

# Predictions, perception, and a sense of self

Fabienne Picard, MD  
Karl Friston, PhD

Correspondence to  
Dr. Picard:  
Fabienne.Picard@hcuge.ch

## ABSTRACT

In recent years there has been a paradigm shift in theoretical neuroscience in which the brain—as a passive processor of sensory information—is now considered an active organ of inference, generating predictions and hypotheses about the causes of its sensations. In this commentary, we try to convey the basic ideas behind this perspective, describe their neurophysiologic underpinnings, and highlight the potential importance of this formulation for clinical neuroscience. The formalism it provides—and the implementation of active inference in the brain—may have the potential to reveal aspects of functional neuroanatomy that are compromised in conditions ranging from Parkinson disease to schizophrenia. In particular, many neurologic and neuropsychiatric conditions may be understandable in terms of a failure to modulate the postsynaptic gain of neuronal populations reporting prediction errors during action and perception. From the perspective of the predictive brain, this represents a failure to encode the precision—or confidence in—sensory information. We propose that the predictive or inferential perspective on brain function offers novel insights into brain diseases. **Neurology® 2014;83:1112-1118**

Recent years have seen the accumulation of compelling evidence to suggest that the brain, instead of being a passive recipient of information from the external world (and body), actively predicts its sensory input. In this constructivist formulation, predictions are updated continuously through comparison with incoming sensations (see reference 1 for an accessible introduction). The underlying theory of predictive processing involves a cascade of recurrent neuronal processing in cortical hierarchies, with “top-down” *predictions* and “bottom-up” *prediction errors* being exchanged between successively higher levels of the cortical hierarchy. This neurobiological form of *predictive coding* is predicated on the notion of the brain as a Bayesian or inferential machine<sup>2,3</sup> that is trying to infer the causes of sensory input. Here, Bayesian inference simply refers to the updating of beliefs or expectations—encoded by the activity of specific neuronal populations—using sensory information. The expectations prior to observing sensory outcomes are updated into *posterior beliefs* after the information is available. In this commentary, we try to describe the essential properties of the Bayesian brain and highlight recent advances in understanding neuropsychiatric conditions in terms of false inference. Our account is necessarily brief but hopefully serves as an entree into this growing, and potentially interesting, body of literature.

Bayesian updating allows an optimal integration of sensory evidence to furnish optimal beliefs about the causes of our sensations.<sup>4</sup> In this setting, the function of the cortex is to predict the flow of sensory stimuli as accurately and as simply as possible. It follows that our image of the external world—and of ourselves—is as much constituted by these beliefs or expectations as by the incoming sensory signals per se. In other words, our expectations are the primary content of our percepts—they are “models” or “fantasies” generated by the brain to explain our sensations. Quite literally, the brain is a “fantastic” organ.

In this view, the brain is regarded as a predictive machine that continuously seeks to improve its beliefs or hypotheses through trial and error.<sup>2,3,5</sup> The “trials” here could correspond to sensory sampling, e.g., visual information sampled by saccadic eye movements. Inference about the

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From the Department of Neurology (F.P.), University Hospital and Medical School of Geneva, Switzerland; and Wellcome Trust Centre for Neuroimaging (K.F.), London, UK.

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causes of these sensory samples calls on the best prediction that higher-order cortical areas make about the next sample. These predictions are themselves selected from multiple possible predictions according to the preceding samples and beliefs about the prevailing context. This enables the brain to make real-time predictions in different sensory streams that it tests against the sensory data actually sampled. For example, if we consider the control of our eye movements during visual searches, this visual “palpation” has natural time constants that are relatively easy to simulate using predictive coding. Typically, we make saccadic movements every 250 ms,<sup>6</sup> during which time the evidence for hypotheses (about the causes of visual impressions) is accumulated. In these schemes, hypotheses compete with each other to specify the next point of fixation—that minimizes uncertainty about the winning hypothesis. Each successive eye movement is then enacted through oculomotor reflexes that fulfill descending predictions about the anticipated state of the oculomotor system. This is a particularly important example because eye movements are well studied, easy to measure, and show characteristic deficits in certain conditions (e.g., schizophrenia—see below).

