Review

Errors or Adaptations? A Critical Review of Predictive Processing in Psychiatry

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Errors or Adaptations? A Critical Review of Predictive Processing in Psychiatry

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Abstract

Predictive processing (PP) accounts often characterize post-traumatic stress disorder (PTSD) and depression as maladaptive and epistemically distorting, due to wide divergences between brain generated top-down models and bottom-up sensory inputs. This review questions that characterization. First, trauma survivors with PTSD or depression may lessen risk by overestimating threats, with hypervigilance sustaining a desirable gap between anticipated problems and harms that would otherwise occur. Second, PP psychiatric frameworks prescribe how trauma survivors ought to assess matters yet give limited justification, introducing tacit normative assumptions into neurocomputational models. This repeats in certain PP assessments of schizophrenia and psychosis, which presuppose Western concepts of self as normative neurocognitive ideals. Third, PP accounts claim that cognition evolved primarily for action, not veridical representation; but their notion of prediction error can tacitly invoke the concept of veridical representation. Fourth, PP defenders have asserted that depressive slow-downs follow from maladaptive brain-based regulatory models. However, physiological problems may instead make activity strenuous, such that slowing down is adaptive. This position advanced in this review is that atypical mental outlooks need not be epistemically distorted, and that mismatches between anticipatory models and outcomes—when they occur—can sometimes index adaptive success rather than failure.

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1. Introduction

Predictive processing (PP) approaches hold that the brain operates through hierarchical inference, generating top-down expectations about sensory input, revising them by minimizing prediction error [1–4]. The core hypothesis is that the nervous system builds predictive models from statistical regularities in prior experience [5,6]; and that organisms perceive and cognize according to these priors rather than directly from sensory input, unless incoming signals significantly conflict with and thereby update them [1,7–11].

PP theorists have proposed links between unreliable brain-generated predictive models and mental disorders, including psychotic as well as affective disturbances. Suggested mechanisms include overly negative priors or unrealistically positive ones that thwart expectations [12–23]. Also listed are aberrant precision weightings [19,24], defined as the relative trust given to sensory input compared to that assigned to prior expectations. The brain might increase the weight assigned to a top-down model (prior) when heavy fog degrades incoming signals. Whether this is healthy or not, according to certain PP frameworks, depends on the predictive reliability, that is, the plausibility and helpfulness of the specific prior.

Thus, PP-based psychiatry tends to construe persistent prediction error as pathological, interpreting deviations from statistically typical predictive profiles as neurocomputational failures of inference [12,25–34]. However, the framing risks overlooking several points that are especially salient in clinical and early-intervention contexts. First, conditions of threat or hardship may not be

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anomalies but enduring features of an individual's environment, as in contexts of chronic trauma or social adversity.

Second, the expectation of harm may motivate actions that avert it, thereby sustaining a desirable gap between predictive models and outcomes registered in sensory information.

Third, preventive success may be greater when threats are heavily overanticipated, an outcome observed in conditions such as post-traumatic stress disorder (PTSD) or depression, partly due to the activation of neuromodulatory systems associated with vigilance and threat sensitivity [35–38]. Here, adversities may fail to materialize precisely because they are strongly anticipated and actively avoided, though this may also increase false alarms.

In such cases, an established prior can be considered adaptive insofar as it successfully prevents anticipated dangers, which also amounts to being epistemically attuned to potential problems. Almost paradoxically, then, a functioning predictive model in these contexts requires sustained prediction errors. Classifying such responses as inherently overreactive or paranoid neglects the possibility that adverse outcomes do not occur precisely because they are strongly anticipated and averted.

PP accounts simultaneously introduce tacit normative assumptions by implying a standard for how individuals ought to weigh objective risk and what counts as an overly precise (sensitive) prediction model. The bases for such assumptions are unclear, since a given trauma may affect one individual more severely than another. The upshot is that hypervigilance or paranoia may just be reasonable caution—here conceived as a trade-off that increases false alarms in return for improved detection of otherwise missed cues, the identification of which reduces the risk of traumatic recurrence.

In some cases, there may be no trade-off at all. What PP theorists label as hyperprecise, low margin-of-error models—which definitionally means error-prone—may simply and more parsimoniously amount to increased acuteness, as will be discussed when reviewing PTSD. PP interpretations of schizophrenia can also be normatively loaded, for instance, by treating deviations from Western conceptions of self and agency as markers of the disorder [39]. Yet schizophrenia appears less severe in parts of the Global South [40–42], possibly in part because people there understand selfhood and agency differently than in the West [43].

A key concept in PP is active inference [4,44], which reduces prediction errors via behaviors or processes that change the world (or in some case interoceptive conditions), so that sensory inputs conform to prior expectations. However, the defensive scenarios outlined above, insofar as they anticipate threats to avert them, emphasize actions that preserve a gap between expected harms and actual outcomes, thereby sustaining large prediction errors. Further, there are cases where anticipating failure brings about the predicted outcome [45]. Although PP theorists might claim that error-prone hyperprecision is part of this process [2,25,44], the scenario could also be cited as an instance where pathology involves the alignment between priors and outcomes.

None of these considerations, however, challenge the PP assumption that cognition evolved primarily as a coping-oriented, action-guiding capacity [2,46–50]. Practical tools such as shovels or subway maps facilitate effective engagement with the world without mirroring it with high-fidelity precision [51,52]. By parity of reasoning, cognitive patterns that deviate from veridical representation may still be functionally and epistemically appropriate, including in clinical populations. Some PP proponents acknowledge this when they argue that the brain evolved to minimize expected surprise rather than to represent the world accurately [1,47,50,53,54]. Yet their framings still fail to capture cases in which individuals habitually have negative expectations, which circumvent bad outcomes, thereby increasing both prediction error and surprise.

Enactive cognitive science [55] and ecological psychology [56]—plus their historic antecedents in American functionalism [57] and phenomenology [58]—provide complementary perspectives that illuminate mental health. According to these action-based approaches, a setting's properties are brought out by patterns of organism–environment interaction: water appears walkable or swimmable depending on a creature's capacities [55–59]. Action-oriented frameworks also illuminate affectivity.

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For example, objects tend to look farther away to sad individuals [60], a perception that may reflect a predictively accurate model. After all, depression associates with altered bodily processes, including irregularities in interoception, autonomic activity, fronto-striatal coordination and subcortical signaling, alongside neuroimmune adjustments such as inflammation [61–63]. This is typically fatiguing [61–65]. Depression accordingly coincides with physiological burden [64–70], generating objectively strenuous circumstances in which targets are genuinely harder to reach, with the appearance of greater distances indexing this [52]. In such cases, the predictive model could be a result rather than a cause of somatic changes and need not be inaccurate.

Conversely, when depression, PTSD or psychosis-risk states involve heightened sensitivity to negative possibilities, this stance may be epistemically revealing and adaptive insofar as it motivates actions that forestall unwanted outcomes. In wartime contexts, restlessness and rigid interpretation—such as hearing bangs as gunshots rather than fireworks—can be life-preserving, even if attacks are statistically infrequent [43]. Following sexual abuse, elevated vigilance may persist given the high risk of re-victimization [43,71]. Trauma-induced depression may likewise promote analytic processing and anticipation of problems, thereby thwarting their reoccurrence [72,73].

Importantly, acknowledging these dynamics does not deny the substantial costs of PTSD, depression or psychosis, including neural and physiological changes associated with sustained stress, dopaminergic irregularities and effortful coping [74,75]. Rather, the suggestion is that divergences between anticipated harm and actual outcomes can in some contexts be both adaptive and epistemically warranted. Predictive models may sometimes be consequences rather than causes of somatic burden, and in such cases may still be predictively accurate.

These considerations pose challenges for PP theories. First, some versions advance broad unifying claims [1–3,8] such that even limited counterexamples are theoretically problematic. Second, PP already faces difficulties in securing strong empirical confirmation, especially on a neurobiological level [76–79]. If psychiatric conditions including psychosis can arise from both predictively accurate and inaccurate models and if these models can function as either causes or consequences of disorder, then the falsifiability of PP at least as applied to clinical diagnosis, prognosis and treatment is substantially weakened.

2. Anticipatory Processes in Action-Oriented Cognition

Several themes recur throughout this article concerning the integration of affect, cognition and action in both ordinary and clinical contexts, and this section reviews them. The aim is to clarify certain commitments that inform the subsequent critiques of PP.

Western commentators have historically portrayed the emotions as epistemic contaminants, though exceptions have been noted [80,81]. However, since the 1990s, neurobiologists and cognitive scientists have increasingly broken this trend. Among them are Schulkin [81,82] and Damasio [83], who emphasize the involvement of emotional processes in rational adjudication, sustained effort and selective attention, citing functional psychology as an inspiration. Gigerenzer makes similar points [84], and Pessoa's integrative framework further dissolves sharp boundaries between "cognitive" and "emotional" systems at the neural level [85].

