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Linking the Past to the Future by Predictive Processing: Implications for Psychopathology

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Most theories of psychopathology have focused on etiology at a specific level (e.g., genetic, neurobiological, psychological, or environmental) to explain specific symptoms or disorders. A few biopsychosocial theories have provided explanations that attempt to integrate different levels and disorders to some extent. However, these theories lack a framework in which different levels of analysis are integrated and thus do not explain the mechanism by which etiological factors interact and perturb neurobiology which in turn leads to psychopathology. We propose that predictive processing (PP), which originated in theoretical neurobiology literature, may provide a conceptually parsimonious and biologically plausible framework to achieve such integration. In PP, the human brain can be cast as implementing a generative model whose task is to minimize the surprise of sensory evidence by inferring its causes and actively controlling future sensory signals via action. This account offers a unifying model of perception, action, and emotion implicated in psychopathology. Furthermore, we show that PP can explain how different factors or levels result in psychopathology via updates of the generative model (the depth of the PP framework). Finally, we demonstrate the transdiagnostic appeal of PP by showing how perturbations within this framework can explain a broad range of psychopathology (the breadth of the PP framework), with a focus on bridging well-established psychosocial theories of psychopathology and PP.

General Scientific Summary

A useful theory of psychopathology explains the mechanism by which intra- and extra-individual etiological factors interact to influence neurobiology and psychopathology. The current article provides an overview of one such theory: predictive processing. Predictive processing is a theory of brain function that offers a unifying model of perception, action, and emotion underlying psychopathology, as well as a computational toolkit for testing hypotheses concerning how these factors can give rise to a broad range of psychopathology.

Keywords: active inference, Bayesian brain, computational modeling, predictive processing, theories of psychopathology

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Psychopathology is considered a function of internal and external forces, such as genetic and temperamental predispositions, biochemical fluctuations, as well as familial, social, and cultural factors (Kendler, 2019; Millon, 2009). Although there is no definitive conceptualization of mental disorder (Kendler, 2016), one useful definition states that psychopathology is the observable consequences of the above mentioned factors that cause harmful dysfunction or, more broadly, maladaptive functioning given an environment (Del Giudice, 2016; Wakefield, 1992).

Most psychopathological theories aim to explain specific symptoms, syndromes, or disorders by focusing on specific intra-individual explanations such as temperament/personality (e.g., neuroticism (Griffith et al., 2010)), behavior (e.g., learning theories [Bouton et al., 2001]), cognition (e.g., cognitive schemas and errors [Clark et al., 1999]), motivation (e.g., inhibition/activation system [Gray & Rowe, 2000]), and neurobiology (e.g., dopaminergic

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model [da Silva Alves et al., 2008]) or extra-individual explanations such as various types of stressors (Lippard & Nemeroff, 2020). Stress-diathesis (Ingram & Luxton, 2005; Monroe & Simons, 1991) and biopsychosocial (Engel & Engel, 1980) frameworks have attempted to integrate explanations across psychological, social, and biological factors. Although there have been mechanistic developments using these frameworks (e.g., how stressors influence the hypothalamic-pituitary-adrenal axis in emotional problems [Kudielka & Kirschbaum, 2005]), they remain insufficient in providing tools for specific and quantitative explanations of psychopathology (Kendler, 2019; McFall, Townsend, & Viken, 1995). In particular, there is often a lack of detailed information regarding quantitative mapping of (a) a hidden factor such as a core schema to the observable behaviors such as self-report of depression, within a particular domain of analysis and (b) mechanistic mapping of neurobiology onto psychopathology (Friston et al., 2017; Miller, 2010; Miller & Yee, 2015). In other words, biological, psychological, and social risk factors have all proved useful in understanding and predicting psychopathology, but the field lacks a unified quantitative foundation that explains how these factors interact over time to give rise to psychopathology (Kendler, 2019). Building (mathematically) formalized models can help tackle both problems. If an algorithmic model can generate measurable pathological outcomes from the hypothesized causal processes, then this model becomes a promising mechanistic explanation (Sharp & Eldar, 2019; Wang & Krystal, 2014) and suggests new targets for intervention and neurobiological investigations (Marr, 1982; Miller, 2010).

The predictive processing (PP) framework offers both a Bayesian account explaining how an individual interacts with the environment (Clark, 2013; Friston, 2010) and a concrete modeling toolkit allowing for an explicit examination of underlying processes (e.g., Schwartenbeck & Friston, 2016). Accordingly, PP—as a conceptual framework and a modeling toolkit—facilitates the integration of various levels of analysis within and across domains, and—as a theory of brain and behavior—bridges the gap between psychosocial and neural theories. In addition, born in theoretical neurobiology, this framework offers a plausible and testable account of neural implementation. An emergent property of PP is that it brings perception, action, emotion, and cognition-related processes into a unifying and continuous framework, fostering a holistic and integrative understanding of how these factors may interact to give rise to psychopathology. Originally developed to explain various aspects of general human functioning, its application to understanding a variety of pathological phenomena is in line with the current structural models of psychopathology showing that normal and abnormal personality can be treated within a single structural framework (Markon et al., 2005) and newer classification approaches emphasizing the continuity or dimensionality of psychopathology across the entire population (Kotov et al., 2017).