**PREDICTIVE CODING IN THE BRAIN** In predictive processing, signals ascending from the sensory cortex (or subcortical structures such as the thalamus) are compared with descending predictions to form prediction errors. These prediction errors ascend to higher cortical regions to update posterior expectations and improve top-down predictions. In this scheme, the predictions are the stuff of perception, with the ascending prediction errors providing continuous feedback, which confirms or disconfirms the predictions or perceptual hypotheses.<sup>4,5,7</sup> The figure provides a schematic overview of predictive coding in the brain, with a special focus on the visual system and pontine oculomotor system. The ensuing view of perceptual synthesis suggests that neuronal message passing among different levels in cortical hierarchies comprises 2 streams: a descending stream of top-down predictions and an ascending stream of bottom-up prediction errors.

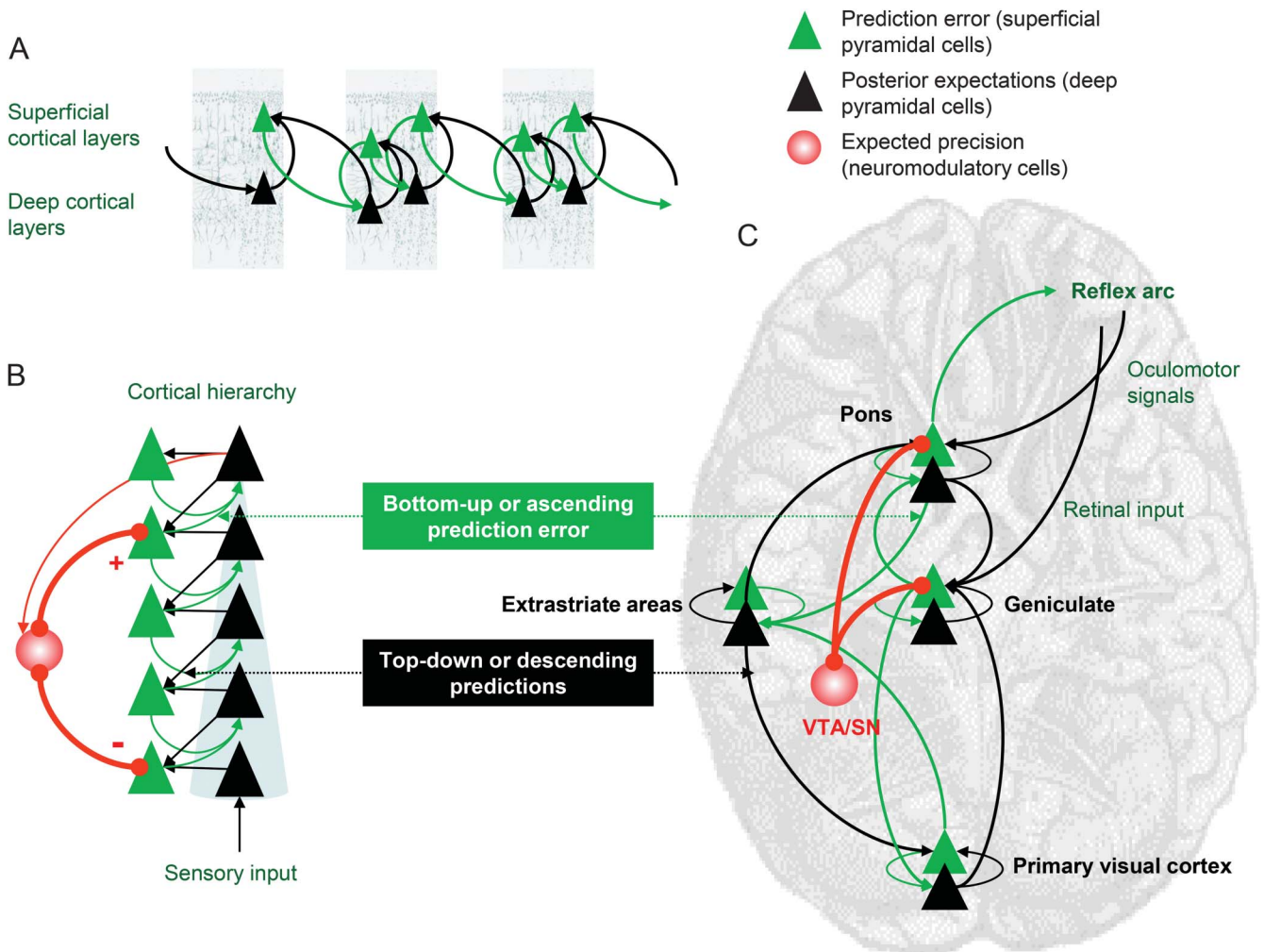
Perhaps the simplest example of prediction errors—and their resolution through hierarchical message passing—is the event-related potential in electrophysiology. This transient response to a change in sensory

