



Factor analysis linking functions for simultaneously modeling neural and behavioral data[☆]

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ABSTRACT

A growing number of researchers have advocated for the advancement of cognitive neuroscience by blending cognitive models with neurophysiology. The recently proposed joint modeling framework is one way to bridge the gap between the abstractions assumed by cognitive models and the neurophysiology obtained by modern methods in neuroscience. Despite this advancement, the current method for linking the two domains is hindered by the dimensionality of the neural data. In this article, we present a new linking function based on factor analysis that allows joint models to grow *linearly* in complexity with increases in the number of neural features. The new linking function is then evaluated in two simulation studies. The first simulation study shows how the model parameters can be accurately recovered when there are many neural features, that mimics real-world applications. The second simulation shows how the new linking function can (1) properly recover a representation of the data generating model, even in the case of model misspecification, and (2) outperform the previous linking function in a cross-validation test. We close by applying a model equipped with the new linking function to real-world data from a perceptual decision making task. The model allows us to understand how differences in the model parameters emerge as a function of differences in brain function across speed and accuracy instruction.

Introduction

The field of cognitive science is faced with many options for studying how experimentally-derived variables are systematically related to the dynamics underlying a cognitive process of interest. To date, much of our understanding of cognition has been advanced by two dominant, but non-interacting groups. The largest group, cognitive neuroscientists, rely on statistical models to understand patterns of neural activity. These models are typically purely data-mining techniques, and often disregard the computational mechanisms that might detail a cognitive process. The other group, mathematical psychologists, is strongly motivated by *theoretical* accounts of cognitive processes, and instantiates these theories by developing formal mathematical models of cognition. The models often assume a system of computations and equations intended to characterize the process assumed to take place in the brain. To formally test their theory, mathematical psychologists rely on their model's ability to fit behavioral data. A good fit is thought to reflect an accurate theory, whereas a bad fit would refute it.

Although both groups are concerned with explaining how the mind

gives rise to behavior, they tend to approach this problem from two different perspectives. Thinking in terms of Marr (1982)'s levels of analysis, mathematical psychologists tend to focus on the computational and algorithmic levels by developing theories about how the mind works and instantiating these theories with a cognitive model. Typically, the model possesses a set of statistical or mathematical mechanisms controlled by a set of parameters. The process of fitting a model to data produces estimates of these parameters, and these estimates are then used to articulate the cognitive processes at work across conditions, subjects, or even groups. On the other hand, cognitive neuroscientists focus more on the implementation level by observing how changes in the independent variable of an experiment give rise to changes in a neural measure of interest. For example, this approach might correlate the speed of the observed response times with the measured activations in the brain. While this experimental approach has served as the cornerstone for major scientific findings regarding the localization of function, it has been criticized for contributing little to our theoretical understanding of how the mind works (Coltheart, 2006). For example, in relating brain measure to response time, we can say *which* brain areas correlate with the speed of

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the decision, but we cannot say anything about *why* because these analyses are not based on mechanisms of a cognitive model. As one example, such an analysis would not provide insight into whether a fast decision arose due to a particularly easy stimulus or due to a quick guess elicited by the subject.

Our own view is that while progress can be made by maintaining a tight focus on one level, certain opportunities are missed. As a result of their single-level focus, both approaches suffer from critical limitations (Love, 2015), and these limitations have inspired researchers to combine neural and behavioral measures in an integrative fashion. The importance of solving the integration problem has spawned several entirely new statistical modeling approaches (cf. Turner et al., 2016a; de Hollander et al., 2016) developed through collaborations between mathematical psychologists and cognitive neuroscientists, collectively forming a new field often referred to as “model-based cognitive neuroscience” (e.g., Forstmann and Wagenmakers, 2015; O’Doherty et al., 2007, 2003; Forstmann et al., 2011; van Maanen et al., 2011; Turner et al., 2013a; Mack et al., 2013; Boehm et al., 2014; Love, 2015; Palmeri et al., 2015; Turner et al., 2015; Anderson, 2007; Anderson et al., 2008a, 2008b; Borst et al., 2010; Cassey et al., 2017). The field of model-based cognitive neuroscience balances the theoretical focus of computational models from mathematical psychology with the localization focus of neuroimaging by seeking out areas of the brain that correspond directly to mechanisms in the model, rather than experimental variables. This mechanistic focus provides an interesting opportunity for establishing a common theoretical framework for the mind, enabling mass aggregation across the entire field of cognitive neuroscience (cf. Turner et al., 2016b; Love, 2015, 2016).

Joint modeling framework

One approach for performing analyses in model-based cognitive neuroscience is the recently developed joint modeling framework (Turner et al., 2013a, 2015, 2016b; Turner, 2015; Cassey et al., 2017). The joint modeling framework has been used to apply constraints to evidence accumulation models derived from structural properties of the brain (Turner et al., 2013a; Turner, 2015), functional magnetic resonance imaging (fMRI; Turner et al., 2015), and electroencephalography (EEG; Turner et al., 2016b). More recently, Turner et al. (2016b) extended the framework to propose an alternative approach for “data fusion”, where patterns in the fMRI, EEG, and behavioral measures are jointly modeled to gain better insight into the cognitive process at hand (Love, 2016).