Taken together, these strands establish a conceptual and empirical backdrop for evaluating PP interpretations of psychiatric conditions. One reason is that emotional disturbances often characterize mental illness. A more PP-oriented consideration is that affective processes regulate uncertainty-reducing cognitive and perceptual activity, narrowing attention and excluding irrelevant information [83–86]. This framing remains compatible with diverse psychological approaches while supporting greater circumspection in the application of categorical diagnostic classifications.

Neurobiological evidence ties affective processing with a wide range of cognitive functions. The amygdalae, for instance, have been implicated in selective attention through their role in integrating affective relevance with perceptual and cognitive processing [85]. Rather than operating solely as a hub that assigns emotional significance to internal bodily signals, amygdalar circuits are closely coupled with hippocampal and cortical systems involved in memory consolidation, language

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processing and concept deployment [86–91]. Epistemically complex emotions, including shame, envy and embarrassment, are likewise associated with distributed cortical and subcortical networks involved in semantic processing and social cognition [92–94].

Another example is the insular cortices. This region associates with emotions such as empathy, fear and disgust [95,96], which are again epistemically complex insofar as they guide social behavior and the appraisal of environmental threats. Through connections with the basal ganglia, insula-centred circuits have been implicated in habit learning, reward evaluation and action selection [97]. Related cortico–basal ganglia networks have further been linked to the temporal and syntactic organization of language and music, domains that depend on anticipation and expectation [98–100].

Everyday psychological phenomena further illustrate the close coupling of affect, cognition and action [101]. Objects and situations acquire significance relative to affectively shaped goals; superficially similar linguistic expressions can convey different meanings depending on emotional tone; and affective dynamics systematically modulate attention, associative processes and memory [102–107]. Affective interests additionally influence concept formation [25,82,83,109], as illustrated by the differing functional meanings of a material such as oil for a carpenter, a mechanic and a dry cleaner [110]. Supporting this view, affective neural systems—such as amygdalar circuits—are involved in both object recognition and the guidance of action readiness [85].

Affectivity accordingly shapes perception by modulating sensitivity to possibilities for action [101]. Consider a hiker engaged in skilled, purposeful movement. She might perceive a frozen river surface as stable or unstable based on bodily capacities and situational conditions, experiencing this difference affectively as safety or threat, and practically as either affording or not affording locomotion [111]. Ecological approaches formalize this relationship by treating affordances as inherently prospective, since perceiving what can be done requires sensitivity to outcomes that have not yet occurred [58].

While PP conflicts with ecological psychology by presupposing internal brain-based models that construct experience of the world, PP obviously shares ecological psychology's prospective slant. On a PP account, the hiker, if walking on an urban sidewalk, would not register the absence of open utility holes or loose concrete tiles; she would instead operate on a predictive model that assumes easy passage, unless interrupted by an unexpected hazard, with PP advocates arguing that this maximizes efficiency [1–9].

Emotions such as worry and hope involve expectations that integrate affect with prediction. For PP advocates and non-advocates alike, affectivity is central to anticipation [112–118], which is almost definitionally cognitive insofar as it forecasts what is not present. This dynamic is evident in language. For instance, "but" and "and" are logically identical; yet listeners typically anticipate different emotional closures to the statements "I love you but…" and "I love you and…" [43]. Similarly, the phrase "in the event" constrains expectations to anticipate continuations like "that" or "of," yet not "pie" or "to" [119]. This last example comes from a pioneering book in information theory that basically advances the PP contention that predictive models enhance efficiency and reduce uncertainty.

These patterns therefore align with PP accounts, which propose that cognition involves the generation of expectations about future sensory states [1,4,120,121]. Within this framework, affectivity is understood to modulate both the content and the relative weighting of the generative models that guide perception and action [19,122–124]. Emotions, interests and moods direct attention toward task-relevant information, modelling the world by shaping expectations about action–outcome relations and regulating sensitivity to prediction error [4,44,121,125,126].

Relevant to PP and active inference, areas such as the dorsal striatal circuits support habit formation, which is modulated by affective and motivational systems like the amygdala bulbs [127–132]. Habits improve efficiency by constraining action selection, reduce uncertainty and increase focus while lessening attentional demands [133,134]. They again model what shows up in perception, and when anticipated outcomes do not occur, the resulting prediction error can promote the revision

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of these habitual models [4,44,135–138]. This is partly why PP theorists have characterized habit as higher-level priors [4,44,135–138].

Whether specifically advancing a PP standpoint or not, bodily activity supports these predictive processes. Posture influences arousal, cardiovascular activity and neural processing [139,140], while eye movement patterns associated with fear and disgust differentially support stimulus localization or discrimination [141]. Cultural habits further shape affordance perception, influencing what is perceived as edible, familiar, or aversive within a given environment [142].

In PP terms, this discussion of habits and bodily action bears directly on how precision is calibrated. In contexts such as Japan, where jaywalking is uncommon, behavioral cues associated with street crossing carry high precision—that is, the expected probability of prediction error is low. The movement of others at a crosswalk reliably signals when it is safe to proceed, stabilizing higher-level priors that guide pedestrian behavior [143]. When an individual relocates to a setting in which these regularities do not hold—for example, where crosswalk norms are frequently ignored—repeated prediction errors would ordinarily update these priors. The movement of others would cease to function as a dependable cue, and precision weighting would recalibrate accordingly [143]. Consistent with hierarchical PP accounts, much sensory input remains backgrounded under ordinary conditions and becomes salient only when it violates established expectations and generates prediction error.

Now, retaining a Japanese jaywalking prior after permanently relocating would be maladaptive. PP theorists interpret mental disorders as structurally similar. Psychopathology is modeled as a disturbance in the balance between prior expectations and sensory evidence, particularly through aberrant precision weighting—that is, misestimation of the relative reliability of priors and incoming signals [1–3,15,34].

Building on observations like this, the next section discusses a recurrent PP hypothesis. This is that certain psychiatric conditions are marked by excessively precise or rigid higher-level priors that fail to update appropriately, thereby sustaining maladaptive expectations over time.

3. Precision, Prediction and Psychopathology

This section provides a basic overview of PP accounts of psychopathology. Subsequent discussions examine specific disorders in greater detail and critically evaluate PP framings.

On PP accounts, mental health conditions may arise when higher-level priors become overly rigid or precise, limiting belief updating in response to environmental input [15,34]. Another issue can be overly precise thresholds for sensory prediction errors [1–3,12,13,120,121]. For example, the faint sound of a tennis ball bouncing on a court ten stories below a library room would not violate most expectations of a quiet workspace. However, people with ADHD or autism may be overly exacting, thereby registering a gap (error) between the situation and their expectations [12,13].

For PTSD, a PP proposition is that trauma establishes hyperprecise threat-related priors; these dominate perceptual inference, biasing ambiguous cues toward danger, impairing extinction or safety learning [27,33,144,145].

In psychosis, similarly, disrupted precision weighting is hypothesized to underlie hallucinations and delusions [34,146,147]. One purported mechanism is excessively precise higher-order beliefs that resist counterevidence. As an example, PP literature discusses an experiment in which participants were led to expect the song “White Christmas”; they were instead played white noise, with a percentage nonetheless reporting that they heard the song [1,27,148]. Another culprit is abnormally precise sensory prediction errors [1–3,12,13,120,121]. Here, the brain might assign great significance to slight irregularities in another’s behavior, interpreting it in paranoid ways even though the deviation would go unnoticed by most. According to PP theorists, weak priors can also cause problems [2,3]. This could happen if the brain ignores statistical patterns and instead interprets innocuous inputs, such as indistinct shadows and faint sounds, as indicating an intruder in a dark bedroom.

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Trauma may heighten receptivity to certain sensory inputs, with an interviewee from one study reporting that she can smell cigarettes on those who have brushed their teeth and showered because, during her childhood, the odor signaled an abuser's proximity [43]. Under Paulus and Stein's interpretation, an initially neutral stimulus—tobacco—becomes conditioned as a threat cue, generating interoceptive signals such as elevated pulse and internal shakiness [149]. This response can persist even after the danger has passed. Paulus and Stein add that in anxiety-prone individuals, the anticipatory signal is excessively precise [149]. That is, the brain emphasizes low-grade differences between expected and actual arousal, such that worry and behavioral avoidance are easily triggered.

PP sometimes characterize such dynamics in terms of active inference, in which agents act to bring sensory inputs, including interoceptive ones, into alignment with their predictions [122]. When struggling with volatile environments, the brain might shift into a "safety first" mode, selecting actions that minimize the likelihood of unpleasant surprises. This is active inference, and it may improve things briefly; yet it can also prevent the person from learning that the situation is safe [150].

Relevant PP framings of mental illness are consistent with cognitive penetrability, understood as the idea that higher-level cognitive states—such as beliefs, desires or intentions—can directly shape perceptual experience [151]. For example, when riding a subway, a person may mistakenly reverse the east–west orientation of the windows, producing the impression of moving in the opposite direction [143]. Hypothetically, one could have a prior in which east and west are constantly inverted, thereby creating a persistent hallucination of travelling contrary to the train's actual trajectory.