stimulation is generated primarily by (superficial pyramidal) cells thought to be responsible for encoding prediction error. As the prediction error is resolved—through descending predictions that activate inhibitory interneurons—the response to (even sustained) input declines, leading to characteristic waveforms in the event-related potential. These dynamics can play out over several hundred milliseconds, where late components are usually associated with top-down (predictive) mechanisms. In neuronal implementations of predictive coding, the ascending (prediction error) and descending (prediction) streams are uniquely associated with particular populations in specific cortical laminae (see figure). The connectivity implicit in forming and accumulating prediction errors is remarkably consistent with known (canonical) microcircuitry in the brain.<sup>8–10</sup> Both streams thus depend on each other, and the brain is trying to minimize prediction errors at all levels of the cortical hierarchy. Minimizing hierarchical prediction errors provides an internally consistent explanation for how sensations are generated—at multiple levels of abstraction or description. Heuristically, when the internal processes generating predictions in the brain match the external processes generating sensations, prediction error is minimized. By minimizing prediction error, internal processes therefore emulate or represent external processes. Statistically speaking, this corresponds to Bayesian inference about how sensory data are generated. This hierarchical predictive coding is thought underlying perceptual synthesis and inference, but what about learning?

Accurate predictions suggest that cortical hierarchies embody a model of how sensations are produced or generated. This model is known as a *generative model* and has to be learned or acquired through experience. In other words, generative models have to be formed (via experience-dependent plasticity) to enable perceptual inference or synthesis.<sup>10</sup> This acquisition—through the associative synaptic plasticity of recurrent connections in cortical hierarchies—underlies perceptual learning. It is interesting that both perceptual inference and learning are driven by the same imperative—to minimize prediction errors. This ensures that the current prediction best matches the sensed outcome, facilitating rapid and efficient perceptual processing that needs only the sensory information that cannot be explained or predicted. This information is the ascending prediction error.

In vision, an object can be inferred before it is completely disclosed, given a sufficient matching between the prediction and early or partial visual cues.<sup>7</sup> Should matching fail, creating a prediction error, an improved or updated prediction—that produces a smaller prediction error—will emerge. But