The Scope of the Present Study

The present study highlights the value of the PP framework in psychopathology research. Our aim is not to argue for an alternative to existing models; rather, it is to provide a conceptual framework through which predictions based on existing psychosocial theories can be explicitly examined and linked to neurobiological processes. Although this link is achieved primarily via formalization of computational processes, the technical details for achieving this are not within the purview of the present article. Rather, we hope to familiarize a broad audience with the conceptual foundations of PP and how it can be applied to understanding psychopathology and thereby encourage the pursuit of the subsequent formal modeling. This means that we will focus on how PP can help conceptualize and operationalize widely studied psychological constructs and facilitate their quantification but will not cover the actual algorithms carrying out this quantification, for which we refer the audience to recent tutorial papers (e.g., Friston et al., 2017; Sajid et al., 2021; Schwartenbeck & Friston, 2016; Smith et al., 2021; Smith, Parr, & Friston, 2019). As an example, we will discuss how threat sensitivity, a construct key to anxiety and phobia, can be conceptualized in PP as high precision for sensory signals carrying information regarding threat. Although this precision can be modeled as the inverse variance of a probability distribution over a set of possible discrete sensory and action states, the exact algorithms will not be covered.

In what follows, we first give a brief overview of PP to show how it provides a synergistic framework for explaining perception, action, cognition, and emotion. A considerable proportion of this paper is devoted to explaining the foundation of PP and how it can explain human functioning generally, but we view this as necessary to understanding the following content, which is a selective review of how psychopathology, including existing etiological theories, can be conceptualized in the PP framework. While there are a number of excellent reviews of the conceptual (e.g., Friston et al., 2014; Kube et al., 2020; Paulus et al., 2019; Smith et al., 2019), technical (e.g., Buckley et al., 2017; Linson, Parr, & Friston, 2020; Smith, Lane, Parr, & Friston, 2019; Smith, Parr, & Friston, 2019), as well as neuroscientific bases of PP (Friston et al., 2014; Smith, Badcock, & Friston, 2021), our aim is to show how more general models of psychopathology, spanning multiple levels of analysis and across diagnostic boundaries as well as social and psychological factors, can be understood through PP. We also highlight some future proposals of how the PP can provide a conceptual framework for specific pathological phenomena. We will conclude by discussing the value of PP in the context of computational psychiatry, and its current limitations.

The Predictive Processing Framework

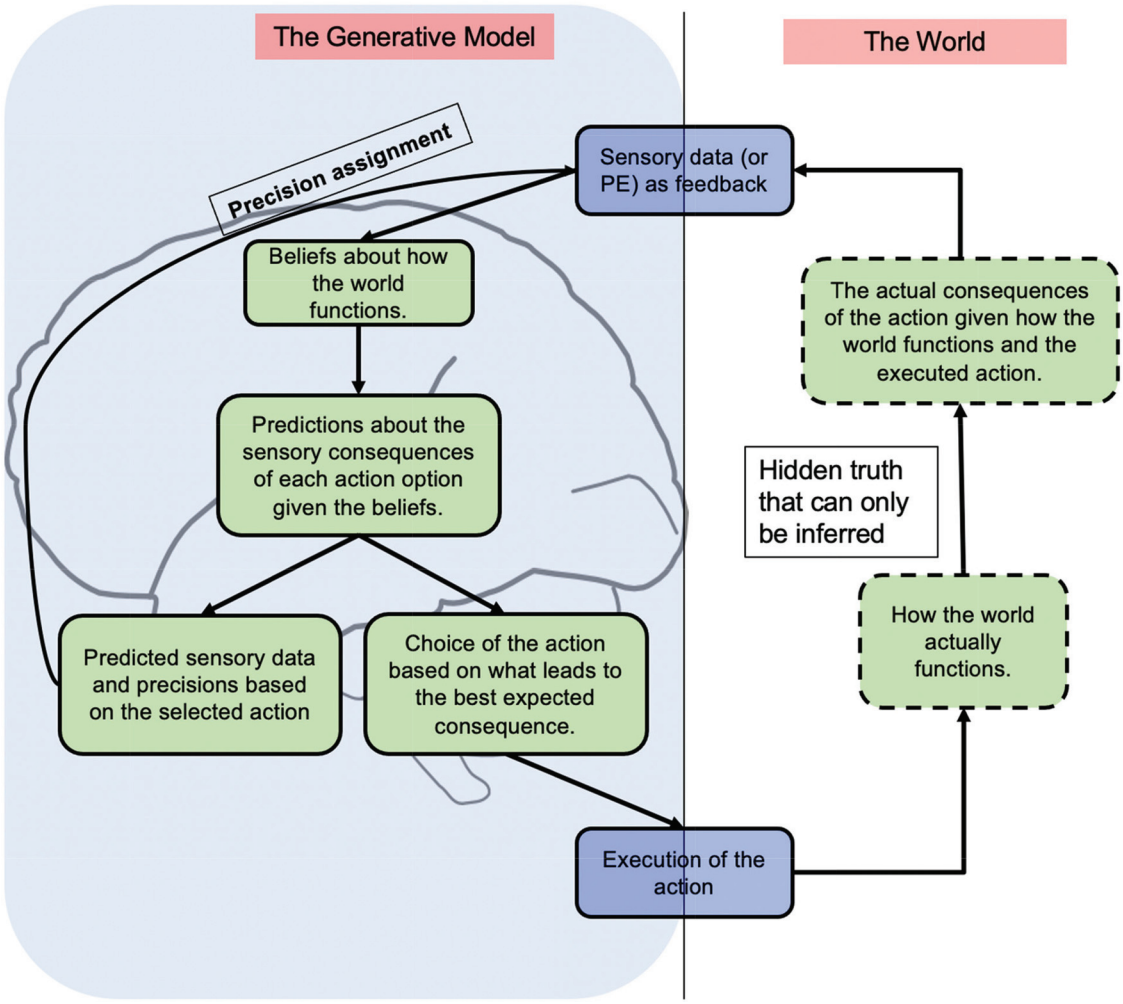
In research, we construct models based on existing knowledge and compare model-generated predictions against data to check and update our models. Similarly, PP proposes that the brain maintains a **generative model** predicting the sensory experience resulting from the organism's actions given the environment, and these predictions are tested against incoming sensory data (Friston, 2010; Friston et al., 2006; Gregory, 1980; Helmholtz, 1925; Powers, 1973). It is *generative* because it models not only the sensory data but also the causal processes that yielded those data. Essentially, a generative model carries the beliefs about how the world functions and predicts the consequences of interacting with the environment. This type of model is thought to be evolutionarily conserved because an organism can increase its odds of survival if it embodies a model of its interactions with the environment (Ashby, 1947; Conant & Ashby, 1970; Craik, 1943; Dayan et al., 1995; Friston et al., 2006).

