

Contradictory “heuristic” theories of autism spectrum disorders: The case for theoretical precision using computational models

Autism

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Davis and Plaisted-Grant (2014) argue persuasively that *reduced* neural noise is consistent with most behavioral and physiological differences between autism spectrum disorder (ASD) and non-ASD individuals. This is surprising given that the same empirical results were previously used to support the opposite point of view—*increased* neural noise is the primary cause of ASD (Rubenstein and Merzenich, 2003). When two incompatible theories explain the same results, the field needs to revisit its framework for defining and evaluating theories. Formalizing theories using computational models can clarify a theory’s predictions and distinguish it from theories of related disorders (e.g. Sikström and Söderlund, 2007, who instantiated a formal model of hypersensitivity in individuals with attention deficit hyperactivity disorder as reduced neural noise).

Davis and Plaisted-Grant provide a valuable distinction between internal and external noise in theories of ASD. However, it is unclear how much more progress can be made from heuristic theories. They make clear, interesting predictions for a homunculus acting based on a single noisy neuron (e.g. cases where motion coherence should be intact for ASD individuals), yet it is unclear how these heuristic theories scale beyond a single neuron. For example, how does having less (or more) neural noise at *every* level of a hierarchy of neurons influence behavior? If only a subset of neurons has less (or more) neural noise, how should that influence behavior?

As a step toward resolving the current disarray of results and theories, I briefly motivate and sketch a framework for developing computational models of ASD integrated over levels of analysis. Formalizing theories is not merely a mathematical exercise; the resulting models explain counterintuitive behaviors that are difficult to understand from introspection. For example, computational formalizations of Parkinson’s disease explain a previously inexplicable result: dopamine simultaneously impairs *and* aids learning in Parkinson’s patients (Frank et al., 2004).

Although multiple computational formalisms should be explored simultaneously, the Bayesian framework is

particularly well-suited for formalizing ASD theories (Pellicano and Burr, 2012). It is based on Bayes’ rule, which describes how agents should update their prior belief about something after observing evidence. Previously, Pellicano and Burr (2012) argued that Bayesian models support a top-down ASD theory (weaker expectations), rather than a bottom-up theory (enhanced senses). However, Bayesian models are a framework or language for formalizing and evaluating theories. Thus, the Bayesian framework is equally compatible with top-down and bottom-up theories. Top-down theories predict ASD and non-ASD individuals have different priors, whereas bottom-up theories predict different likelihoods. If differences between ASD and non-ASD individuals can only be captured by having top-down (different priors) *and* bottom-up (different likelihoods) for each group, then the resulting model would in fact be a combination of both theories. Discerning the relative influence of top-down and bottom-up contributions is advantageous because many researchers believe that only a combination of top-down and bottom-up theories can explain the many differences between ASD and non-ASD individuals.

Once researchers formalize theories in formal models, the models can be used to design experiments that test the relevant components of the theories. For most Bayesian models, the influence of the prior and likelihood can be dissociated using specially designed behavioral experiments. Intuitively, this is because within a given trial, the likelihood term applies multiple times (once for each observation in the trial), whereas the prior only applies once (regardless of the number of observations). In fact, this is precisely why Bayesian models have been so influential over the last decade: They provide a principled framework for identifying

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the differential contributions of prior knowledge and current evidence. Furthermore, the Bayesian framework can unite multiple levels of analysis and domains from the neural level to the behavioral level (e.g. Friston, 2010), including perception, cognition, and even complex social tasks.

One may be skeptical that this flexibility makes the Bayesian framework unfalsifiable. This worry is misplaced because, like any computational formalism, the Bayesian framework is a *language* for constructing models. For example, the decreased and increased neural noise hypotheses can be formulated as either top-down or bottom-up theories by changing the noise in the prior or likelihood. How decreased or increased noise in specific neural areas affects behavior can be understood by decreasing or increasing the noise of the corresponding portions of a hierarchical Bayesian model and observing the resulting model's behavior. These models (and therefore, theories) are falsifiable, and the ultimate test of whether the Bayesian framework is useful for formalizing ASD theories depends on whether the differences between ASD and non-ASD individuals can be captured by a simple Bayesian model, rather than one containing many extraneous components.

As Davis and Plaisted-Grant (2014) write, the “primary utility [of verbal theories] is to motivate psychological and physiological experiments,” and I agree that they have sketched an interesting path for future research. Although verbal theorizing is valuable, future work must delve

deeper by building and formalizing introspective theories using computational models.

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