**Figure** Hierarchical message passing in the brain



(A) This figure summarizes the architecture of neuronal message passing that underlies predictive coding in the brain.<sup>8,10</sup> The postulate is that neuronal activity encodes expectations about the causes of sensory input and these expectations attempt to minimize prediction error. Prediction error is simply the difference between (bottom-up) sensory input and (top-down) predictions of that input. This minimization rests on recurrent neuronal interactions between different levels of the cortical hierarchy in which bottom-up signals relay prediction error to higher levels, which respond by changing expectations to provide better top-down predictions (optimization of the posterior expectations or beliefs). This is a relatively simple and neuronally plausible scheme for which there is a large amount of circumstantial evidence.<sup>8</sup> This evidence suggests that superficial pyramidal cells in the upper layers of cortex (green triangles) compare expectations at each hierarchical level (or sensory input at the lowest level) with top-down predictions from deep pyramidal cells (black triangles) of higher levels. The resulting prediction error is then returned to the deep pyramidal cells so that they can change their predictions. (B) This schematic shows a simple segment of the cortical hierarchy with ascending prediction errors and descending predictions. Here we have included neuromodulatory gating or gain control (red) on superficial pyramidal cells that determines their relative influence on deep pyramidal cells encoding expectations. (C) This provides a particular example focusing on the visual system. The black arrows denote descending (or backward) connections that convey predictions. The green arrows denote ascending (or forward) driving connections from cells of a lower area to a higher area that convey prediction errors. The prediction errors are weighted by their expected precision, which depends on neuromodulatory input—here from ventral tegmental area (VTA) and substantia nigra (SN). Predictions try to cancel (or at least reduce) prediction errors in lower levels. In this example, extrastriate visual cortex sends (top-down) predictions to primary visual cortex, which then sends predictions to the lateral geniculate body. Extrastriate visual cortex also sends (proprioceptive) predictions to the pontine nuclei. These predictions are transmitted to the oculomotor system to produce movement through classical reflexes.<sup>36</sup> Predictions from the pontine nuclei are also sent to the lateral geniculate body (c.f., corollary discharge). Every top-down (descending) prediction is reciprocated with a bottom-up (ascending) prediction error to ensure that predictions are constrained by sensations.<sup>36</sup> Thus the minimization of prediction error occurs at all levels of the cortical and subcortical hierarchy and at the level of peripheral reflexes. The resolution of proprioceptive prediction error is particularly important because this enables descending predictions about the state of the body to cause movement by dynamically resetting the equilibrium or set point of classical reflexes. In other words, the proprioceptive prediction errors about the oculomotor system eliminate themselves directly through peripheral motor reflexes.

how does the brain select among the myriad of prediction errors that it could explain away? In predictive coding, the *precision* of prediction errors reflects the degree of confidence in the information conveyed by ascending prediction errors at any level of the

hierarchy. Precision is the inverse of variability or uncertainty, so precise prediction errors at one hierarchical level will come to dominate inference in the remaining levels. A simple analogy here would be the confidence we place in—or weight we place

on—information from a reliable or precise source. In this sense, the expected precision of different sources of (sensory) information can be associated with attending to a particular source.<sup>11</sup> Psychologically, when one source (sensory modality or hierarchical level) is expected to have a higher precision than others, its precision increases and we attend to—or select—this source to update our predictions. Physiologically, this precision weighting is thought to be implemented neuronally by a top-down modulation of the gain of cells reporting prediction errors.

This means that ascending prediction errors are precision-weighted; in other words, a mismatch with a high expected precision will engender an amplified or salient prediction error (c.f., attentional gain). Put simply, ascending precision-weighted prediction errors report what is newsworthy in the incoming signal—not the sensory signal per se.<sup>1</sup> The cortex is therefore more of a (salient) error detector than a feature detector. The notion of precision is an important aspect of predictive coding and suggests that the brain not only predicts the content of sensory input but also requires top-down predictions of the precision or confidence that contextualizes that input. It is the failure of this neuromodulatory contextualization that many people now consider a candidate target for pathophysiology in several neuropsychiatric syndromes (see below).

