accumulated between two choices on every trial. Assuming neural population representations of evidence (Shadlen & Kiani, 2013), the representation of evidence is expected to vary within a trial at every time step based on the amount of variance

in the neural population response due to oscillatory local field potentials (Mitchell, Sundberg, & Reynolds, 2007, 2009). Because EEG is thought to be generated from synchronous slow-wave extracellular potentials (Buzsaki, 2006; P. L. Nunez & Srinivasan, 2006) and alpha power (8-12 Hz) decreases are a well known phenomenon that predict accuracy (Ergenoglu et al., 2004): the reduction of humans' resting-state alpha rhythm over parietal and occipital cortex may reflect a neural mechanism of internal noise reduction. van Vugt, Simen, Nystrom, Holmes, and Cohen (2012) found evidence that power decreases of the theta/alpha EEG bands (4-9 Hz) predicted evidence accumulation increases, which would be an expected consequence of the power decrease reflecting internal noise decrease since the authors did not explicitly account for internal variance ς^2 in their modeling framework. We predict that trial-to-trial alpha power influences trial-to-trial evidence accumulation variances in the form of internal noise reduction.We also expect that internal noise has no effect on performance in high external noise conditions as predicted by the Perceptual Template Model (Dosher & Lu, 2000). We propose to test this hypothesis directly by finding (1) single-trial and subject-level connectors between alpha phase or power and parameter estimates of the diffusion coefficient ς^2 in hierarchical models, (2) in-sample and out-of-sample prediction for model validation.

If enough evidence for this noise suppression mechanism is found, then neural representations of estimates of ς^2 will be evaluated in models with the all three evidence accumulation parameters (α, ς^2, and δ) estimated. Model validation will occur by testing particular hypotheses of both individual differences and intrinsic trial-to-trial differences within subjects on all data collected from Phases 1 and 2. We (M. D. Nunez et al., 2015) have previously found evidence that subjects who better suppress internal noise better enhance visual signal via attention mechanisms (as predicted by the Perceptual Template Model; Lu & Dosher, 1998). An additional hypothesis is that those subjects who better suppress internal noise should have larger decreases in their endogenous alpha rhythm over parietal cortex during visual decision making tasks, reflecting internal noise differences across subjects. Individuals' alpha rhythms can then be used to separate individual differences in the three evidence accumulation parameters. We further hypothesize that internal noise as reflected by the alpha rhythm will have intrinsic trial-to-trial variability that predicts performance on the task. This hypothesis will be explored within subjects via endogenous trial-to-trial variability in each subject's alpha rhythm.

Time line

Each phase will take approximately one year. Phase 0 is complete. In the first year, we will:

(1a) Conduct the proposed experiment with the handedness manipulation (25 subjects), where we will put our proposed neural correlate of τ_m to a critical test.

(1b) Conduct the proposed experiment with the perceptual noise manipulation (25 subjects), where we will put our proposed neural correlate of τ_e to a critical test.

(1c) Use the newly collected data to fit a model in which the successful neural correlates are used to enable individual estimation of the τ_e and τ_m parameters and test the additivity assumption.

(1d) Explore the newly collected data to identify new neural correlates of drift rate δ, drift coefficient ς^2, and boundary separation α.

In the second year, we will:

(2a) Conduct the proposed experiment with the difficulty manipulation (25 subjects), where we will put our proposed neural correlate of δ to a critical test.

(2b) Conduct the proposed experiment with a speed/accuracy instruction (25 subjects), where we will put our proposed neural correlate of α to a critical test.

(2c) Conduct an individual-differences (correlational) analysis to test the neural correlate of ς^2.

(2d) Use the newly collected data to fit a model in which the successful neural correlates are used to enable individual estimation of the δ, ς^2, and α parameters.

Throughout, we will take time to (1) preregister our confirmatory analyses and (2) make data, code, and article preprints available, all on the Open Science Framework.

