LETTER

A more precise look at context in autism

In a recent paper, Rosenberg et al. (1) present a compelling explanation for the perceptual symptoms of autism in terms of a failure of divisive normalization. In divisive normalization the output of individual neurons is scaled (or divided) by the combined activity of the neural population in which they are embedded, and thus local visual context provides a means of gain control—a volume dial—for stimulus-evoked responses. However, to properly understand the wider mechanistic implications, beyond local inhibition or gain in the visual cortex, one has to posit a biologically plausible instantiation of context-sensitive neural responses across multiple timescales and hierarchical levels that allows for the influence of attention and prior beliefs, which the authors so elegantly simulate.

Predictive coding frameworks for sensory perception propose that 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 (2). A neurobiological account of aberrant precision in autism has recently been proposed (3) in which sensory precision is too high relative to the precision of prior beliefs.

Precision-weighting and divisive normalization are both mechanisms that serve to contextualize neural responses. In fact, increased sensory precision could arise from weaker lateral inhibition or gain control in the visual cortex, exactly as formulated in Rosenberg et al. (1). Furthermore, even precise (or disinhibited) visual responses within a single level of the cortical hierarchy may still be down-weighted if the precision of predictions at a higher level (e.g., NMDA-mediated synaptic gain) mandates this. Top-down attenuation of sensory precision accounts for psychophysical and physiological aspects of attention (4), providing a hierarchically and biologically plausible account of the "attentional field" modeled in Rosenberg et al. (1). Finally, beyond a single sensory event, adaptive perception and action rests on the accurate estimation of fluctuations in the precision of environmental contingencies (5). Here, nonhierarchical neuromodulators (e.g., acetylcholine, norepinephrine) putatively track the volatility in the environment across time and this estimation globally scales the weight one should place on sensory evidence relative to prior beliefs.

In all three examples above, precision functions as a form of divisive normalization—where variance measured across a broad pool of perceptual regions and timescales acts as a denominator in a divisive computation—to scale or contextualize the driving neural responses to sensory input (not unlike dividing a difference in group means by its SE to form a *t*-statistic). Crucially, the operation of precision within the predictive coding framework furnishes divisive normalization with a biologically plausible role beyond excitation/inhibition balance in local cortical circuits. This role lies in the postsynaptic gain of superficial pyramidal cells encoding the prediction error within a cortical hierarchy and lies in the integrity of neuromodulatory function (2). Precision-weighting in autism could be aberrant in a number of ways, each resulting in context-insensitive perception and action (3); however, these are empirical questions that we look forward to seeing addressed with time.

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