**NEURONAL MESSAGE PASSING AND PREDICTIVE CODING** In terms of neuronal implementations of predictive coding, the current hypothesis is that the representational (prediction-driving) and mismatch (prediction error-driving) neuronal populations correspond to different classes of neurons within a given cortical level.<sup>9,10</sup> Specifically, prediction errors are thought to be encoded by superficial pyramidal cells that are the source of ascending or forward connections in cortical hierarchies. Conversely, the predictions themselves may originate in the deep pyramidal cells that are the source of long-range top-down or descending projections. There is a large amount of empirical evidence in support of this scheme—in particular the functional asymmetries in forward and backward connections and the fact that the superficial pyramidal cells have numerous mechanisms for gain control (e.g., a preponderance of NMDA receptors and classical neuromodulatory receptors such as D1 and D2 receptors<sup>8</sup>). This is important because the postsynaptic gain of superficial pyramidal cells may encode the precision of the prediction errors—and may mediate things like attentional gain,<sup>11</sup> sensory attenuation,<sup>12</sup> and affordance. Here, affordance corresponds to the latent possibilities for action given an agent's capabilities; for example, balls are for throwing or bouncing.<sup>13</sup> In other words, precision may

be involved in selecting cues that elicit utilitarian action.<sup>12</sup>

So far, we have restricted ourselves to perceptual synthesis in the exteroceptive (i.e., visual) domain. However, exactly the same arguments may apply to perceptual inference and ensuing consciousness of the physiologic state of the body (interoception). Crucially, *interoceptive inference* may underlie emotions and feelings: our “sentient self” (the material me) would therefore be constituted by successive interoceptive predictions, which are thought to be updated within the insula (and anterior cingulate cortex).<sup>14</sup> This model of interoception as a predictive process reconciles the James-Lange/Damasio somatic marker model of consciousness and models involving cortical influences. The James-Lange/Damasio somatic marker model argues that the mental self is anchored in the internal state of the body and that emotions arise from the (continuous) perceptions of changes in the body. The model of interoception as a predictive process indeed posits a combination of descending predictions and ascending interoceptive signals (prediction errors), with simultaneous exchanges between the cognitive predictions (depending on the prevailing context and past experiences) and sensory (interoceptive) feedback.

**ACTION AND PERCEPTION** In addition to perception and cognition, the predictive processing model also applies to action and offers a unifying model for perception and action, which have been regarded classically as distinct functional systems. In the predictive formulation, action is regarded as the fulfillment of descending proprioceptive predictions through classical reflex arcs. In more detail, the brain generates continuous proprioceptive predictions about the expected location of the limbs and eyes that are hierarchically consistent with the (higher) goal of the movement. In other words, we believe that we will execute a goal-directed movement and this belief is unpacked hierarchically to provide proprioceptive and exteroceptive predictions. These predictions are then fulfilled automatically by minimizing proprioceptive prediction errors at the level of the spinal cord and cranial nerve nuclei. The ensuing hierarchical control accommodates real-time adjustment/correction of the movement to nuance the fulfillment of descending proprioceptive predictions or corticospinal motor commands—or to update these commands.<sup>15,16</sup> Neurophysiologically, these updates may occur very quickly (each ~150 ms, i.e., at around 6–7 Hz—the theta band). The time constant of 125–150 ms seems to correspond to the minimal amount of time necessary to form a new percept.<sup>1,17–19</sup> The argument here is that the same inferential mechanisms underlie apparently diverse functions (action and perception) but are essentially

the same—for example, in the primary visual cortex for vision, the insula for interoception, the caudate nucleus for reward,<sup>20</sup> and the motor cortex for movement and proprioception.<sup>21</sup> Crucially, because all of these modality-specific systems are organized hierarchically, they are all contextualized by the same conceptual (amodal) predictions. In other words, action and perception are facets of the same underlying imperative—namely, to minimize hierarchical prediction errors through selective sampling of our sensory inputs.

### PRECISION, PATHOPHYSIOLOGY, AND PSYCHOPATHOLOGY

Predictions are generated continuously within the brain—descending down different neuronal streams (e.g., auditory, visual, interoceptive, and proprioceptive), with hierarchically decomposed representations for different perceptual attributes within each modality (e.g., form, color, and movement in the visual modality). Hierarchical predictive coding also implies the continuous Bayesian updating of representations at many spatial and temporal scales “to settle into a mutually consistent whole in which each hypothesis is used to help tune the rest”<sup>21</sup>—allowing the brain to make the best predictions at many levels of abstraction.<sup>7</sup> Crucially, the balance among inference at different levels of the hierarchy depends—in a very sensitive way—on the precision assigned to prediction errors at various levels. Neurobiologically, this is another way of saying that distributed hierarchical processing depends on the neuromodulatory mechanisms that encode precision and enable message passing among different neuronal systems.