Broader impact

The success of the proposed project will have broad repercussions and will help shape the new field of neurocognitive modeling. A more powerful joint-modeling technique will allow for better aggregation of multiple modes of data and provide a more accurate view on the effect of experimental manipulations, interventions, and treatments.

The proposed approach has the novel potential of making quantitative predictions about brain activity from behavioral data. This is novel because most—if not all—neuroimaging experiments are exploratory rather than confirmatory. Our new framework will allow confirmatory methods to take hold in brain research. When applied to clinical populations, behavioral measurements could potentially be used to make predictions about brain deficits.

More broadly, our methods will not be limited to the specific cognitive model (the decision-diffusion model) nor will they be limited to the EEG data we will collect. Our modeling strategy, which borrows heavily from the statistical literature on latent variable modeling, will be generic so that fMRI or single-cell data may be included instead of or in addition to EEG data.

As part of a commitment to allow others to maximally benefit from this project, the new method will be actively disseminated through publications, conferences, workshops, teaching, software, and tutorials. Furthermore, all data and software created will be made freely available online. By making the method accessible with tutorials and freely available software and example data sets, the proposed project will enable all researchers to add joint modeling to their analytic toolkit.

The project will involve junior and senior research associates who will receive training and start a career in neuroscience or cognitive science.

Results from prior NSF support

PI Vandekerckhove was previously awarded grant #1230118 from the National Science Foundation's Methods, Measurements, and Statistics panel. The $260,000.00 grant, titled "Cognitive Structural Equation Models" spans a 36-month period starting in September 2012.

The project is completed and all goals have been reached, although further work continues. The project goals were to (1) establish a formal modeling framework for the joint modeling of multiple modes of data, such as might arise from an experiment involving multiple tasks, (2) provide software for the implementation of such models, and (3) disseminate information about the new framework through theoretical and applied papers and presentations at conferences.

Intellectual merit

The proposed modeling framework, including methods of statistical inference, was described in detail in Vandekerckhove (2014). This paper also included an application to an executive functioning data set, in which we were able to infer a latent measure of dysphoria that jointly explained behavior in a proactive interference task and in personality surveys. Various incarnations of the modeling framework are applied in M. D. Nunez et al. (2015), M. D. Nunez, Vandekerckhove, and Srinivasan (2016), Murphy, Vandekerckhove, and Nieuwenhuis (2014), Wiech et al. (2014), Salum et al. (2014a, 2014b), Oravecz, Vandekerckhove, and Batchelder (2014), and most recently in Kupitz et al. (2015) and Guan, Lee, and Vandekerckhove (2015).

At the same time, we have published several papers on the technical issues involved in fitting complex cognitive models, such as model selection (Vandekerckhove, Matzke, & Wagenmakers, in press), tutorials on the implementation of cognitive models in general-purpose statistical software, and efficient computation (Wabersich & Vandekerckhove, 2014a, 2014b).

Finally, we have worked on broader related issues of cognitive modeling (Lee, Newell, & Vandekerckhove, 2014; Zhang, Lee, Vandekerckhove, Maris, & Wagenmakers, 2014), for a total of 15 papers resulting from the project. Further publications involving transfer of training, the latent structure of attention, and the joint modeling of behavioral and neural data are in preparation.

Broader impacts

The impact of the project has been substantial. Cognitive latent variable modeling is now the basis of several further collaborations that are planned (including with M. D. Lee of UCI, R. Srinivasan of UCI, S. Jaeggi and M. Bushkuehl of UCI, and P. Kuppens and M. Pe of the University of Leuven). It has also formed the basis of the project of five graduate students (M. D. Nunez, M. Guan, I. Danileiko, B. Baribault, and C. Kupitz) at UCI, several of whom were able to attend conferences, workshops, or summer schools to great benefit. The *Cognition and Individual Differences* lab has more than doubled in size and was able to greatly increase its output thanks to NSF support. The current proposal partly results from this work.