Scientists using a computational approach based on the PP framework would construct a generative model simulating the

information processing given the specific experimental setting, which carries their explicit beliefs regarding how the observable data are generated from the hypothetical mechanisms. If a generative model constructed by scientists can yield observable data that match the empirical data, then they declare that there is good evidence supporting this model. If the observable data are measurements taken from human subjects while interacting with the environment (e.g., doing a task), then the scientist is constructing a generative model based on the scientist's theory of the subject's generative model of the world (Schwartenbeck & Friston, 2016).

Figure 1 illustrates how the generative model interacts with the world via the cycle of receiving sensory inputs from the world and executing action (output) to the world. The loop starts with the generative model encoding a set of beliefs about how the world functions. Based on these beliefs the generative model predicts what action will lead to what sensory consequences, and then chooses the action that will lead to the best expected consequence. Once the action is selected, the generative model also predicts what sensory consequences are expected upon executing the action. The generative model impacts the world by action execution. Now based on how the world *actually* functions, which is hidden from the generative model, the world generates *actual* consequences given the executed action. These consequences form the sensory feedback to the generative model. Importantly, not all actual consequences are received by the generative model faithfully; instead, consequences provided by the world are weighted by their precision, with higher precision translated into higher weight for the sensory signal. This sensory evidence either confirms or disconfirms the beliefs, updating the model. Perception and action are thus unified in this synergistic framework. The nature of this cycle can be captured by the title of William T. Powers' classic book on a conceptually similar framework—*Behavior: The Control of Perception* (Powers, 1973; also see the online supplemental materials). Naturally, human beings share commonalities and display individual differences in perception and action. Conceptually, genes encode natural preferences, and thus can be regarded as basis of the initial setup of the generative model. Life experiences provide constant feedback sculpting this

Figure 1
The Interaction Between the Generative Model and the World



Note. See the online article for the color version of this figure.

generative model, resulting in updated beliefs and updated predictions regarding future perception and action. Consequently, an individual's perception, emotion, cognition, and action at a given moment reflect the generative model cumulatively shaped by both nature and nurture.

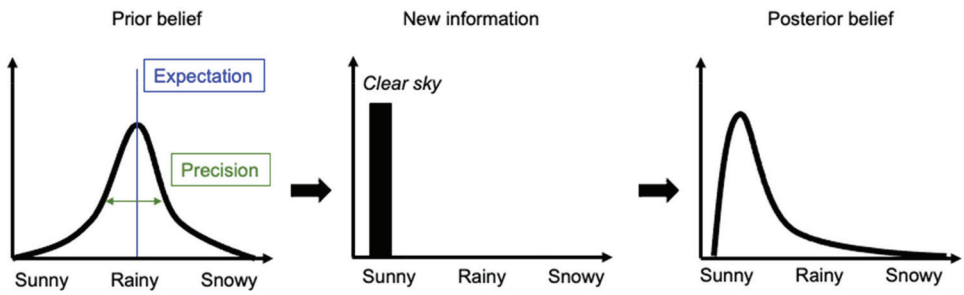
The Synergy of Perception and Action via Belief Updating

Free energy measures the uncertainty in a system (Friston et al., 2006; see the online supplemental materials for the mathematical definition). A central idea of PP is the **free energy principle**, which states that any self-organizing system (e.g., a sentient agent) will adapt so as to minimize its free energy (Friston, 2010; Friston et al., 2006). An organism conforming to this principle is capable of adapting to its environment by predicting the consequences of its interaction with the environment (Friston et al., 2006; Friston et al., 2013). To minimize **free energy** is to minimize the divergence between predicted and experienced sensory data in **perception** or predicted and preferred sensory states in **action selection**. Minimizing free energy is hence equivalent to maximizing the predictive power of the generative model maintained by the brain (Friston et al., 2006, 2013). Thus, adaptation becomes a process of constructing and updating the generative model.

Under the umbrella conceptual framework of PP, **predictive coding** theory often refers to the study of neurobiological processes via which the brain infers the causes of the sensory data (also see the online supplemental materials). Predictive coding theory describes the brain as a "prediction machine" that is constantly making **predictions** about its sensory input. These predictions are based on the **beliefs** (i.e., a probability distribution over a set of **hypotheses** about the world) that are encoded in the generative model regarding how the world functions at a given moment. These hypotheses are about what causes the sensory experience in perception and what sensory outcome will occur following a potential action. The most probable hypothesis is the **expectation**. At a given moment, we make predictions regarding sensory data (i.e., prior sensory predictions) based on (prior) expectations. At the next moment, if the sensory input differs from the predictions, a **prediction error** (PE, under simplifying assumptions, PE reflects an increase in free energy; Buckley et al., 2017) is generated, based on which we update our beliefs, yielding updated expectations (i.e., posterior **expectations**) about the world and updated predictions about the sensory input (i.e., posterior sensory predictions). This is akin to Bayesian belief updating or inference. Importantly, the generative model maintained by the brain encodes not only the beliefs, the sensory predictions, and PEs, but estimates about their "**precision**" or inverse variance. Whether we should update our beliefs and to what degree depends on precision (Clark, 2015; Friston et al., 2013). Precision reflects confidence regarding how sensory data are generated, with high precision indicating lower uncertainty and higher confidence. The precision afforded to a type of sensory evidence or PE can be conceptualized as describing its salience. The precision of a prior belief reflects how easy or hard it will be to update based on new sensory evidence or PE, and the consequence of the updated belief is the posterior belief. Hence, a more precise belief (e.g., I highly expect it will rain today) will need a more precise PE (e.g., a clear sky) to warrant being updated than a less precise PE (e.g., a cloudy sky; see Figure 2). If new sensory evidence or PE does not drive an update of the prior belief, then the posterior belief will be equivalent to the prior belief.