PP models of psychosis extend this logic. On influential accounts, delusions and certain symptoms of schizophrenia arise when aberrant precision weighting disturbs the balance between prior expectations and sensory evidence [15,34,152]. Highly precise priors may resist revision despite prediction errors [15,34,153]. This is while overweighted sensory errors can drive unstable updating and implausible hypotheses to explain noisy input [15,147,154,155]. Examples of such occurrences have already been given.

PP also proposes overly imprecise priors as underliers of schizophrenia [156–158]. Here, the brain may have a weak prior predicting silence in a room devoid of people, televisions or other sources of vocalization, thereby increasing the likelihood that internal thoughts and dialogues are misattributed to an external source. PP theorists also propose that suffers could have weak model for differentiating self-produced internal speech, construing it as coming from without [157]. Psychotic symptoms are thus framed not as wholly sui generis phenomena but as extreme variants of the same inferential mechanisms that ordinarily support adaptive perception and action [15,153,159].

In depression, negatively biased and overly stable priors about the self or the future may blunt responsiveness to positive prediction errors, thereby sustaining pessimism [31]. Another account suggests that the disorder can arise when the brain deploys models that poorly govern internal allostatic processes [25]. One possible causal contributor is dysregulated release of cortisol and corticotropin-releasing hormone (CRH), which may diminish energy when it is needed or elevate it when it is not [81,143], contributing to cycles of depressive slowing and mania.

This last example provides a convenient way to illustrate both points of overlap and divergence between PP and other action-based interpretations [160,161]. Emotions such as sadness associate with reduced energy and action capacity, often accompanied by changes in perceived distance or effort, such that stairs and hills appear farther away or more sharply inclined [60,162–164]. From standard PP perspectives, perceiving the environment as steeper or more distant reflects an epistemically disordered prediction error. This, in turn, lowers expectations of success and heightens anticipated energy expenditure, possibly in conjunction with allostatic dysregulation [25,165,166].

By contrast, ecological psychology, enactive cognitive science and their antecedents in pragmatist or functionalist psychology and phenomenology customarily emphasize how reduced energy reshapes an agent's capacity to cope with environmental demands. On this view, there is little

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in the way of misrepresentation or aberrant modeling in the first place. Diminished vitality does not distort perception so much as enact a world that is really more demanding [167].

In sum, PP usually prioritizes brain-generated models or representations as the central determinants of experience [33,166,168]. Pragmatists, phenomenologists, ecological psychologists and enactivists place greater emphasis on ongoing organism–environment dynamics, sometimes treating them as constitutive of experience itself [55–59,167,169]. Commentators from these camps often avoid talking about mental representations or models altogether. To the extent that they do invoke them, representations are typically understood as consequences rather than antecedents of experience, again defined in terms of organism–environment dynamics. As subsequent sections will discuss, this contrast highlights normative impositions tacitly inserted into PP interpretations of depression, PTSD and certain forms of psychosis.

4. Depression, Prediction Error and Anticipatory Problem-Solving

A PP proposal is that depression arises from maladaptive and epistemically distorting prediction errors, a neurocomputational view this section critically interrogates.

One objection is that ruminative worry—which both PP and non-PP accounts associate with depression and sometimes psychosis—can function as a prospective, problem-oriented strategy aimed at anticipating and averting future harm. On this view, adaptive and epistemic success depends on generating prediction errors, understood as discrepancies between feared and actual outcomes. A second objection is that depression sometimes emerges under conditions where so-called distortions may instead reflect environmentally calibrated and in some cases warranted expectations.

A claim within PP accounts is that depression involves maladaptive top-down models. These disrupt interoception or bias attention toward adverse or threat-related information via altered priors and precision-weighting, such that pessimistic expectations are sustained and self-reinforced [20,25,26,31,121,170–173].

Consistent with this view, empirical research indicates that depression associates with heightened anxiety and bleak past- and future-oriented beliefs [35,87,174,175]. Relative to controls, those with depression exhibit heightened amygdalar activation both when anticipating negative outcomes and when processing sad stimuli [35,36]. In addition, the neural activity of non-depressed individuals during the anticipation of aversive stimuli resembles the baseline profile of depressed patients [36]. Within a PP framework, excessively precise (confident) negative priors (belief or models held before seeing data) can become resistant to counterevidence; this may stabilize delusional beliefs of guilt, persecution or nihilism, so that depressive and psychotic symptoms may lie within a continuum of maladaptive precision-weighting [15,152,176].

As stated, the assumption that depression is necessarily maladaptive warrants qualification. Like drinking water to relieve a dehydration headache, the intrinsically aversive experience of depression may motivate corrective action. Mood disruption may accordingly sometimes amount to a defensive or regulatory signal [177,178]. For example, when depression follows overwork, a person may withdraw or sleep to recover from unpleasant deflation. Similar patterns appear in non-human animals. In one experiment, squid given local anesthetic after tentacle amputation responded as though uninjured, suffering greater predation than their untreated counterparts [179]. More broadly, injury and sickness behaviors across species associate with safety-prioritizing, energy-conserving responses [180,181].

A line of evidence for the preceding view comes from rodent models, which induce a depression analogue via gastrointestinal illness or starvation. In the first case, rats show diminished libido and appetite alongside increased sleepiness and psychomotor slowing [73]. These patterns exemplify sickness behavior, a reallocation of physiological resources that conserves energy and facilitates immune response [73,181]. The second case likewise suppresses sexual behavior but increases motor activity and environmental exploration, facilitating food acquisition [73]. Human data from

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prolonged caloric deprivation shows similar motivational reprioritization [182]. In each case, the specific type of physiological strain elicits appropriate actions.

Commentators across traditions suggest that experiences of adversity can intensify deliberative modes of cognition [72,73,101,105,177,183–191], a finding that both aligns with and challenge PP claims.

On the one hand, intense and persistent rumination associates not only with depression but also with clinical and especially subclinical psychosis [192]. PP theorists note this connection [193,194], attributing psychosis to overly rigid predictive models that persist despite disconfirming sensory evidence [34,146,147]. In addition, depressive slowing coheres with active inference accounts of depression [25,194–197]. Here, the sufferer may calibrate behavior to a maladaptive prediction error, acting in ways that bring sensory input and environmental circumstances into alignment with it. Suppose, for example, that the brain adopts an overly precise interoceptive model that accentuates small downward deviations from expected energy levels, leading to an exaggerated experience of exhaustion. If the person then slows down and behaves in a fatigued manner, sensory inputs are brought into alignment with the prior [149].

On the other hand, evidence links dysphoria to increased systematic processing [185], which may contribute to healing [72,189]. Consistent with this, depression coincides with rumination and behaviors sustaining it [72,189]. This includes social withdrawal, though this is sometimes from large groups with dyadic relations more frequently maintained [191]. The afflicted may abandon previously rewarding activities, such as sex [72,189–191], paralleling the earlier mentioned rodent research [73].

Here, anticipatory caution and motor retardation [198,199], alongside enhanced analytic performance under low mood [72,200], are aspects in a coordinated reorientation—a slowing down to reevaluate matters. Likewise, the finding that dreaming and trauma-focused reflection can hasten recovery [72,184,201] supports the premise that depression recruits adaptive information-processing mechanisms. Taken together, these considerations support the qualified claim that ruminative depressive tenancies may under certain constrained conditions function as an epistemic strategy for resolving personal difficulties. This is with the caveat that excessive and rigid forms may nonetheless contribute to maladaptive trajectories across affective and psychotic disorders.

In fairness, PP authors occasionally get close to defending adaptive accounts of depression. Badcock and colleagues [203] (p. 188), for example, characterize such behavior as a "better safe than sorry" strategy aimed at minimizing expected surprise. On this view, the depressed brain adopts generative models that reduce social uncertainty when environmental cues signal volatility and an elevated probability of aversive interpersonal outcomes, often by increasing sensitivity to socially relevant prediction errors and promoting risk-averse behavior.

An alternative possibility, however, is that negatively biased but perhaps accurate self-models are actively protected from confirmation. Here, intervention aimed at adaptive change would instead reengage individuals in ways that increase prediction error—namely the discrepancy between entrenched negative self-representations and positive social input—allowing gradual model revision, long-term error reduction and symptom alleviation. This suggests that effective treatment may sometimes involve strategically increasing early prediction error, with implications for staged or early-phase intervention in at-risk populations.

Taken together, the observations in this section complicate the claim that depressive prediction errors are inherently maladaptive or epistemically distorting. This is not to deny that they sometimes are, but only to note that this is not always the case.

5. Adaptive and Epistemically Calibrated Prediction Errors

Building on what has been said, this section develops a more fine-grained account of how PP explains interoception in depression, focusing on Barrett et al.'s account. It extends the critique that prediction errors may be adaptive and epistemically calibrated while laying groundwork for the later discussion of psychosis.

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Barrett et al. invoke allostasis to situate depression within a PP framework centered on disruptions in interoceptive regulation [25]. Whereas homeostasis refers to the maintenance of relatively fixed physiological set points (e.g., sodium balance), allostasis concerns regulatory processes with variable optimal ranges. In many species, animals undergo allostatic seasonal changes—winter torpor to conserve energy, brown fat growth to support thermogenesis [204,205].