Part of the supported output (and in line with the goals of the currently proposed project) was the creation and publication of free and open-source software for the implementation of the new models. We have published software for the implementation of cognitive models in JAGS and R (Wabersich & Vandekerckhove, 2013a, 2013b, 2013c, 2013d, we have done the same in Stan, but major updates to Stan made our plug-ins defunct and a collaborative implementation with the Stan development team is pending) as well as software to facilitate the implementation of complex Bayesian models (Vandekerckhove, 2015). Several of these packages are now used in teaching, and we have used them to teach cognitive latent variable modeling in workshops and summer schools. A tutorial paper on implementing CogSEMs using these tools is in progress (Baribault & Vandekerckhove, in preparation).

References

Baribault, B., & Vandekerckhove, J. (in preparation). A tutorial on cognitive latent variable models.

Buzsaki, G. (2006). *Rhythms of the brain*. Oxford University Press.

Donders, F. C. (1868). Over de snelheid van psychische processen. *Onderzoekingen gedaan in het Physiologisch Laboratorium der Utrechtsche Hoogeschool (1968–1869)*, *2*, 92–120.

Dosher, B. A., & Lu, Z.-L. (2000). Noise exclusion in spatial attention. *Psychological Science*, *11*(2), 139–146.

Ergenoglu, T., Demiralp, T., Bayraktaroglu, Z., Ergen, M., Beydagi, H., & Uresin, Y. (2004). Alpha rhythm of the EEG modulates visual detection performance in humans. , *20*(3), 376–383. (00240) doi: 10.1016/j.cogbrainres.2004.03.009

Frank, M. J., Gagne, C., Nyhus, E., Masters, S., Wiecki, T. V., Cavanagh, J. F., & Badre, D. (2015). fMRI and EEG predictors of dynamic decision parameters during human reinforcement learning. , *35*(2), 485–494. doi: 10.1523/JNEUROSCI.2036-14.2015

Geddes, J., Ratcliff, R., Allerhand, M., Childers, R., Wright, R. J., Frier, B. M., & Deary, I. J. (2010). Modeling the effects of hypoglycemia on a two–choice task in adult humans. *Neuropsychology*, *24*, 652–660.

Gold, J. I., & Shadlen, M. N. (2007). The neural basis of decision making. *Annual Review of Neuroscience*, *30*(1), 535–574. doi: 10.1146/annurev.neuro.29.051605.113038

Guan, M., Lee, M. D., & Vandekerckhove, J. (2015). A hierarchical cognitive threshold model of human decision making on different length optimal stopping problems. In R. Dale et al. (Eds.), *Proceedings of the 37^{th} Annual Conference of the Cognitive Science Society.* Austin, TX: Cognitive Science Society.

Hajcak, G., Moser, J. S., Holroyd, C. B., & Simons, R. F. (2006). The feedback-related negativity reflects the binary evaluation of good versus bad outcomes. *Biological psychology*, *71*(2), 148–154.

Klauer, K. C., Voss, A., Schmitz, F., & Teige-Mocigemba, S. (2007). Process components of the implicit association test: A diffusion–model analysis. *Journal of Personality and Social Psychology*, *93*, 353–368.

Krusienski, D. J., Sellers, E. W., McFarland, D. J., Vaughan, T. M., & Wolpaw, J. R. (2008). Toward enhanced p300 speller performance. *Journal of neuroscience methods*, *167*(1), 15–21.

Kupitz, C., Buschkuehl, M., Jaeggi, S., Jonides, J., Shah, P., & Vandekerckhove, J. (2015). A diffusion model account of the transfer-of-training effect. In R. Dale et al. (Eds.), *Proceedings of the 37^{th} Annual Conference of the Cognitive Science Society.* Austin, TX: Cognitive Science Society.

Lee, M. D., Newell, B., & Vandekerckhove, J. (2014). Modeling the adaptation of search termination in human decision making. *Decision*, *1*, 223–251.

Link, S. W. (1975). The relative judgement theory of two choice response time. *Journal of Mathematical Psychology*, *12*, 114–135.