A large number of neurologic and psychiatric syndromes can be cast, mechanistically, as a failure of neuromodulation or gain control. They can be regarded as failures of active inference. Examples of false inference in the perceptual domain include “physiologic” illusions and hallucinosis, particularly in the visual system.<sup>22</sup> This sort of false (perceptual) inference is easy to simulate by reducing the precision of visual input relative to the precision of prediction errors at higher levels in the visual hierarchy (see reference 16 for an example). More pernicious forms of false inference—at higher hierarchical levels involving inferences about affordance (action possibilities) and agency (the agent of action, e.g., self vs other)—have been proposed to underlie delusions and hallucinations in psychosis.<sup>23,24</sup>

The Bayesian approach to predictive coding is indeed particularly appropriate for understanding conditions like schizophrenia because the symptoms can be seen as a failure of (active) inference. For example, delusions and hallucinations can be regarded as false inference in the perceptual domain,<sup>23,24</sup> while psychomotor poverty can be regarded as a failure of

descending motor predictions to elicit movement.<sup>13</sup> Crucially, all these instances of false inference can be explained in a parsimonious way in terms of a failure to estimate the confidence in, or precision of, prediction errors. The nice thing about this is that it provides a functional explanation for the emergence of clinical symptoms while furnishing a neurophysiologically plausible explanation in terms of abnormal neuromodulation (possibly involving dopamine). When one tries to model the symptoms and signs of psychosis and psychomotor poverty, it transpires that the most likely primary deficit is a failure of *sensory attenuation*. For example, during action we normally attenuate the sensory consequences of our own movement (for example, we are not aware of the optical flow produced by eye movements). Sensory attenuation causes us to underestimate forces we produce in relation to the same force that is applied passively—this is the force-matching illusion.<sup>25</sup> Effectively, this is because we pay less attention to self-caused sensations. Crucially, patients with schizophrenia show a failure of sensory attenuation and are resistant to the force-matching illusion.<sup>26</sup> In the current setting, this corresponds to a failure to attenuate sensory precision during self-made acts.<sup>12,24</sup> In other words, people with schizophrenia (and possibly autism) cannot ignore the sensations they cause themselves. It has been proposed that this failure drives compensatory increases in precision (postsynaptic gain) at higher levels of the hierarchy—at the price of explaining sensations in a delusory fashion<sup>12</sup> (i.e., by placing too much confidence in their prior beliefs). Of interest, when these explanations involve the consequences of movement, they necessarily involve a failure to attribute agency.<sup>12,24</sup> In short, a neuromodulatory deficit at low (sensory) levels of the hierarchy results in a failure to contextualize—or attend to—incoming sensory information. Compensatory changes in precision at higher levels of the cortical hierarchy endow hierarchical beliefs with too much confidence, thereby providing a Bayesian account of delusions and hallucinations.<sup>23,24</sup>

A failure in sensory attenuation can sometimes lead to paradoxical improvements in psychophysical performance—for example, a peculiar resistance to illusory phenomena in schizophrenia. Similarly, the failure of sensory attenuation leads to the hypervigilant attention to sensory detail at the expense of a hierarchically deep explanation for sensations. This imbalance in precision has been proposed as an explanation for many of the features of autism<sup>27</sup> (c.f., loss of central coherence<sup>27</sup>). When this sort of mechanism is applied to control of eye movements, one can also reproduce many of the smooth pursuit deficits that are a paradigmatic example of soft neurologic signs in schizophrenia (e.g., reference 28). In this context, a failure to attenuate visual input precludes confident

eye tracking when a target is behind an occluder, leading to deficits in slow pursuit.<sup>28</sup> Similar simulations of parkinsonism result in perseverative (repetitive and nonadaptive) behavior and characteristic hypokinesia.<sup>13</sup> The key point here is that apparently disparate clinical phenomena are underpinned by one simple mechanism—false inference due to aberrant encoding of precision through a neuromodulatory failure of synaptic gain control.