In humans, these adjustments may occur as an evolutionary carryover, once adaptive but less so in settings with central heating, warm clothing and building insulation. But if, in addition to this, brain-generated predictions misrepresent the environment as colder and more energetically demanding than it is, the result may approach a mild hallucination in PP terms [15,34,152–155,159]. Individuals may neurotically engage in energy-conserving behaviors such as binge eating, huddling under blankets and oversleeping, thereby withdrawing from daily activities.

These tendencies are among some of the depressive symptoms listed in the *DSM-5-TR*, illustrating Barrett et al.'s proposal that the condition arises from the brain generating maladaptive predictive models that misallocate metabolic resources [25]. Barrett's research team, however, neglects several considerations.

First, trauma exposure is a well-established contributor to depressive trajectories and is particularly salient in pathways conferring elevated psychosis risk [206–216]; yet this factor receives comparatively limited attention in allostatic predictive accounts despite its relevance to normative judgments about whether depressive responses reflect dysfunction or context-sensitive adaptation.

Second, depressive presentations may sometimes just be energy-depleting physiological processes. For example, inflammatory responses and sickness behavior are associated with energy conservation [217–221]. Older adults often show shifts in thermoregulatory preference toward warmer environments [222–225], and sleep more than younger people [215–217]. Though depression is heterogeneous in frequency and etiology among the elderly, research suggests high rates in certain cohorts, such as those in care facilities [226–239]. If these behavioral changes are based on predictive models, then the errors are minimal. Actions such as warmth-seeking are also adaptive and epistemically attuned to individuals' physiological conditions and do not appear to involve significant prediction error.

A third consideration neglected by Barrett and colleagues is that temperamental dispositions that appear maladaptive in one context may be advantageous in another. This suggests that symptom expression may reflect environmental mismatch rather than intrinsic dysfunction.

To elaborate, bold fish have advantages when encountering novel food, unless it is in a baited trap, in which case their more wary counterparts fare better [240]. While one might object that an optimized fish would update predictive models according to the situation, this neglects that bold fish may not survive a first encounter with a trap, rendering revision moot. For a human example, considered how neuroticism correlates with creativity and intelligence in anxiety-prone individuals [241,242], productive in some contexts. Similarly, certain forms of autism can bestow advantages in employment fields requiring extreme attention to detail, systematizing, intense focus and bias avoidance as well as the ability to work in relatively solitary conditions [243–247]. While one does not want to therefore conclude that autism never meets the threshold for being a disorder, the notion that it always does involves a somewhat arbitrary normative imposition.

Barrett and colleagues' PP account of depression maintains that "anything which regulates (i.e., acts on) a system must contain an internal model of that system" [25] (p. 2). Yet the claim faces counterexamples: brainless slime mold regulate their internal milieu and solve complex environmental problems [248], presumably without representations. Old work in artificial intelligence suggests the environment itself can serve as a more efficient model than internal representations [249], with the idea also appearing across various embodied accounts of mind [56–59,143,250].

Despite these considerations, PP accounts emphasize fine-grained neural regulation of bodily processes. In Barrett and colleagues' words, if "a senior colleague ... approaches your office door, … your brain will predict your colleague's arrival by constructing an embodied simulation," thereby

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"redirecting blood flow to the legs from other organs that need it less," computing "how much glucose is needed to stand up" [25] (p. 9). Aside from offering no supporting citations, Barrett et al.'s illustration raises several other concerns.

First, it neglects peripheral regulation, such as contraction-induced vasodilatation whereby muscle use mechanically draws blood into active tissue [251]. Second, if bodily thresholds are so finely tuned that merely standing up requires temporary redirection of blood from vital organs, jogging could be life-threatening. Third, PP accounts stress efficiency, yet it is debatable how economical it is for the brain to micromanage such processes. Fourth, when PP theorists offer evidence, it is often circumstantial. For instance, Smith et al. infer that depressed individuals underestimate metabolic resources based on reduced synchrony in heartbeat tapping tasks [19]. Extending this finding to a global misestimation of metabolic capacity, however, remains uncertain. Alternative interpretations, such as attentional disengagement from low-salience tasks among depressed individuals, remain plausible.

At the same time, Barrett et al. are correct that depression can exert deleterious physiological effects. Inflammation, while adaptive in acute healing contexts, can damage surrounding tissue when prolonged [25]; similarly, depression has been associated with structural and functional neural changes. One causal pathway is that depressive rumination involves sustained activation of the ventrolateral prefrontal cortex, potentially contributing to neural wear under chronic conditions [72]. This may relate to ventrolateral prefrontal dysfunction linked to heightened expectations of negative social evaluation [252], a pattern also emphasized in PP accounts [14].

These observations, however, do not decisively support the claim that depression constitutes an epistemic breakdown, or what PP accounts interpret as persistent gaps between expectations and available information [20,25,31]. Against this, depression may follow from the accurate recognition that circumstances fail to meet optimistic aspirations [183]. Cognitive impairment in depression may also result from mechanisms aimed at minimizing further harm, such as obsessing over a past trauma to avoid its recurrence. This does not deny that depression may warrant treatment. But the ill effects of rumination—including neural degradation—may be comparable to injuries sustained while fleeing danger: the intense contemplation and rapid retreat may be adaptive, even if the resulting brain or muscle injuries benefit from medical intervention [43].

Relatedly, depression frequently manifests as questioning rumination rather than delusional conviction. If rumination and associated physiological changes function to anticipate and avoid future adversity, then maintaining a discrepancy between feared outcomes and actual events may in some contexts be adaptive. In such cases, sustained mismatch between top-down anticipation and bottom-up events may reflect precautionary calibration rather than epistemic failure.

6. Prediction Error and Threat Avoidance in PTSD

PTSD can be profoundly impairing, and severe anxiety may disrupt cognitive integration. Without minimizing the burden, which may become maladaptive in safe environments, this section reviews how, under conditions of ongoing or potential threat, PTSD-related vigilance may serve a protective function. In such contexts, heightened arousal may increase sensitivity to recurrent but unpredictable threat and support adaptive self-preservation [178,253–256]. To the extent that this strategy mitigates danger, a discrepancy emerges between anticipated harms and comparatively benign outcomes. This scenario complicates PP formulations that equate PTSD pathology with prediction errors [27,30,32,145,257] and sometimes link these to hallucinations and other forms of psychosis [27,257].

To begin with, while anxiety is a PTSD marker [75,201,256], it can function as an affective signal with epistemic significance [258,259], with moderate levels improving task performance [260–263]. Because anxiety involves sustained anticipatory tension, it organizes experience into temporally extended patterns of concern [264], fostering forward-looking counterfactual modeling ("what-if" cognizing) [258]. As with fear, anxiety accordingly heightens sensitivity to potential harms. However,

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whereas fear typically targets an identifiable object or event, anxiety is often more diffuse, arising in response to ambiguous or unspecified threats [265].

Corticotropin-releasing hormone (CRH) regulates arousal and wakefulness [265,266]. Although it serves non-stress functions as well, prolonged increases in its activity can sustain anxiety and heighten responsiveness to threat cues [266]. Accordingly, elevated CRH in PTSD may be deemed "predictive" insofar as it enhances vigilance and sensitivity to potential danger [143]. Anxiety may likewise operate as a temporally extended affective mode modulating fear sensitivity and biasing information processing toward risk.

Within a PP framework, such bias can be operationalized as increased precision-weighting of threat-related priors, such that ambiguous or indeterminate stimuli are preferentially interpreted through danger-expectant generative models [8,44]. Sustained arousal can further amplify interoceptive prediction errors and stabilize higher-level threat anticipation, reinforcing vigilance and risk-sensitive behaviors [29,123]. Elevated anxiety is a core feature of PTSD, which may develop following sexual violence, combat exposure or severe accidents [267]. Neurobiological differences are frequently observed in affected individuals; however, it remains unclear whether these reflect trauma-induced alterations, pre-existing susceptibility or their interaction [38]. Increased CRH signaling has been linked to PTSD pathophysiology and may contribute to altered arousal regulation and heightened vigilance [268,269].

PP accounts of psychosis propose that aberrant precision-weighting—through overly precise priors or dysregulated prediction-error signaling—can yield hallucinations and persecutory ideation when threat expectations dominate perceptual inference [15,34,147]. In this light, PTSD-related hypervigilance and sustained threat anticipation may, under certain conditions, increase vulnerability to paranoia or other psychotic symptoms. This is by entrenching threat-biased generative models that reshape explore–exploit dynamics during chronic stress [27,30]. The proposal aligns with broader trauma-to-psychosis pathway models that identify post-traumatic intrusions, negative beliefs and threat appraisal as routes to paranoid interpretations [270,271].