With some of the key terms described above (also see the online supplemental materials for a table of key terms), we can discuss how percept formation can be understood under the PP framework. The assumption of the PP framework is that the generative model conforms to the free energy principle, that is, to minimize free energy, as free energy reflects uncertainty (or lack of knowledge) about the world. We will next see how percept formation and action selection emerge naturally from this assumption. It is crucial to note that the generative model interacts with the world only via executing actions and receiving sensory inputs as the feedback (see Figure 1). The generative model implemented by the brain does not *know* the processes via which action leads to sensory feedback. Sensory data are the only new information to which the generative model has access. The brain must (actively) infer the cause of the sensory data for perception to occur. Let us take the example of someone who owns two cats: an orange tabby and a gray tabby. As the owner is working at her desk, her visual system senses an orange object on the table. Her brain does not

Figure 2
Example of Belief Updating



Note. A belief is a probability distribution over hypotheses. A belief with the lowest precision will be a flat prior distribution, conveying the information that every option is equally probable. Precision reflects how narrow the distribution is about the expectation. When novel information comes in, the belief distribution will be updated forming posterior distribution, with a posterior expectation and posterior precision. See the online article for the color version of this figure.

know that it is her orange tabby in front of her but can *infer* from the sensory data (orange sensation) that the orange tabby is the most likely explanation of what caused this sensory data. This "orange tabby" hypothesis is tested by matching the incoming sensory data against the predicted sensory data of her cat. To the extent that the hypothesis is accurate, that is, the predicted and incoming sensory data match, a percept is formed. Hence, the percept represents the posterior expectation of the cause of the sensory data. This explanation is in line with the widely accepted theory that visual perception is a constructive process in which the brain must actively infer the causes of the sensory data (Palmer, 1999). However, if the prior hypothesis is inaccurate, for example, "grey tabby," the mismatch between the predicted (gray sensation) and actual (orange) sensory data generates a PE. PE reflects an increase in free energy, and thus a system conforming to the free energy principle is motivated to minimize PE. The PE alerts the generative model that the gray tabby hypothesis is unlikely to explain the data, prompting the model to update the belief and generate updated predictions about the sensory data. By doing so, PE drives the updating of the belief about what causes the sensory data until PE is itself minimized and the pet owner accurately perceives her cats.

Whether and how much novel sensory evidence or PE can drive an update of the belief depends on the relative precision of the PE and the prior belief. In the example of the pet owner at her desk, if the owner thinks that her gray tabby is in another room, she has a strong (high precision) expectation of seeing her orange tabby. Imagine a case in which this is wrong, and her gray tabby is on the desk. Now, if her office is brightly lit, the generative model will allocate high precision to the sensory evidence of "greyness" given the high-quality physical environment, causing a strong PE that will update her belief of which tabby she is seeing. If her room is poorly lit, then her high-precision (wrong) expectation that her orange tabby is on the desk is likely to be retained, since the sensory evidence is allocated low precision in conveying the color of the cat.

Besides the signal quality, the precision of PE can also be modulated in a top-down manner by higher-order cognitive factors. This effect is exemplified by spatial attention. Imagine that the pet owner's orange tabby has a bed on the left of her desk. When she would like to find her cat, there is a strong (high precision) belief that the orange cat will appear in the left visual field rather than the right visual field. This belief will lead to a higher precision allocated to sensory data coming from the left compared with the right visual field, allowing sensory signals from the left to win the competition for higher-level cognitive resources (Feldman & Friston, 2010; Parr & Friston, 2019). This precision assignment describes the psychological phenomenon of "attending to the left." It also speaks to why the perceptual inference is active in PP. This is consistent with the classical findings of spatial attention that perceptual judgment is sharper for attended than for unattended locations.

Above we described how perception emerges from minimization of free energy. Another way free energy (or PE) can be minimized is by actively sampling the environment to bring the sensory signal more in line with the predictions (Clark, 2013; Friston et al., 2006). Hence, action selection can be regarded as the brain making inferences about what actions to execute to bring the self to a preferred (sensory) state (i.e., a state that minimizes free energy; Botvinick & Toussaint, 2012; Friston et al., 2013; Powers, 1973). In a familiar context, the generative model computes the free energy associated with each action option and selects the one that has the lowest free energy. In an unfamiliar context, the generative model lacks prior knowledge to make confident predictions about what actions can lead to what outcomes (sensory states such as hunger or satiation). In such a context, exploratory actions possess epistemic value due to the amount of knowledge that can be gained by executing these actions (i.e., the epistemic value reflects the amount of free energy that is expected to be reduced by executing the action). The value of an action can thus be quantified using free energy, consisting both of extrinsic value (reflecting the preference of the expected sensory state following the action) and epistemic value (reflecting the amount of knowledge about the environment that can be gained following the action). Thus, in a familiar context, the extrinsic value dominates the action selection, leading to more habit-driven behaviors, whereas, in an unfamiliar context, epistemic value may dominate the action selection, leading to more exploratory behaviors (Friston et al., 2013, 2015; Parr & Friston, 2017a; also see the online supplemental materials). Thus, one's confidence in their belief regarding the environment, that is, "whether I am in a familiar versus unfamiliar context" influences whether extrinsic or epistemic value dominates the action selection decision.