Prediction errors in interoception and cognitive processing (for instance in reward anticipation) are often experienced as aversive and threatening (c.f., reference 29) and may result in feelings of uncertainty and discomfort.<sup>30,31</sup> The aberrant modulation of (attention to) prediction errors may also be associated with anxiety disorders<sup>32</sup> and has been proposed as the basis of hysterical or functional medical symptoms.<sup>33</sup> In a similar vein, we have postulated that a pathologic absence of precise prediction errors may lead to feelings of trust, well-being, and inner peace, such as those encountered in some forms of epilepsy with “ecstatic” symptoms. This state could be understood as a disruption—by epileptic discharges in the insula—of the “comparator between predictions and real inputs” for multisensory integration and interoception.<sup>34,35</sup> This hypothesis tallies with the description by Mumford<sup>10</sup> of an “ultimate stable state” in which top-down signals would perfectly predict representations in lower cortical levels, which would be the state the brain is trying to achieve: a perfect prediction of the world, like the Oriental nirvana.

**CONCLUSION** The predictive or inferential perspective on brain function provides a formal and compelling framework for connecting hierarchical neuronal processing with our experience of the world. In this framework it may be possible to understand many neurologic and psychiatric conditions in terms of false inference that has perceptual, behavioral, and emotional connotations. In this essay we have tried to describe these ideas and connect them to plausible neurobiological processes. In particular, we have focused on the central role of neuromodulation (and its failures) in nuancing hierarchical inference in the brain. This functional perspective may be important in understanding the impact of pathophysiology on the predictive brain and potential therapeutic interventions.

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#### REFERENCES

1. Clark A. Whatever next? Predictive brains, situated agents, and the future of cognitive science. *Behav Brain Sci* 2013; 36:181–204.
2. von Helmholtz H. Concerning the perceptions in general. In: *Treatise on Physiological Optics*, Vol III, 3rd ed; 1866 [translated by JPC Southall 1925 Opt Soc Am Section 26. Reprinted New York: Dover, 1962].
3. Dayan P, Hinton GE, Neal RM, Zemel RS. The Helmholtz machine. *Neural Comput* 1995;7:889–904.
4. Friston K. A theory of cortical responses. *Philos Trans R Soc Lond B Biol Sci* 2005;360:815–836.
5. Gregory RL. Perceptions as hypotheses. *Philos Trans R Soc Lond B Biol Sci* 1980;290:181–197.
6. Friston K, Adams RA, Perrinet L, Breakspear M. Perceptions as hypotheses: saccades as experiments. *Front Psychol* 2012;3:151.
7. Rao RP, Ballard DH. Predictive coding in the visual cortex: a functional interpretation of some extra-classical receptive-field effects. *Nat Neurosci* 1999;2:79–87.
8. Friston K. Hierarchical models in the brain. *PLoS Comput Biol* 2008;4:e1000211.
9. Bastos AM, Usrey WM, Adams RA, Mangun GR, Fries P, Friston KJ. Canonical microcircuits for predictive coding. *Neuron* 2012;76:695–711.
10. Mumford D. On the computational architecture of the neocortex. II. The role of cortico-cortical loops. *Biol Cybern* 1992;66:241–251.
11. Feldman H, Friston KJ. Attention, uncertainty, and free-energy. *Front Hum Neurosci* 2010;4:215.
12. Brown H, Adams RA, Parees I, Edwards M, Friston K. Active inference, sensory attenuation and illusions. *Cogn Process* 2013;14:411–427.
13. Friston KJ, Shiner T, FitzGerald T, et al. Dopamine, affordance and active inference. *PLoS Comput Biol* 2012;8:e1002327.
14. Seth AK. Interoceptive inference, emotion, and the embodied self. *Trends Cogn Sci* 2013;17:565–573.
15. Shadmehr R, Smith MA, Krakauer JW. Error correction, sensory prediction, and adaptation in motor control. *Annu Rev Neurosci* 2010;33:89–108.
16. Adams RA, Shipp S, Friston KJ. Predictions not commands: active inference in the motor system. *Brain Struct Funct* 2013;218:611–643.
17. Dikker S, Rabagliati H, Farmer TA, Pylkkanen L. Early occipital sensitivity to syntactic category is based on form typicality. *Psychol Sci* 2010;21:629–634.
18. Crouzet SM, Kirchner H, Thorpe SJ. Fast saccades toward faces: face detection in just 100 ms. *J Vis* 2010; 10:11–17.
19. Picard F, Craig AD. Ecstatic epileptic seizures: a potential window on the neural basis for human self-awareness. *Epilepsy Behav* 2009;16:539–546.
20. Cromwell HC, Schultz W. Effects of expectations for different reward magnitudes on neuronal activity in primate striatum. *J Neurophysiol* 2003;89:2823–2838.
21. Rosenkranz K, Rothwell JC. Modulation of proprioceptive integration in the motor cortex shapes human motor learning. *J Neurosci* 2012;32:9000–9006.
22. De Ridder D, Verplaetse J, Vanneste S. The predictive brain and the “free will” illusion. *Front Psychol* 2013; 4:131.
23. Fletcher PC, Frith CD. Perceiving is believing: a Bayesian approach to explaining the positive symptoms of schizophrenia. *Nat Rev Neurosci* 2009;10:48–58.