Non-PP researchers also characterize PTSD accounts as involving a persistent and distorted sense of peril [272,273], and infrequent but consequential threats may exacerbate worries, impeding extinction when safety returns [81,274]. Still, context matters. What appears paranoid in ordinary circumstances—say, believing that world leaders and thousands of others are trying to kill you—may accurately reflect the plight of soldiers, as expressed by the protagonist in Joseph Heller's WWII novel *Catch-22* [275]. Along these lines, neurobiological work shows that defensive responses often scale with the perceived proximity and severity of danger [265,276]. Evolutionary models further suggest that, under asymmetric cost conditions, selection favors error patterns that err on the side of false positives when the cost of missing a real threat is high [253,277]. The resulting arousal is physiological as well as cognitive, as worry forecasts future peril, in some sense representing it [259,278–280]. By definition, the menace has not been actualized, yet it need not be fictive if it materializes or there is a reasonable expectation that it will.

However, there is a sense in which some PP proponents would deny the last claim even before considering PTSD. This is because they maintain that perception itself is a kind of hallucination—albeit one that functions well when it corresponds to practical environmental demands within a certain tolerance of error [3,25,33,168]. More neutrally, agents perceive predictive models that are constructed inside the brain rather than registering the world itself. In plainer language, they experience the brain's guesses or hypotheses about the world [1–3,25,33,168]. These models may track reality or fail to do so, in which case they are delusional or hallucinatory even in the conventional, non-PP sense.

For argument's sake, accept the PP claim that agents perceive brain-generated hypotheses rather than the world itself. This raises questions about what counts as pathologically delusional, and PP answers can depend on somewhat arbitrary normative assumptions. To elaborate, consider a variation of the earlier case of an individual with autism or AD(H)D whose expectation of a quiet room is violated by the faint sound of a tennis ball a in court many floors below. Paralleling this

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example, PTSD resulting from ongoing sexual abuse or war might lead one to register certain sounds—like faint metallic clicks or barely perceptible floor creaking—as contravening an "all-safe" predictive model [27].

As just stated, the same PP logic would construe hyperprecise models as causal factors in PTSD, autism and AD(H)D [12,13,27]. However, in the case of autism and AD(H)D, the reverse may hold: some brains are more sensitive to noise than others, and the fact that affected individuals are more disturbed by it may reflect an adaptive response that leads them to seek less distracting settings. For PTSD, non-PP literature lists many associated deficits, including diminished neural suppression of rapidly repeated auditory stimuli [281]. In PP terms, the situation would—as in the example above—follow from hyperprecise thresholds for sensory prediction errors [1–3,12,13,120,121]. The "hyper-" prefix insinuates pathologically if not delusionally precise priors. But this inserts a normative judgment about how brains should function—about the appropriate neurocomputational balance between tolerating more false alarms versus missing fewer dangerous cues.

This reconstruction exposes a tension within PP accounts. The question is under what environmental conditions sustained anticipatory models should be considered pathological rather than strategically calibrated. Instead of reflecting overly precise thresholds for sensory prediction errors, the lack of neural suppression may reflect greater gain assigned to low-salience but important cues. In high-risk environments, ordinarily negligible acoustic changes can signal imminent danger, and trauma survivors have reported heightened sensitivity to such cues [43]. Because intermittently occurring threats are difficult to definitively rule out, with sexual assault survivors frequently revictimized [71], trauma-exposed individuals may reasonably retain defensive priors even after leaving overtly dangerous settings. This generalized persistence can manifest as sustained anxiety and rapid threat reactivity, which may—yet need not—be maladaptive or delusional.

7. Normative Framings of Trauma in Predictive Processing

The evidence analyzed so far suggests that PP framings overstate the maladaptive aspects of psychological conditions, though the criticism also applies to other perspectives on mental health. PP accounts additionally include unacknowledged normative insertions—not only in how they label the maladaptive, but in the somewhat arbitrary adjudications of either too much or too little precision as epistemically distorting. Furthermore, PP interpretations can inadequately recognize how differing life circumstances should shape such determinations. This section elaborates on these points in the context of PTSD.

Now, Wilkinson et al.'s PP account of PTSD acknowledges aspects of some of what has been said so far. On the one hand, they argue that trauma organizes a hierarchically structured hypothesis centered on the life-threatening event [33]. Given its existential salience, the brain assigns high precision even to low-probability stimuli signaling recurrence, thereby eliciting traumatic re-experiencing [33]. However, flashbacks are usually not confused with present reality, as higher-level contextual beliefs about time and location remain operative [33].

On the other hand, it remains unclear that complex PP machinery is required to explain the familiar finding that trauma reminders trigger vivid recollection, and Wilkinson and colleagues provide little supporting evidence from memory or neuroscience research [33]. Moreover, schizophrenia and related psychotic-spectrum conditions often co-occur with PTSD [282]. As such, comorbidity complicates PP interpretations that attribute PTSD primarily to the non-extinction of danger-predicting priors.

To explain further, hallucinations are rare in PTSD unless sufferers also meet diagnostic criteria for schizophrenia [283]. Schizophrenia associates with unusual neurotransmitter activity [284,285], plus altered neural connectivity and synaptic signaling [286,287]. This is along with inflammatory, oxidative stress and immune processes [288–290], including microbial imbalances in the gut [291]. Evidence also suggests interactions between genetics and neurodevelopmental factors and environmental influences [292]. Just as a technology such as a radar can be rewired to produce atypical readings without predictive models or their corruption having any role, the nervous system

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may likewise generate anomalous signals without distorting priors. In short, PTSD-related hallucinations—if linked to schizophrenia—do not automatically imply predictive models gone awry.

This critique does not diminish the clinical seriousness of PTSD. For example, amygdalar hyperactivity and hippocampal shrinkage often accompany the condition [37,38,75,274,293]. Though distinguishing predisposing vulnerability from trauma-induced change remains challenging, prolonged stress hormone elevation links to glucocorticoid neurotoxicity and hippocampal atrophy [294].

That said, glucocorticoids also contribute to survival mobilization and heightened vigilance via adrenergic interactions [295]. To label such responses as necessarily "maladaptive" is misleading: it would be akin to judging a soldier's instinctive flight from a rolling barrage as pathological simply because it results in a sprained ankle. In both cases, the downstream injuries—whether stress-related neural atrophy or musculoskeletal strain—may warrant treatment. Yet this does not render the precipitating actions themselves as inherently disordered [43].

In sum, the symptomatology of PTSD—emotional numbing, detachment, avoidance, sleep problems, concentration difficulties, impaired inhibitory control and hypervigilance [296–299]—is frequently framed in normatively loaded terms. Hypervigilance implies excessive levels. But it may instead reflect reasonable caution; anxiety, avoidance and sustained wakefulness can be epistemically appropriate insofar as they support "what if" simulations of real but intermittent threats; moreover, emotional numbing and detachment may be functional, since fear can be paralyzing while extreme sympathy for an attacker or even a neighbor's children is usually unproductive when one is under assault. Concentration difficulties might be grasped as diverted attention in contexts such as warzones, where contemplating the literary works of Alejandra Pizarnik or Murasaki Shikibu may represent a misallocation of limited cognitive resources. Likewise, impaired inhibitory control may appear as increased reactivity in situations where slow reflection is costly.

Neuroimaging findings are broadly consistent with this interpretation. Individuals with PTSD often exhibit reduced activity in anterior cingulate and prefrontal regions relative to others [300]. These regions are implicated in higher-order attentional regulation, impulse control and deliberative decision-making.

Diminished activation may therefore indicate a shift away from reflective, strategic processing toward faster, more automatic responding [301], particularly when accompanied by heightened amygdala reactivity that biases the system toward reflexive emotional responses. Contrary to standard assumptions, such profiles may confer practical advantages under conditions of extreme and unpredictable danger. In active combat zones, for example, it would be maladaptive—not prudent—for soldiers to pause and deliberate over whether a sudden explosion is fireworks rather than an attack [43].

It is worth reiterating that none of this denies that trauma can result in pathological outcomes. A war veteran who develops PTSD may experience a condition that unsettles her sense of reality and alters her relationships with others years after she has left the combat theater. Perhaps here most can agree that PTSD markers are maladaptive.

But the situation may be very different for a sexual abuse survivor, even after an extended period of safety. Research indicates that roughly two-thirds of sexual assault survivors report subsequent victimization [71]. For someone who feels she could not cope with the idea of another assault, it may be practically sensible to take even unlikely threats seriously to lower overall risk over time. Her world may come to feel saturated with threat, reshaping expectations and heightening vigilance.

Such an orientation need not be interpreted as delusional; rather, it may reflect context-sensitive calibration to lived danger—an affective stance aimed at guarding against unwarranted assumptions of safety [43].

PTSD may sometimes involve re-exposure—for instance, forming bonds with abusive partners [302]. In such circumstances, clinical assessment and therapeutic intervention may be appropriate. Still, renewed exposure is often difficult to avoid because men statistically constitute the primary source of interpersonal danger across numerous social environments, particularly for women [303].

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Although the data do not suggest that any given man is likely to perpetrate harm, a high cumulative risk is here highlighted.