In summary, the generative model is concerned with the states of the world and predicts the various sensations and actions with the goal of minimizing free energy. By changing both the beliefs (and sensory predictions) and actions the model embodies a self-fulfilling loop, in which we are more likely to experience what we expect to experience (Powers, 1973). In addition to perception and attention, the PP framework has been used to explain a variety of higher-order cognitive functions including memory, conceptual knowledge, and language (Parr & Friston, 2017b; Spratling, 2016).

Predictive Processing Conceptualization of Emotion

There is a long history of contrasting theories regarding how conscious emotion arises. These theories range from viewing emotions as inferred from physiological arousal (Cannon, 1987; James, 1884) and based on contextual information (Schachter & Singer, 1962), to viewing them as being simultaneous with physiological arousal (Cannon, 1987), to viewing them as arising from cognitive appraisals (Lazarus & Folkman, 1984). More recently, conceptual and simulation studies have applied the PP framework, particularly the active inference scheme, to explain and formalize the process via which conscious emotions can be inferred from context-dependent sensory data (Hesp et al., 2019; Smith, Parr, & Friston, 2019). Sensory data can come from two sources, one generated from the internal physiological milieu of the body (interoceptive) and the other caused by external stimuli (exteroceptive and proprioceptive). Applications of PP to understanding emotion and mood have focused on perception of the internal bodily signals or interoception, for example, in the active interoceptive inference theory of emotion (Barrett et al., 2016; Seth & Friston, 2016). Conceptually, in this view, conscious emotion has been described as the posterior expectations (i.e., the best hypothesis) about the causes of interoceptive signals (Barrett et al., 2016; Seth & Friston, 2016; Smith, Lane et al., 2019).

Simulation studies have demonstrated that a conscious emotion or emotion awareness can emerge from a generative model that encodes expected interoceptive sensory data given a context (Hesp et al., 2019; Smith, Parr, & Friston, 2019). Accordingly, the final

conscious experience of an emotion is the inferred cause (e.g., fear) of the sensory data (e.g., increased heart rate) which is the same as the posterior expectation about what emotion caused the sensory data. Hence, "I feel fearful" because the generative model concludes that fear is the most likely emotion based on a good match between predicted interoceptive sensations under the hypothesis of fear (compared with happy or sadness etc.) and the actual interoceptive sensations. Whereas conscious emotion or "feeling" is perhaps the most widely studied aspect of emotion under PP, this framework can also explain other aspects of emotion, including the behavior involved in emotions. For example, the fear response of a mouse running away after spotting a cat has been successfully simulated using the active inference scheme (Linson & Friston, 2019).

Applying the Predictive Processing Framework to Psychopathology

So far, we have described how the PP framework can explain basic cognitive and emotional processes. Next, we will describe how biological, psychological, and environmental influences can contribute to the formation of dysfunctional generative models in the PP framework, ultimately giving rise to psychopathology (illustrated in Figure 3).

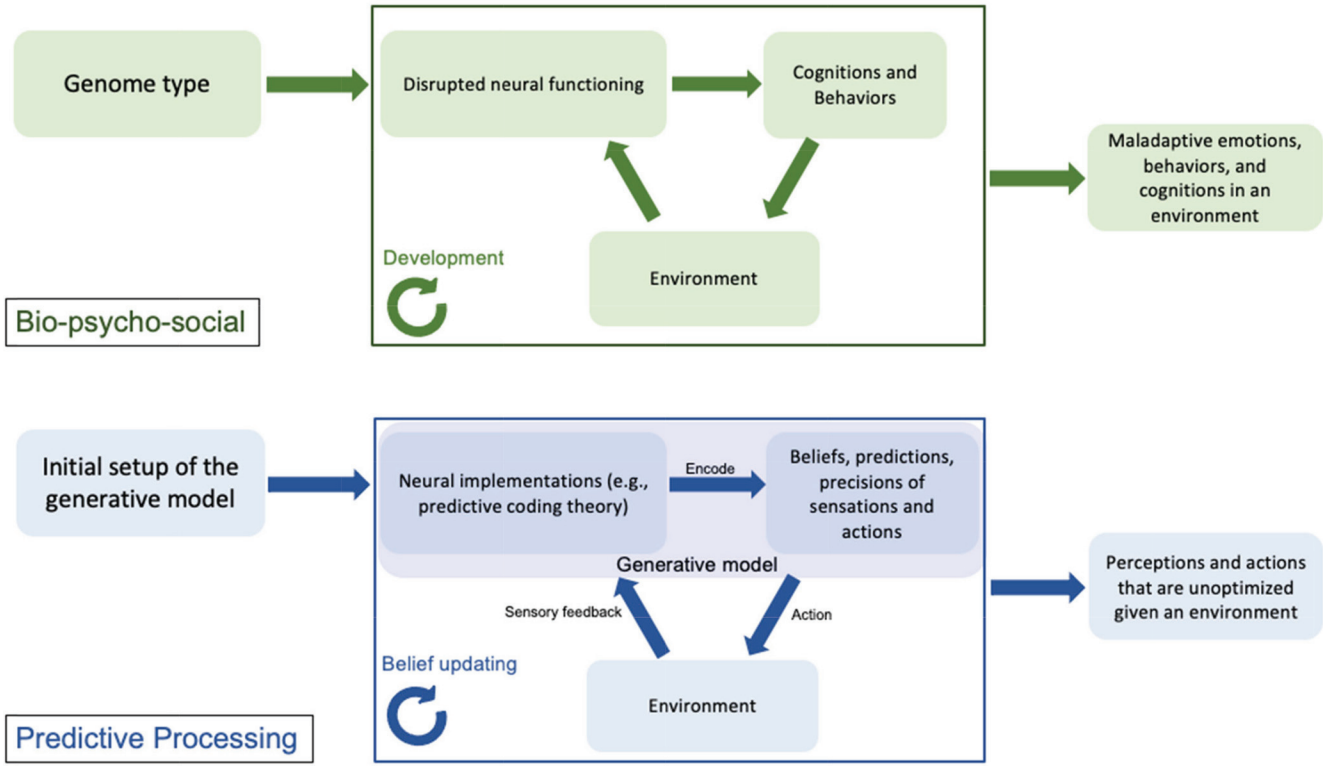
Biopsychosocial Factors Under the PP Framework