24. Adams RA, Stephan KE, Brown HR, Frith CD, Friston KJ. The computational anatomy of psychosis. *Front Psychiatry* 2013;4:47.
25. Shergill SS, Bays PM, Frith CD, Wolpert DM. Two eyes for an eye: the neuroscience of force escalation. *Science* 2003;301:187.
26. Shergill SS, Samson G, Bays PM, Frith CD, Wolpert DM. Evidence for sensory prediction deficits in schizophrenia. *Am J Psychiatry* 2005;162:2384–2386.
27. Frith U. *Autism: Explaining the Enigma*. Oxford, England: Blackwell ed; 1989.
28. Adams RA, Perrinet LU, Friston K. Smooth pursuit and visual occlusion: active inference and oculomotor control in schizophrenia. *PLoS One* 2012;7:e47502.
29. Hajcak G, Foti D. Errors are aversive: defensive motivation and the error-related negativity. *Psychol Sci* 2008;19:103–108.
30. Gray MA, Harrison NA, Wiens S, Critchley HD. Modulation of emotional appraisal by false physiological feedback during fMRI. *PLoS One* 2007;2:e546.
31. Sarinopoulos I, Grupe DW, Mackiewicz KL, et al. Uncertainty during anticipation modulates neural responses to aversion in human insula and amygdala. *Cereb Cortex* 2010;20:929–940.
32. Paulus MP, Stein MB. An insular view of anxiety. *Biol Psychiatry* 2006;60:383–387.
33. Edwards MJ, Adams RA, Brown H, Parees I, Friston KJ. A Bayesian account of “hysteria”. *Brain* 2012;135:3495–3512.
34. Picard F. State of belief, subjective certainty and bliss as a product of cortical dysfunction. *Cortex* 2013;49:2494–2500.
35. Picard F, Kurth F. Ictal alterations of consciousness during ecstatic seizures. *Epilepsy Behav* 2014;30:58–61.
36. Hobson JA, Friston KJ. Waking and dreaming consciousness: neurobiological and functional considerations. *Prog Neurobiol* 2012;98:82–98.