In PP terms, increasing tolerance for false positives may reduce exposure to severe harm when the cost of a missed signal is high. Although a less guarded stance might not culminate in revictimization, this can only be known retrospectively, and trauma-shaped individuals may reasonably regard certain statistical risks as unacceptable.

Accordingly, norm-infused evaluations warrant caution—for example, characterizing PTSD as involving "perception of threats where none exist" [25] (p. 9), or celebrating a psychopharmaceutical for reducing "avoidance of trauma-related activity by week 10" [304] (p. 35). Likewise, the claim that PTSD involves assigning "more negative and threatening interpretations than [events] warrant" [33] (p. 7) may insufficiently acknowledge the existential weight of objectively hazardous circumstances. Finally, restricting diagnosis to cases in which alarm responses are demonstrably false would implausibly exclude individuals living in active combat zones or abusive domestic settings from a PTSD diagnoses, meaning the objection lacks credibility [43].

8. Hallucinated Perception or Constructed Realty?

Proponents often trace PP to eighteenth- and nineteenth-century work by Bayes and Helmholtz [1–3,7,12,153], though its lineage reaches back farther to early Modern representational theories of mind. In contemporary computational psychiatry and neurobiological models of psychosis, these commitments are formalized in Bayesian probabilistic frameworks that explain perceptual disturbance, delusion formation and aberrant salience attribution. In this context, proponents frequently describe perception as "controlled hallucination" [33] (p. 4) and state that "your brain is wired for delusion" [166] (p. 66). Less polemically, agents experience "the brain's best guess" rather than the world itself [168] (p. 1). On this view, the brain constructs "internal models of the body in the world," tested against incoming sensory evidence [25] (p. 1).

Writers working within PP often note that affectively primed expectations can shape perceptual decision-making—for instance, making individuals more likely to report seeing a gun when none is present [305,306]. In neurocomputational psychiatry, such findings are typically interpreted as reflecting shifts in precision-weighting or the influence of threat-related priors, mechanisms frequently discussed in relation to psychosis-spectrum and trauma-related conditions.

Consider an experiment conducted after the 2013 Boston Marathon bombing. The study recruited participants from a Boston university. Wormwood et al. report that elevated anger increased the likelihood of mistaking people holding neutral objects for being armed [307]. Although participants had unlimited response time, stimuli appeared for only 500 milliseconds. The resolution was also low enough to remain highly ambiguous even under extended viewing.

Nonetheless, two authors of the just-cited study—Barrett and Wormwood—state in another article that "neural 'guesses' largely shape what you see, hear, and otherwise perceive" [308] (para. 5). Discussing the shooting of harmless citizens, Barrett elsewhere adds: "Human brains are built for this sort of delusion, through the same process that produces daydreams and imagination" [166] (p. 236). This comes close to suggesting that psychosis-like processes are the norm, though on this account they need not always be harmful.

There is a variant of Wormwood et al.'s paradigm that presents participants with non-ambiguous images but imposes strict time limits on identification [309–311]. Misattributions of guns again occur. But Baumann and DeSteno found that when participants were later asked—outside the rapid-response window—to restate their answers, error rates fell to near zero [310].

The response pattern may therefore reflect transient inhibitory-control constraints under speeded conditions rather than predictive aberrations leading people to see what is not there. Comparable effects occur with neutral stimuli. For example, a common neuropsychological test requires participants to press a button for frequent target letters and withhold responses to rare non-targets, with the procedure measuring impulse control instead of perceptual distortion [312–316].

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The cited studies on threat assessment therefore do not show that top-down priors directly rewrite perceptual content at the level of early sensory processing. Rather, they more plausibly indicate that negatively valenced affective states modulate downstream response selection, decision thresholds and action tendencies, which may or may not be driven by predictive models.

Prior beliefs and emotions may, however, still alter perception in ways that do not involve predictive processing. With depression—to repeat an example—the world is not merely seen (or hallucinated) as more onerous; it can become so because fatigue, inflammation or psychomotor slowing reduce a sufferer's capacity to act, making goals objectively harder to attain [181,317–320]. In this sense, depression can be understood as a bodily enactment of a more effortful environment [321]. Avoidance of social gatherings may thin networks of support, just as persistent expectations of failure may be partly realized when reduced motivation leads individuals to avoid demanding tasks [322–326].

Comparable dynamics are evident in PTSD as well, where chronic irritability or defensive aggression [326–330] can provoke hostility in others, thereby generating a social environment that both reflects and amplifies the individual's symptoms. Such feedback loops may stabilize threat-related expectations or interpretations that overlap with mechanisms often discussed in psychosis research [331].

Thus, altered affect is not merely an internal disturbance but a way of engaging the world that actively shapes the field of possibilities available to the agent. At one level, PP addresses this through active inference, whereby behavior brings about environmental changes and sensory inputs that align with an organism's predictive models [122,123]. On this view, organisms do not simply "imagine" a world—they help structure the conditions that confirm their expectations.

Even so, a broadly hallucinatory emphasis continues to run through many PP formulations, including those stressing active inference, such as Barrett et al.'s version [25]. In fact, PP can have a neurocomputational flavor that mirrors *The Matrix*, where characters mistake an AI-governed dream world for reality. This comparison is based not only on the PP claim that perception is a "controlled hallucination" [25,33,168]. It also rests on PP assertions that mental representations can directly generate bodily harm [25,332,333], a premise in *The Matrix* as well.

Delusional paranoia provides a straightforward case in which brain-generated representations appear to produce bodily harm, since hostile suspicion toward others may provoke retaliatory violence. Barrett and colleagues [25], along with other PP theorists [332,333], also advance an active inference account according to which the brain deploys energy-dysregulating priors. This generates internal sensory inputs (fatigue, agitation, etc.) that correspond to the brain's predictive model.

To elaborate, a subset of patients with depression and comorbid conditions such as chronic fatigue syndrome or fibromyalgia exhibit low cortisol levels [334,335]. A PP framing might interpret this as the result of the brain chronically anticipating sleep—for example, through high precision thresholds that assign significant weight to even slight energy depletion. This, in turn, could reduce cortisol, which typically decreases as one becomes drowsy [336–338]. As a result, feelings of exhaustion and related interoceptive signals would align with—and be reinforced by—the predictive model that simultaneously elicits them. The upshot, from this standpoint, is that neurally instantiated expectations can give rise to genuine somatic pathology [25,332,333].

Once more, however, causality may run the other way, as inflammation, sickness, fatigue and immune activity shape brain function [181]. This was already noted in earlier work on gastrointestinal and starvation-linked depression [73]. These cases also bear on atypical depression, where irritable bowel syndrome is frequently comorbid [335,339]. The condition itself is exhausting, and cortisol may decline partly to promote rest. If perception is accordingly altered, with fatigue shown to make distances appear greater [160–164,340,341], this does not vindicate PP claims that the brain is wired for delusion or controlled hallucination. Instead, agents may be accurately calibrating to the reality that environments and tasks are objectively harder to navigate [143].

Comparable examples abound. Sad individuals show reduced exploratory impulses and a preference for enclosed spaces [342], which aligns with their vulnerability. Anxious participants in wall climbing tasks reach cautiously and less far, such that intervals between handholds appear

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larger [343,344]. People exhausted by financial strain likewise register stairs as steeper, coinciding with the fact that they are harder to climb [345]. In such cases, there appears to be little prediction error, since perception reflects an attunement of bodily conditions to environmental challenges.

According to this action-based standpoint, even depressive torpor, binge eating and inflammatory responses can be adaptive. For example, if winter in 10th century Iceland prevented significant outdoor work, an inflammatory slowdown might increase sleep, preserving calories, especially when combined with heavy eating, building fat and aiding thermoregulation [143,180,346–348].

In predictive terms there would here be little discrepancy between top-down expectations and bottom-up inputs for seasonally deflated tenth-century Icelanders. The agent's reduced activity would fit winter ecologies and interoceptive signals of lowered vitality, consistent with accounts of interoceptive predictive regulation.

Moreover, because demanding productivity standards were unlikely to structure daily life in that context, lethargy might not be experienced as pathological. The critique nonetheless partly vindicates PP accounts: what renders seasonal low mood a "disorder" today may be the widening gap between normative expectations of sustained output and the diminished capacities winter brings.

9. Predictive Processing and Schizophrenia

Schizophrenia, which may comprise a cluster of different brain conditions with similar symptom profiles [349–351], is often treated as a paradigmatic case of psychosis, given its association with hallucinations and paranoid delusions. Since general PP accounts of these phenomena have already been reviewed, this section focuses on a more specific proposal: that schizophrenia arises from impaired prediction of the detectable consequences of one's own actions [39,157,352–354]. On this view, if individuals have difficulty anticipating their own inner speech or behavior as self-generated, they may experience auditory hallucinations or perceive themselves as being taken over by outside forces [355].

Aside from the fact that brain imaging does not directly reveal predictive models and other computational structures [76–79,356–360], the PP accounts outlined above face another obstacle: they rely on Westernized norms about self and agency, even though both vary significantly and often in complex ways across and within cultures [361–395].