In the PP framework, both normal and abnormal cognitions, emotions, and behaviors are part of a process to *minimize* free energy. In line with current conceptualization of mental disorder being environment-dependent (APA, 2013; Wakefield, 1992), whether the perceptions and actions are adaptive or maladaptive depends on the context. For example, consuming a highly preferred food (an action with a higher extrinsic value) can be viewed as an action leading to a smaller free energy state than consuming less preferred food items (actions with lower extrinsic values). This preference is a function of inherited ancient prior beliefs (e.g., preference as a belief that consuming calorie-rich foods will reduce uncertainty about future hunger states and therefore lower free energy) that were adaptive during the paleolithic era, but in our current context increases the risk of obesity and hypertension.

Furthermore, PP allows belief updating at various temporal scales, from instantaneous (as in percept formation), to moderate (as in a change in emotional state), to long-term (as in habit

Figure 3

The Correspondence Between Biopsychosocial Framework and Predictive Processing Framework



Note. Genetic risk factors determine the initial generative model setup. At a given moment, the neural activity conceptualized under predictive coding maps onto the neural functioning (or endophenotype, such as activation in neural circuitry supporting threat anticipation) in the biopsychosocial model. The generative model encodes the beliefs about how the world functions, and generates predictions about sensations, actions, and precisions, governing what information is salient, what behaviors to take, and what sensory consequences can be experienced. This maps onto how neural activity manifests in cognitions and behaviors. For example, a belief about how the world functions maps onto the idea of cognitive schema. Via interacting with the world, the generative model updates its beliefs and predictions, mapping onto learning or development. Ultimately, pathological phenomena can be conceptualized as the output of a generative model that is unoptimized given an environment. See the online article for the color version of this figure.

formation or trait-level changes). Belief updating at a longer temporal scale constrains the belief updating at a shorter temporal scale, and belief updating at a shorter temporal scale can accumulatively lead to update at a longer temporal scale. Such a temporal hierarchical system makes it possible to conceptualize various factors such as trait and state level factors and neurodevelopmental processes (see the online supplemental materials) in psychopathology comprehensively.

Genes and Temperament

To our knowledge, there has been no direct mathematical formalization of how genetic and personality factors impact psychopathology under PP, although they are often implied in the model specification as prior preferences. Because they are pivotal in our understanding of psychopathology, in this section we attempt to explicitly outline some preliminary ideas of how PP can be used to study how genetic and temperamental influences give rise to psychopathology. Broadly, the influence of genes in the PP framework can be described in terms of initial layout of the generative model, encoding initial preferences shaped by evolution and inherited individual differences (Badcock et al., 2017; Friston et al., 2006). One way to incorporate this genetic influence can be in terms of inherited precisions that influence what the individual is sensitive to at the beginning of their life, which can be modified by subsequent life experiences. For example, family studies show that bipolar mood disorders share less genetic commonality with unipolar depression than with schizophrenia (Kendler et al., 2020). However, the endophenotypic mechanisms remain unclear. Using PP for computational phenotyping (Schwartenbeck & Friston, 2016), one can conceptualize how the genetic factor impacts precision assignment in a particular functional domain. For example, people who exhibit high precision in beliefs regarding what causes the sensory data may show proneness to psychotic symptoms (e.g., delusions), which are more common in bipolar and schizophrenia than unipolar depression (Cuellar et al., 2005; Goes et al., 2007). Different phenotypes may emerge from different levels of sensory precision. Such a conceptualization can be translated into a formal model. For example, researchers may recruit offspring of patients from all three types of disorders. Using model comparison, researchers may find that a generative model equipped with higher sensory precision fit the data best for offspring for both bipolar and schizophrenia, but not unipolar depression. Thus, the similarity and dissimilarity in genetic factor across the three forms of psychopathology may be mediated by nuanced precisions.

Closely related to genes is temperament and personality. Neuroticism is a reliable risk factor for internalizing disorders, and heritable (Krueger, 1999; Watson & Clark, 1984). One way neuroticism can be conceptualized is as a belief with a high probability that the world is unpredictable and threatening, and thus the generative model allocates a higher precision to sensory evidence carrying negative or threatening information compared with sensory evidence carrying safe or positive information. Such precision assignment will lead to a belief update driven largely by threatening information (more salient than positive or safe information), manifesting in hypersensitivity to stressors. Here again, one can use PP to construct explicit models (conceptually and formally) on how the temperament diathesis can impact sensitivity to external events, which in turn biases what information drives the update of the beliefs about the world.

Environment

Even if two individuals—identical twins, for example—have the same initial generative model, life experiences shape different trajectories (Badcock et al., 2017; Friston et al., 2006). Repeated stressful events can shape a generative model into one that is adaptive in a highly uncertain environment. Such a model may result in a self-fulfilling prophecy, in which prior beliefs about the world lead to a vicious cycle of allocating high precision to negative or punishing sensory evidence, allowing them to drive the belief updating. And because it is easier for the environmental factors to influence prior beliefs with lower precision than ones with higher precision, the PP framework can accommodate ideas such as clinical staging, in which the precision of maladaptive beliefs grow over time and become harder to change. PP is also consistent with the diathesis-stress model, in which symptoms to which an individual is especially prone to require less environmental stress to become pathological. For instance, if the genetic or temperamental diathesis can be seen as a generative model assigning abnormally high precision to sensory information about threat, then the individual will be more sensitive to potential threat. Consequently, the individual may process more threat-related information than safety-related information, resulting in pathological manifestations.