Loughnane, G. M., Newman, D. P., Bellgrove, M. A., Lalor, E. C., Kelly, S. P., & O'Connell, R. G. (2016). Target selection signals influence perceptual decisions by modulating the onset and rate of evidence accumulation. *Current Biology*, *26*(4), 496–502.

Lu, Z.-L., & Dosher, B. A. (1998). External noise distinguishes attention mechanisms. *Vision research*, *38*(9), 1183–1198.

Luck, S. J., Woodman, G. F., & Vogel, E. K. (2000). Event-related potential studies of attention. , *4*(11), 432–440. (00632) doi: 10.1016/S1364-6613(00)01545-X

Makeig, S., Bell, A. J., Jung, T.-P., Sejnowski, T. J., et al. (1996). Independent component analysis of electroencephalographic data. *Advances in neural information processing systems*, 145–151.

McFarland, D. J., Miner, L. A., Vaughan, T. M., & Wolpaw, J. R. (2000). Mu and beta rhythm topographies during motor imagery and actual movements. *Brain Topography*, *12*(3), 177–186. doi: 10.1023/A:1023437823106

Meister, M. L., Hennig, J. A., & Huk, A. C. (2013). Signal multiplexing and single-neuron computations in lateral intraparietal area during decision-making. *The Journal of Neuroscience*, *33*(6), 2254–2267.

Mitchell, J. F., Sundberg, K. A., & Reynolds, J. H. (2007). Differential attention-dependent response modulation across cell classes in macaque visual area v4. , *55*(1), 131–141. (00355) doi: 10.1016/j.neuron.2007.06.018

Mitchell, J. F., Sundberg, K. A., & Reynolds, J. H. (2009). Spatial attention decorrelates intrinsic activity fluctuations in macaque area v4. , *63*(6), 879–888. (00284) doi: 10.1016/j.neuron.2009.09.013

Mulder, M. J., Wagenmakers, E.-J., Ratcliff, R., Boekel, W., & Forstmann, B. U. (2012). Bias in the brain: A diffusion model analysis of prior probability and potential payoff. *Journal of Neuroscience*, *32*, 2335–2343.

Murphy, P. R., Vandekerckhove, J., & Nieuwenhuis, S. (2014). Pupil-linked arousal determines variability in perceptual decision making. *PLoS Computational Biology*, *10*, e1003854.

Norcia, A. M., Appelbaum, L. G., Ales, J. M., Cottereau, B. R., & Rossion, B. (2015). The steady-state visual evoked potential in vision research: a review. *Journal of vision*, *15*(6), 4–4.

Nunez, M. D., Nunez, P. L., & Srinivasan, R. (2016). Electroencephalography (EEG): neurophysics, experimental methods, and signal processing. In H. Ombao, M. Lindquist, W. Thompson, & J. Aston (Eds.), *Handbook of statistical methods for brain signals and images* (pp. 175–200). CRC Press.

Nunez, M. D., Srinivasan, R., & Vandekerckhove, J. (2015). Individual differences in attention influence perceptual decision making. *Frontiers in Psychology*, *6*(18), 18.

Nunez, M. D., Vandekerckhove, J., & Srinivasan, R. (2016). How attention influences perceptual decision making: Single-trial EEG correlates of drift-diffusion model parameters. *Journal of Mathematical Psychology*. doi: 10.1016/j.jmp.2016.03.003

Nunez, P. L., & Srinivasan, R. (2006). *Electric fields of the brain: The neurophysics of EEG*. Oxford University Press.

O'Connell, R. G., Dockree, P. M., & Kelly, S. P. (2012). A supramodal accumulation-to-bound signal that determines perceptual decisions in humans. *Nature neuroscience*, *15*(12), 1729–1735.

Oravecz, Z., Vandekerckhove, J., & Batchelder, W. H. (2014). Bayesian cultural consensus theory. *Field Methods*, *26*(3), 207–222.