For example, one study examining 46,503 individuals across 33 countries indicates that low agency predicts mental illness in Western contexts but not in East Asia [390]. There, people report greater social interdependence, aligning with regional values that emphasize adapting oneself to specific situations to promote harmony [361,395]. By contrast, Americans often idealize agency and report higher levels of it [43,390].

Still, assuming for the sake of argument that basic PP assertions about the self and schizophrenia are correct, one might expect rates to be higher in regions reporting greater interdependency and less agency.

Although some data indicate, for example, that Japanese schizophrenia rates are high, most of Asia shows lower rates than the US [396–398]. However, a 2026 systematic review and meta-analysis of 60 articles—sampling 20,910,871 individuals—reports very low rates in Japan, China, South Korea and India—together about four times less than in the US [399]. Across all the above cited data sources and comparatively speaking, sub-Saharan rates are also modest [396–399].

These observations do not necessarily preclude PP explanations; yet they do raise a question about whether PP framings apply locally rather than globally.

The rest of this section and the next one pursue this question, first by providing more detail on how PP proponents link schizophrenia to aberrant models of the self and agency; second, by offering a review of cultural variation in self and agency that highlights key nuances; and third, by considering potentially confounding variables, such as family socioeconomic status at birth, which correlates inversely with both agency and schizophrenia [400–404].

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PP theorists argue that the sense of agency depends on individuals correctly predicting what their actions will feel and look like. If the brain does not correctly anticipate perceptual inputs resulting from its own decisions, then self-generated stimuli—whether internal or environmental—are not properly "tagged" as one's own [15,153,157,352,353].

Accordingly, PP accounts of schizophrenia treat disturbances of agency and the sense of self as failures of predictive inference, particularly in how the brain anticipates the consequences of its own actions. [15,153,353,405]. As a result, internally generated signals (like inner speech) can be experienced as external (e.g., hearing voices), or one's own movements may feel controlled by something else [157]. PP theorists also argue that aberrant prediction errors can drive maladaptive belief updating, helping to stabilize delusional interpretations of anomalous experiences [34,147].

Computational PP work proposes that part of the problem may be timing: if predictive signals are delayed, the system cannot properly match actions with their outcomes [157,351,352]. This can produce both reduced and exaggerated senses of control: patients may feel that their actions are controlled by others, or conversely that they caused unrelated events [39,354]. This also fits broader PP accounts of hallucination that emphasize the overweighting of priors in perception [148,159].

Several PP models also describe schizophrenia as a disruption of the brain's self-model [15,153,353,406]. When precision-weighting and sensorimotor integration break down, the boundary between self and world becomes unstable, leading to forms of disembodiment [406]. At the same time, aberrant salience—driven by misweighted prediction errors—makes irrelevant internal or external signals seem unusually important [15,21,34,147]. This distorts judgments about what caused an event, including whether it was self-generated, contributing not only to delusions and hallucinations but also to disturbances of agency (e.g., thought insertion, delusions of control) [21].

Rossetti and colleagues, in a review, discuss how people with schizophrenia sometimes feel too little control over their own actions and sometimes too much [355]. They explain this by arguing that the brain's basic "this is my action" signal is often weak, leading to under-attribution (failing to recognize one's own actions). But in some situations strong higher-level expectations can override this and produce over-attribution (mistakenly feeling in control), consistent with PP accounts emphasizing imprecise sensorimotor predictions and overly strong higher-level priors [8–10]. Overall, Rossetti's team maintains that the core problem is this weakened low-level self-prediction, with over-attribution arising as a secondary effect.

In a follow-up experimental study, Rossetti's team empirically examined their position. They did so by testing intentional binding effects, here defined as the subjective temporal compression between a voluntary action and its sensory consequence, with shorter perceived intervals taken to indicate higher agency. Patients with schizophrenia showed reduced or absent binding, which Rossetti et al. interpret as a weakened link between action and outcome, thus a diminished sense of agency [39].

Now, a person could experience lower agency and less time compression without necessarily having a distorted predictive model, meaning Rossetti et al. may infer too much from their data. Other PP theorists do similarly, often using suggestive rephrasings. For example, the sense of self becomes predictive models of the self [354,407]; object recognition is reframed as perceiving according to a prior model [2,3,5–8,12]; neural signals become predictive signals [354,408]; and cognitive expectations are recast as controlled hallucinations, with sensory input primarily registered when it deviates from what the brain anticipates [2,3,5–8,12,121].

Accordingly, some caution is warranted in adjudicating these claims, though the tendencies just noted do not on their own disqualify PP accounts, including those of Rossetti's team.

That said, recent work on intentional binding presents complications that Rossetti et al. [39,355] do not consider. For instance, sexual arousal and depressive traits diminish the effect [409–411]. The latter may reflect reduced control due to exhaustion, without necessarily implicating an aberrant predictive model. Depression is comorbid with schizophrenia [412,413], and Rossetti and colleagues' results [39] could therefore be partly an artifact of this, without substantiating PP interpretations.

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A second issue with Rossetti et al.'s study is that their sample is drawn from a hospital in Italy, despite evidence that intentional binding and related phenomena vary across cultures.

One experiment comparing Western and non-Western participants (e.g., Canada, Western Europe and the USA vs. China, the Middle East and South Asia) found that Western participants showed stronger intentional binding for pleasant than for irritating outcomes, whereas this difference was absent among non-Western participants [414]. Among Chinese Tibetan and Uyghur participants, intentional binding increases when individuals are primed with culturally congruent cues, such as images of traditional clothing, suggesting that group identity can enhance volitional experience [415]. In another study, Austrian participants reported greater control when stimuli followed their actions quickly, whereas timing had little effect for Mongolian participants [416].

As already stated, some PP theorists link schizophrenia to aberrant models of self and agency. But this begs the question: aberrant from whose perspective? Also, if self and agency vary across cultures, should one expect corresponding shifts in schizophrenia rates? The next section considers these matters.

10. Predictive Processing, Culture and Psychosis

Although claims about culture often overgeneralize, neglecting internal heterogeneity and change [417], regional differences in agency and self-construal are well documented. These patterns matter for PP accounts linking schizophrenia to aberrant predictions about self-referential information [157,352–354], especially in relation to Rossetti et al.'s work [39].

Rossetti's team, as noted, proposes aberrant predictive models of agency as contributing to schizophrenia [39]. However, agency is complex. It can correlate inversely with interdependent self-construal [390]. Yet independent and interdependent self-understandings can also correlate positively [418], with the former associated with higher agency [390].

Additionally, there are different kinds of agency. Participants across cultures tend to prefer primary over secondary agency—that is, exerting control rather than adapting to situational constraints. Nonetheless, secondary agency is more strongly endorsed by Japanese and Japanese Canadians than by European Canadians [395].

These results already put pressure on Rossetti et al.'s position. As noted, low agency does not predict mental illness in Japan [390], with recent work indicating low schizophrenia rates there [399]. Additionally, agency—whether measured through intentional binding or other paradigms—varies across cultures [390–395,414–416]. Rossetti's team therefore makes a substantial inferential leap when generalizing from a small Italian sample [39].

A central caution in cultural psychology is to resist strict either–or binaries. Although interdependence is more prevalent outside the West, this does not imply the absence of independence. Ubuntu perspectives in Africa, for example, frame the self as highly relational, with some aspects of privacy distributed across a village rather than confined to individuals—similar to how some matters are shared within Western families but not beyond them [376]. However, rural South African women may still value individual autonomy while pursuing it through peer involvement and social relations that sustain relational self-experiences [419].

Also challenging simple binaries, Latin Americans often combine interdependence with high levels of individual self-expression and assertiveness [371,372]. Psychological well-being in these contexts may not map straightforwardly onto agency. It may instead relate to how successfully individuals translate factors like agency and socioeconomic resources into culturally valued ways of living [420].

In Amerindian historical contexts, the self has often extended beyond the human to include animals, spirits and landscapes as persons within familiar genealogies [366,367,374,378,421–425]. Nonetheless, personal autonomy persists [426], often scaffolded by nomadic histories [427] and flexible social structures [426]. For some of Australia's Aboriginal peoples, the self is embedded in the land as an agentive meshwork of ancestors, places and non-human beings [428]. "Sentient

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ancestors transform themselves into 'objects' such as hills and boards, which then become the fixed points by which Aboriginal people individuate themselves as mature subjects" [428] (p. 73).

Work on South and East Asia suggests the self is often interdependent, yet contextual signals can shift this, with Eastern or Western cues priming people, respectively [362,365,386]. These effects are stronger among younger participants [362,386]. Other data indicate that Americans skew toward independence and Japanese toward interdependence; these categories correlate inversely in those cultures but are not dichotomous, as independence and interdependence correlate positively for Chinese participants [418].

Further challenging the individual–collective binary, Korean and Chinese students value group work more than Westerners yet appreciating it for exposure to diverse opinions; for similar reasons, they also prefer class discussion [383]. An additional consideration is that ample within-region variation occurs. For example, interdependence and independence are higher, respectively, in historical rice versus wheat farming regions in China [429]. Rice cultivation requires greater coordination than wheat, fostering more interdependent self-construals [429,430].