Finally, an early life environment is especially important in shaping beliefs about the world. In a nurturing childhood environment, the individual is more likely to develop a belief system that the world is predictable and controllable. In a nurturing environment, stress is temporary, and the individual can bring themselves to a preferred sensory state by actions, leading to adaptive behaviors in the face of challenges. In contrast, childhood adversity can be regarded as repeated or prolonged period in an unpreferred state (high free energy resistant to reduction). Such an environment can engender a strong (high-precision) belief characterized by that the world is uncontrollable and punishing, reflecting high uncertainty about what actions need to be executed to safeguard physical and mental well-being (Peters et al., 2017; Slavich & Irwin, 2014). The generative model developed in such an environment will be optimized to survive in an unpredictable world, prioritizing short-term over long-term reward, in line with social psychological theories on childhood adversity and psychopathology (e.g., Amir et al., 2018). When the environment has changed to be less unpredictable/uncontrollable, the same generative model then yields predicted perceptions and actions that are maladaptive. In this sense, the idea of generative model holding beliefs about the world maps onto the cognitive schema that represents views of the world and the self in traditional cognitive theory (Clark et al., 1999).

Psychology

In addition to biological (see the online supplemental materials), genetic, and environmental factors, several psychological factors play a role in the development of psychopathology. Take cognitive flexibility as one example. Cognitive flexibility is commonly associated with several clinical disorders (e.g., Geurts et al., 2009; Murphy et al., 2012). Cognitive flexibility requires timely updating of the beliefs (and related predictions and precision assignments) as context

changes. Impairments in flexibility can happen by failing to update the beliefs to account for context updates, leading to context insensitivity. We have mentioned above that the answers to the questions "which context am I in?," "Is this a familiar context or a novel one?," and "Has the context changed?" are part of the beliefs and are subjective to being updated by novel sensory evidence. Thus, context-insensitivity can be conceptualized in the PP framework as a failure of updating the belief regarding the familiarity of the context. This can be caused by the precision of the prior belief with a high probability over "I am in a familiar context" being too high relative to the precision of the novel sensory evidence. Consequently, a failure to account for the changes of context can lead to a selection of action based on a suboptimal calculation that is suitable for an outdated context.

PP Conceptualization of Exteroception and Threat Detection in Anxiety

Clinically, anxiety is characterized as an anticipatory response to possible, but uncertain, future threats (APA, 2013; Barlow, 2000, 2004; Grupe & Nitschke, 2013). Mainstream cognitive conceptualizations of anxiety emphasize that it develops and persists due to prioritized or biased attention toward threatening stimuli, a bias that is characterized by (a) facilitated attention, (b) difficulty in disengagement, and (c) attentional avoidance (e.g., voluntarily directing attention away from threats) for threatening stimuli (for review, see Cisler & Koster, 2010). Facilitated attention or perception of threatening stimuli is hypothesized to be an automatic process, while attentional avoidance is viewed to be more controlled, and difficulty in disengagement to be a mix of the two (Cisler & Koster, 2010). Here we provide a novel narrative using the PP framework to explain these diverse aspects of threat-related (exteroceptive) attentional biases in anxiety.