Parra, L. C., Spence, C. D., Gerson, A. D., & Sajda, P. (2005). Recipes for the linear analysis of EEG. , *28*(2), 326–341. doi: 10.1016/j.neuroimage.2005.05.032

Pernet, C. R., Sajda, P., & Rousselet, G. A. (2011). Single-trial analyses: why bother? *Frontiers in psychology*, *2*, 322.

Pfurtscheller, G., Brunner, C., Schlögl, A., & Lopes da Silva, F. H. (2006). Mu rhythm (de)synchronization and EEG single-trial classification of different motor imagery tasks. *NeuroImage*, *31*(1), 153–159. doi: 10.1016/j.neuroimage.2005.12.003

Philiastides, M. G., Heekeren, H. R., & Sajda, P. (2014). Human scalp potentials reflect a mixture of decision-related signals during perceptual choices. *The Journal of Neuroscience*, *34*(50), 16877–16889.

Philiastides, M. G., Ratcliff, R., & Sajda, P. (2006). Neural representation of task difficulty and decision–making during perceptual categorization: A timing diagram. *Journal of Neuroscience*, *26*, 8965–8975.

Ratcliff, R. (1978). A theory of memory retrieval. *Psychological Review*, *85*, 59–108.

Ratcliff, R., Gomez, P., & McKoon, G. (2004). Diffusion model account of lexical decision. *Psychological Review*, *111*, 159–182.

Ratcliff, R., Hasegawa, Y. T., Hasegawa, Y. P., Smith, P. L., & Segraves, M. A. (2007). Dual diffusion model for single–cell recording data from the superior colliculus in a brightness–discrimination task. *Journal of Neurophysiology*, *97*, 1756–1774.

Ratcliff, R., & McKoon, G. (2008). The diffusion decision model: Theory and data for two–choice decision tasks. *Neural Computation*, *20*, 873–922.

Ratcliff, R., Thapar, A., & McKoon, G. (2006). Aging, practice, and perceptual tasks: A diffusion model analysis. *Psychology and Aging*, *21*, 353–371.

Ratcliff, R., Thapar, A., & McKoon, G. (2010). Individual differences, aging, and IQ in two–choice tasks. *Cognitive Psychology*, *60*, 127–157.

Ratcliff, R., & van Dongen, H. P. A. (2009). Sleep deprivation affects multiple distinct cognitive processes. *Psychonomic Bulletin & Review*, *16*, 742–751.

Salum, G. A., Sergeant, J., Sonuga-Barke, E., Vandekerckhove, J., Gadelha, A., Pan, P. M., ... Rohde, L. A. P. (2014a). Mechanisms underpinning inattention and hyperactivity: Neurocognitive support for ADHD dimensionality. *Psychological Medicine*, *44*, 3189–3201.

Salum, G. A., Sergeant, J., Sonuga-Barke, E., Vandekerckhove, J., Gadelha, A., Pan, P. M., ... Rohde, L. A. P. (2014b). Specificity of basic information processing and inhibitory control in attention deficit/hyperactivity disorder. *Psychological Medicine*, *44*, 617–631.

Seitz, A. R., Kim, D., & Watanabe, T. (2009). Rewards evoke learning of unconsciously processed visual stimuli in adult humans. , *61*(5), 700–707. doi: 10.1016/j.neuron.2009.01.016

Shadlen, M. N., & Kiani, R. (2013). Decision making as a window on cognition. , *80*(3), 791–806. doi: 10.1016/j.neuron.2013.10.047

Shadlen, M. N., & Newsome, W. T. (2001). Neural basis of a perceptual decision in the parietal cortex (area LIP) of the rhesus monkey. *Journal of Neurophysiology*, *86*, 1916–1936.

Tong, F., & Pratte, M. S. (2012). Decoding patterns of human brain activity. *Annual review of psychology*, *63*, 483–509.

Tuerlinckx, F. (2004). The efficient computation of the cumulative distribution and probability density functions in the diffusion model. *Behavior Research Methods, Instruments, & Computers*, *36*, 702–716.