Evidence suggests a broad range of culturally based cognitive and neural variation, including differences in perception [388,431–442]. Even apart from mental illness, these empirical observations should interest PP advocates, since—if the theory holds—the findings point to culturally inflected priors. Moreover, the data bear on PP accounts of schizophrenia, as the remainder of this section shows.

To consider one pertinent case, experiments show that Westerners tend to focus on foreground objects, whereas East Asian attention is more holistically distributed across contexts [432,438]. Greater gray matter volume in regions supporting this processing predicts interdependent self-construals [441], consistent with such dispositions fostering a sense of extending into the world. However, while the authors take this to show that "culture goes deep under the skin and is eventually 'embrained'" [441] (p. 8)—a view PP theorists would treat as consistent with priors—another conclusion follows: environments scaffold brains, psychic activity and perhaps predictive models as well.

This interpretation bears on PP accounts of schizophrenia but explaining it takes several steps. First, the idea of an environmentally scaffolded brain is reinforced by neuroimaging of the medial prefrontal cortex (MPFC) and anterior cingulate cortex (ACC), both associated with self-processing. For Chinese participants, who are typically more interrelationally disposed than Westerners, thinking about either their mothers or themselves activates these regions. In contrast, Americans show this pattern only when reflecting on themselves [370,436].

Second, schizophrenia is associated with atypical MPFC and ACC functioning, suggesting that symptomatic voice hallucinations may arise from individuals generating dialogue without recognizing it as self-produced [443]. Here, culturally inflected selfhood appears to moderate the condition. Luhrmann et al. found that Americans often perceive schizophrenic voices as intrusive, whereas Ghanaians and Indians more frequently report them as friendly or playful [41]. The American experience may partly reflect individualistic dispositions, especially valued in the US, which can make voices feel violating. This characterization echoes Sadler, a psychiatrist who explains that patients often encounter mental illness as an "ego-alien" takeover that "envelops the person" and "penetrate[s] her inner being, her personal self" [444] (p. 115). The invasiveness Sadler describes is typical of the schizophrenic distress encountered by Americans, less so in Luhrmann and colleagues' inter-African interviews.

Third, these outcomes contrast with what PP theorists focused on the self would typically expect. As a caveat, factors beyond culture and experiences of self can influence schizophrenia rates and shape its course. Stress, for example, is reciprocally related to schizophrenia: patients are more prone to it, and it can exacerbate or trigger the condition [445–449]. Conversely, rates often—though not always—decrease among rural populations [40,450–452], and shamanistic training in contexts where the practice is accepted may reduce toxic identification with symptoms [42].

Still, as noted earlier, some Africans value autonomy yet pursue it through peer involvement that supports relational forms of selfhood [407]. In such cases—and in bbuntu contexts, where privacy

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is often defined between groups rather than individuals [376]—an interdependent sense of self may render voices less invasive. Greater intermingling of subjectivity brings self-as-other closer to the norm, reducing problematic alienation. This may mitigate what one of Luhrmann et al.'s interviewees described as "a hostile takeover of my mind" [42] (p. 451). To use Sadler's phrasing, a voice that "comes over" [444] (p. 115) a person may instead be experienced more like a shift in mood following a friend's arrival [43], as reported in Luhrmann's interviews [41].

Sadler has in fact spawned a literature too vast to cite fully. The gist is that mental illness, while distressing in itself, becomes more so through the felt loss of control, combined with internal incursions that leave patients uncertain about who they are, thereby threatening the sense of self [453–463]. Yet in some regions it is normal to experience oneself as different people across situations within the same day [464–466], and, as noted, both feelings of agency and the degree to which their loss is threatening varies across cultures [390–395]. Additionally, those with more independent forms of selfhood may find internal inconsistency more disturbing, and it is more strongly associated with mental illness for them [467–471].

Taken together, these results raise overlooked possibilities for PP understandings of schizophrenia. Against Rossetti et al.'s interpretation [39], measures such as intentional binding may correlate positively or negatively with schizophrenia, depending on the enculturation of one's sense of self and personal control. Similarly, for PP accounts that attribute schizophrenia to predictive models that poorly distinguish self-generated from externally generated actions [157,352–355], the presented findings are significant insofar as situated forms of selfhood may already involve a reduced distinction between self and other—between individually and situationally generated actions.

A few considerations complicate the overall picture, including both PP theorizing about schizophrenia and proposed revisions to it. First, less agency may reflect lower socioeconomic status [402–404,472], which predicts schizophrenia in industrialized nations [400,401]. Second, schizophrenia and socioeconomic status covary with other factors such as ethnicity in the US, which also predicts schizophrenia rates.

Black Americans, for example, report less agency than whites, along with lower socioeconomic status, higher interdependence (communal orientation) but roughly equal independence [473–476]. Socioeconomic status can itself be a medical liability [477]. Experiences such as racism can worsen difficulties, despite sometimes fostering resilience and other limited benefits [478–484]. Hence, although this group shows much higher schizophrenia rates when compared to whites in the US [485–490], disentangling the causes is challenging. Disenfranchisement may reduce socioeconomic status, which increases schizophrenia risk. At the same time, evidence suggests clinicians may too readily dismiss justified grievances about discrimination as paranoid delusions, contributing to higher diagnosis rates [491–494].

Consistent with this, additional evidence suggests that affective conditions such as depression are often misdiagnosed as schizophrenia in Black patients [493,495–498]. Here, distress may link to persistent racism that constrains agency and socioeconomic advancement; situational depression is then misdiagnosed as schizophrenia, with grievances dismissed as delusional.

Even if schizophrenia rates remain higher after controlling for misdiagnosis, and even if Rossetti et al.'s agency measure [39] proves valid for Black populations, this could still largely reflect other factors. For example, socioeconomic status could be key, since it affects agency and predicts schizophrenia [400–404,472]. This interpretation could help explain why Canada records roughly half the schizophrenia rates of the US [396,397,399], which also reports greater poverty [499–502].

Concurrently, research suggests Canadians show lower in-group interdependence and higher independence, as well as a stronger metapersonal self [503]. The metapersonal self extends beyond the individual and immediate social relationships to include all people, living things, or even the universe [504,505]. The founders of the concept explicitly compare it to Buddhist, Vedic and Indigenous American views [504,505]. This connects to the earlier point that when a less bounded self is culturally normative, schizophrenia symptoms such as voice hallucinations may lose some of their threatening bite.

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Revising Rossetti et al.'s PP position [39], one might say that cultural congruence—rather than absolute agency and self-construal per se—is the relevant factor. At the same time, PP may not be required to account for relationships between agency, self-concept and schizophrenia, as they can and have been explained without it [41–43].

11. Conclusions

This review has argued that PP accounts of mental illness often overstate the extent to which atypical cognition should be understood as epistemically distorted or computationally maladaptive. Various cases support this position. The upshot of most of these scenarios is that anticipating bad outcomes to avert them is often desirable, even though it generates significant prediction errors.

A second major theme has been that these PP interpretations frequently rely on tacit normative assumptions. Judgments about what counts as too much vigilance, too little agency or excessively threat-sensitive perception are not value-neutral. They depend on background assumptions about which environments are normal, what forms of selfhood are desirable and how much false alarm is acceptable in conditions of uncertainty.

These assumptions become especially visible in trauma-related conditions, where the anticipation of danger may be reasonable. They are also evident in schizophrenia research, where Western conceptions of bounded selfhood and individual agency are too often treated as universal baselines. Once cultural and situational variation are taken seriously, claims about aberrant prediction become harder to sustain in any simple or globally uniform way.

The review has also suggested that some phenomena attributed by PP to maladaptive brain-generated models may instead arise from organism–environment dynamics, bodily strain or socially structured forms of vulnerability. Depression, for example, can result from inflammation, exhaustion or adversity that makes the world genuinely more effortful, rather than from distorted internal modeling alone. Similarly, PTSD-related vigilance may preserve safety precisely by sustaining a mismatch between anticipated and actual outcomes.

In such cases, prediction error may be a marker of adaptive success rather than of neurocomputational failure. More broadly, these considerations support greater engagement between PP and ecological, enactive, phenomenological and pragmatist approaches, all of which place emphasis on action, embodiment and environmental context.

None of this shows that PP is without value, nor does it deny that depression, PTSD and schizophrenia can involve serious impairment, suffering and maladaptive trajectories. The more limited conclusion is that PP-based computational psychiatry should proceed with greater conceptual caution, empirical restraint and normative self-awareness.

If atypical outlooks can sometimes be epistemically warranted, culturally congruent or adaptively protective, then psychiatric theory should be slower to equate deviation from statistical or neurocognitive norms with disorder.

Future work in computational psychiatry and translational neuroscience will therefore be stronger when it more carefully distinguishes dysfunction from adaptation. Among other things, this means distinguishing inaccurate prediction from prudent anticipation, and universal mechanisms from culturally local ideals of mindedness.

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