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On hyperpriors and hypopriors: comment on Pellicano and Burr

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Pellicano and Burr [1] present a compelling explanation for the perceptual symptoms of autism in terms of a failure of Bayesian inference. In this letter, we nuance a few observations relating to the nature of their normative explanation. This leads to the interesting suggestion that autism may be a disorder of metacognition.

Normative models – such as the Bayesian brain hypothesis – furnish descriptions of perception or behaviour in terms of optimising something; for example, Bayesian model evidence in the case of the Bayesian brain. The idea presented in [1] is that prior beliefs, which generate top-down predictions, are somehow compromised, leading to increased reliance on bottom-up sensory evidence. To understand the mechanistic implications of this normative explanation, one has to posit a neuronal implementation of Bayesian inference.

The most popular implementation of Bayesian inference is predictive coding [2–5]. In this framework, the influence of prior beliefs, relative to sensory evidence, is controlled by the precision (reliability or confidence) of predictions at higher levels of a hierarchical model relative to sensory precision [5]. The precision controls the width of the likelihood of any sensory input (and priors) in exactly the way described in [1]. Statistically speaking, the brain has to estimate precision (cf., expected uncertainty [6]) at each level of the sensorimotor hierarchy. The parameters of these different levels of precision are known as 'hyperparameters' and prior beliefs about hyperparameters are called 'hyperpriors' ([5], p. 13). In this context, hyperpriors do not mean an inflation of priors, but rather prior beliefs about hyperparameters: in this particular instance, prior beliefs about the precision of beliefs about the state of the world. We make this point because the term 'hypoprior' introduced in [1] may not be entirely appropriate in the context of hierarchical inference in the brain.

In predictive coding, precision is thought to be encoded by the postsynaptic gain of superficial pyramidal cells encoding prediction error [5,7]. This gain corresponds exactly to the synaptic efficacy that underlies the adaptive changes in connection strengths discussed in [1]. The link between precision in hierarchical inference and synaptic gain provides a graceful and physiologically plausible link between aberrant perception in autism [1] (and schizophrenia [8]) and functional disconnection mediated by neuromodulatory abnormalities [9]. Anchoring the normative Bayesian explanation to its neuronal instantiation may be important for empirical studies of autism – when making predictions about psychophysical and electrophysiological responses. For example, it is now possible to simulate event-related potentials under different levels of precision (see [7] for an example in the context of biased competition and the Posner paradigm). Furthermore, it may be necessary to ground therapeutic interventions in an understanding of the underlying pathophysiology.

In summary, the lack of central coherence seen in autism [10] can, in a normative sense, be attributed to attenuated estimates of precision (or hyperpriors) at higher (central) levels of hierarchical models in the brain. Crucially, this means that the abnormality – from a psychological perspective – is not a failure of prediction *per se*, but a failure to instantiate top-down predictions during perceptual synthesis because their precision is too low. In other words, there is a failure of beliefs (estimated precision) about beliefs (predictions) that is, formally, a failure of metacognition. At a physiological level, this failure may be intimately related to neuromodulatory control of the synaptic gain of superficial neurons at specific levels in the cortical hierarchy.

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