Turner, B. M., Forstmann, B. U., Love, B. C., Palmeri, T. J., & Van Maanen, L. (2016). Approaches to analysis in model-based cognitive neuroscience. *Journal of Mathematical Psychology*. doi: 10.1016/j.jmp.2016.01.001

Turner, B. M., Forstmann, B. U., Wagenmakers, E.-J., Brown, S. D., Sederberg, P. B., & Steyvers, M. (2013). A Bayesian framework for simultaneously modeling neural and behavioral data. *NeuroImage*, *72*, 193–206.

Twomey, D. M., Murphy, P. R., Kelly, S. P., & O'Connell, R. G. (2015). The classic p300 encodes a build-to-threshold decision variable. , *42*(1), 1636–1643. doi: 10.1111/ejn.12936

van Ravenzwaaij, D., Dutilh, G., & Wagenmakers, E.-J. (2012). A diffusion model decomposition of the effects of alcohol on perceptual decision making. *Psychopharmacology*, *219*, 1017–2025.

Vandekerckhove, J. (2014). A cognitive latent variable model for the simultaneous analysis of behavioral and personality data. *Journal of Mathematical Psychology*, *60*, 58–71.

Vandekerckhove, J. (2015). Trinity: A MATLAB interface for WinBUGS, JAGS, and Stan [Computer software manual].

Vandekerckhove, J., Matzke, D., & Wagenmakers, E.-J. (in press). Model comparison and the principle of parsimony. *Oxford Handbook of Computational and Mathematical Psychology*.

van Vugt, M. K., Simen, P., Nystrom, L. E., Holmes, P., & Cohen, J. D. (2012). EEG oscillations reveal neural correlates of evidence accumulation. , *6*, 106. (00032) doi: 10.3389/fnins.2012.00106

Voss, A., Rothermund, K., & Voss, J. (2004). Interpreting the parameters of the diffusion model: An empirical validation. *Memory & Cognition*, *32*, 1206–1220.

Wabersich, D., & Vandekerckhove, J. (2013a). jags-alcove: an ALCOVE plugin for JAGS [Computer software manual]. Retrieved from `http://sourceforge.net/projects/jags-alcove/`

Wabersich, D., & Vandekerckhove, J. (2013b). jags-wiener: a diffusion model plugin for JAGS [Computer software manual]. Retrieved from `http://sourceforge.net/projects/jags-wiener/`

Wabersich, D., & Vandekerckhove, J. (2013c). RAlcove: R functions for ALCOVE [Computer software manual]. Retrieved from `http://sourceforge.net/projects/ralcove/`

Wabersich, D., & Vandekerckhove, J. (2013d). Rwiener: R functions for the diffusion model [Computer software manual]. Retrieved from `http://sourceforge.net/projects/rwiener/`

Wabersich, D., & Vandekerckhove, J. (2014a). Extending JAGS: A tutorial on adding custom distributions to JAGS (with a diffusion model example). *Behavior Research Methods*, *46*, 15–28.

Wabersich, D., & Vandekerckhove, J. (2014b). The RWiener package: an R package providing distribution functions for the Wiener diffusion model. *The R Journal*, *6*(1), 49–56.

Wagenmakers, E.-J., Ratcliff, R., Gomez, P., & McKoon, G. (2008). A diffusion model account of criterion shifts in the lexical decision task. *Journal of Memory and Language*, *58*, 140–159.

Wiech, K., Vandekerckhove, J., Zaman, J., Tuerlinckx, F., Vlaeyen, J. W. S., & Tracey, I. (2014). Influence of prior information on pain involves biased perceptual decision–making. *Current Biology*, *24*, R679–R681.

Zhang, S., Lee, M. D., Vandekerckhove, J., Maris, G., & Wagenmakers, E.-J. (2014). Time-varying boundaries for diffusion models of decision making and response time. *Frontiers in Psychology*, *5*(1364), 1364.