To briefly summarize our approach, a typical joint model consists of three components. First, the behavioral data B are described in terms of a behavioral model, such as the classic signal detection theory model (Green and Swets, 1966). For example, the behavioral data could consist of response choices, response times, or even confidence judgments. Importantly, the behavioral model consists of a set of model parameters θ , which are of lower dimensionality than the full behavioral data set B . For example, in the SDT model, θ would consist of parameters such as discriminability (d') and bias (β). Ideally, the chosen behavioral model should consist of mechanisms that provide insight into how the data arise, and how experimental manipulations predictably affect these mechanisms (a property known as selective influence; Dzhaferov, 2003; Heathcote et al., 2015). In this way, the parameters of the model are connected to the experimental manipulations akin to the strategy prevalent in cognitive neuroscience as discussed above. Second, the neural data N are described in terms of a neural model. For example, the neural data might consist of the blood oxygenated level dependence (BOLD) response across time for a set of voxels, or even changes in the EEG measures across time for a set of electrodes. The key property of the neural model is that it should consist of a set of parameters δ that describe the important parts of the neural data N in a way that is of significantly lower dimensionality. For example, the neural model could describe increases in neural activity

across time through a general linear model (GLM). Alternatively, the neural model could also be of a more mechanistic nature, describing the computations purportedly implemented by a brain region, such as those seen in topographic latent source analysis (Gershman et al., 2011; Manning et al., 2014). Third, the features of both the neural and behavioral data are linked by establishing an explicit relationship between the behavioral model parameters θ and the neural model parameters δ .

The focus of the current article centers directly on the third component of the joint modeling framework. In Turner et al. (2013a), we generically assumed a linking distribution \mathcal{M} conjoined θ and δ such that

$$(\theta_j, \delta_j) \sim \mathcal{M}(\Omega),$$

where θ_j and δ_j denote the behavioral and neural model parameters for the j th subject or even j th trial (Turner et al., 2015) respectively, and Ω consists of a set of hyperparameters governing the linking function. Here, the term “hyperparameters” refers to parameters in the “hyper level”, which is one level higher than the level that connects to the data. There are many types of linking functions one could use. For example, one could simply regress the parameters θ and δ against one another such that

$$\theta_j = \delta_j \beta_1 + \beta_0 + \epsilon, \text{ and } \epsilon \sim \mathcal{N}(0, \sigma),$$

where $\mathcal{N}(a, b)$ denotes a normal distribution with mean a and standard deviation b . Here, the parameters of the linking function are $\Omega = \{\beta_0, \beta_1, \sigma\}$. Recently, this approach has been used to link decision models to fluctuations in neural activity. For example, Nunez et al. (2015) used EEG data on a perceptual decision making experiment as a proxy for attention. They controlled the rate of flickering stimuli presented to subjects to match the sampling rate of their EEG data, a measure known as the steady-state visual evoked potential. Importantly, Nunez et al. (2015) showed that individual differences in attention or noise suppression was indicative of the choice behavior, specifically it resulted in faster responses with higher accuracy. In a particularly novel application, Frank et al. (2015) showed how models of reinforcement learning could be fused with the DDM to gain insight into activity in the subthalamic nucleus (STN). In their study, Frank et al. (2015) used simultaneous EEG and fMRI measures as a covariate in the estimation of single-trial parameters. Specifically, they used pre-defined regions of interest including the presupplementary motor area, STN, and a general measure of mid-frontal EEG theta power to constrain trial-to-trial fluctuations in response threshold, and BOLD activity in the caudate to constrain trial-to-trial fluctuations in evidence accumulation. Their work is important because it establishes concrete links between STN and pre-SMA communication as a function of varying reward structure, as well as a model that uses fluctuations in decision conflict (as measured by multimodal activity in the dorsomedial frontal cortex) to adjust response threshold from trial-to-trial.

The regressive linking function between θ and δ captures the basic intuition behind how the neural data are related to the behavioral model through the regression parameters. However, the most basic version of this approach does neglect the multivariate nature of the problem, and often neglects the measurement error present in the neural data. Typically, we are interested in understanding how mechanisms in our model are related to many brain areas at once, and we would like to rule out brain areas that are correlated with our decision model by virtue of being correlated either functionally or structurally with other brain areas. This problem, known as multicollinearity, can distort our interpretation of the function of these brain areas simply because our model has assumed that the candidate brain areas are unrelated to one another.

Another approach is to assume that θ and δ are linked via a multivariate normal (MVN) distribution (Turner et al., 2013a, 2015, 2016b; Turner, 2015). Formally,

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