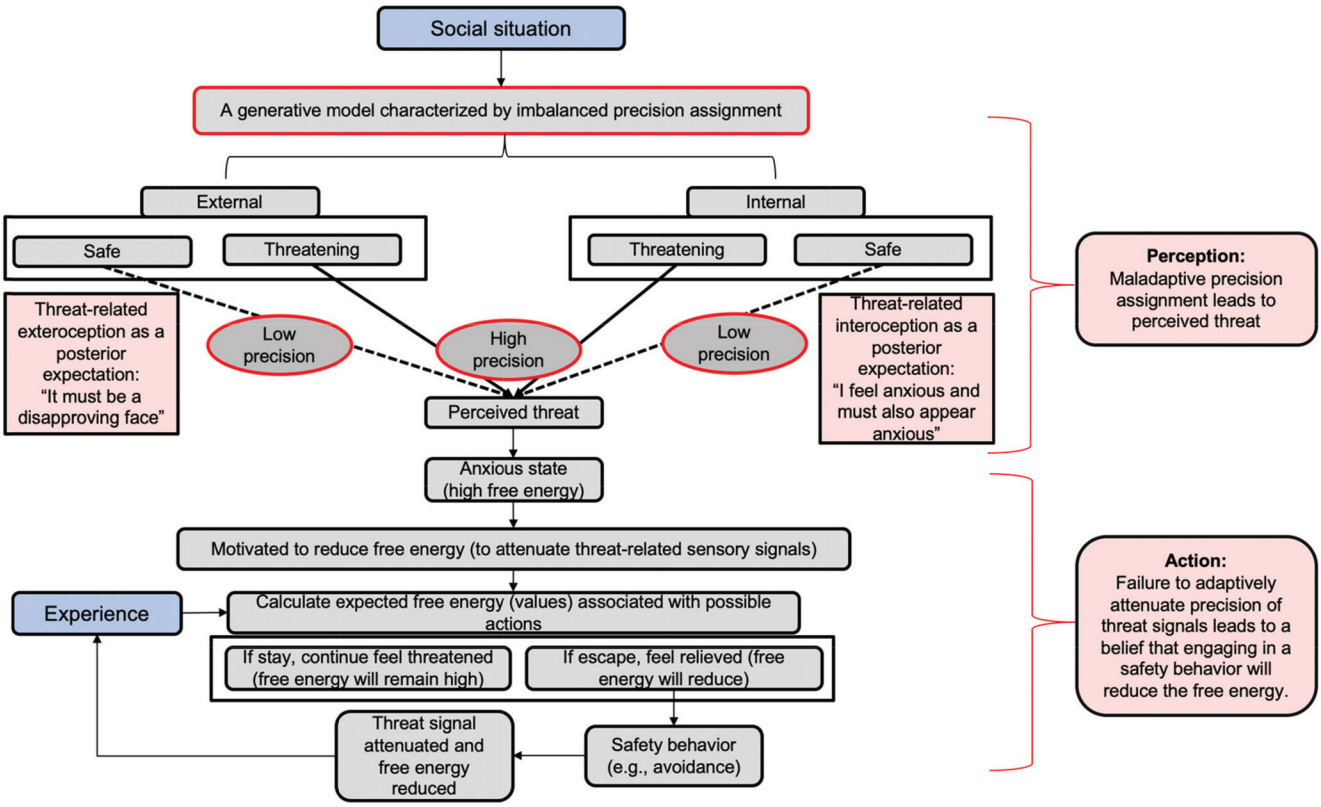
We propose that overidentification of threat may be attributed to an imbalanced precision assignment to threatening versus safety signals. Specifically, genes, personality (e.g., neuroticism), learning (e.g., fearful experiences), and the environment (e.g., uncertain and unpredictable stressors) can shape a generative model that, at the pathological stage, assigns an abnormally higher precision to threat-related beliefs than safety-related beliefs. Consequently, such beliefs will result in predictions favoring threatening sensory signals and a higher precision assigned to threatening compared with safe sensory evidence. When both threatening and safe stimuli are present, the related sensory evidence competes for cognitive resources for downstream processing (as described in aforementioned simulation of spatial attention (Feldman & Friston, 2010) and, owing to its higher precision, threat-related information is more likely to be propagate downstream and be represented in conscious awareness. Threat-related attentional bias (facilitated attention) is thus an emergent property of maladaptively high precision assigned to threatening sensory evidence, allowing it to capture attention more easily than safety sensory evidence. Importantly, individuals with anxiety do not encounter threatening stimuli in a vacuum. Rather, these stimuli are encountered in familiar settings where learning and prior knowledge provide contextual cues on what is likely to occur and thus influence the beliefs and subsequent sensory predictions and precision assignment for encountering threatening targets. Altogether this indicates that facilitated attention to threat in anxiety is not a purely stimulus-driven and automatic effect (Sussman et al., 2016). Rather, it also involves an anxiety-related and context-dependent top-down modulation via precision assignment that amplifies the threatening signals.

The same PP framework can be applied to explaining inability to disengage from threat. Disengagement from threatening stimuli requires the attenuation of the precision allocated to the threatening sensory signals. A higher-precision sensory evidence requires more effort to attenuate than lower precision sensory evidence, resulting in prolonged processing of the attended information. Therefore, individuals with anxiety may show difficulty disengaging from the (no-longer salient) threatening stimuli because of failing to attenuate the precision of threatening signals. Finally, attentional avoidance can be understood from the action selection perspective such that the generative model assigns a higher value to escape behaviors compared with other action options, and this (extrinsic) value reflects the high-precision belief that executing the avoidant behavior will bring the individual to a relaxed state (a preferred state with lower free energy). Experimentally, higher precision over an action will lead to a more confident decision, which can be captured by faster reaction time (RT). Hence, if the above conceptualization is correct, then holding everything else fixed, a model with more imbalanced precision over sensory signals and actions favoring threat-detection and avoidance behavior shall be able to explain real human data including choice and RT distributions in a specific experiment (e.g., discriminating between threatening vs. safe stimuli).

Relatedly, a series of recent simulations studies on posttraumatic stress disorder (PTSD) symptoms can provide insights into how hypervigilance may be developed. Using the active inference scheme (modeled using partially observable MDP) Linson and colleagues (Linson & Friston, 2019; Linson et al., 2020) simulated how the mouse (Jerry) developed stress responses after encountering and escaping his feline foe (Tom). Jerry actively infers the causes of the sensory signals (e.g., Tom's face) from their consequences (e.g., a dot in the horizon) and constantly updates his beliefs about the state of the environment (whether safe or threatening). These beliefs lead to action selection from relax, fight, flight, or freeze depending on the expected free energy. Hypervigilance behavior emerges when Jerry holds excessively a high confidence belief that Tom is present, and thus engages in hyperarousal and freezing when spotting a remote dot (which turns out to be a dog). Under this context, an unimpaired simulated mouse would engage in exploratory behaviors substantially more than the impaired and hypervigilant mouse.

To give a concrete example, next, in Figure 4, we illustrate how PP can be integrated with well-established cognitive behavioral model of social anxiety (e.g., Clark & Wells, 1995). Social situations trigger high-precision threatening beliefs encoded in the generative model. This leads to an imbalanced precision assignment such that threat-related external (e.g., frowning expression) and internal (e.g., rapid heart rate) sensory evidence are assigned higher precisions than benign sensory evidence (e.g., neutral expression and normal heart rate). Given that social signals are often ambiguous (e.g., a puzzled face, mildly high-heart rate), this imbalanced sensory precision assignment will lead to an amplification of any threat-related signals in the stimulus compared with safety-related signals, leading to a biased or selective processing of threat-related information, resulting in perceived threat. Perceived threat can

Figure 4
Model for Social Anxiety



Note. See the online article for the color version of this figure.

further enhance the strength of internal physiological threat-related signals (i.e., high free energy state) that the individual is motivated to reduce by taking actions. This is akin to asking "what do I need to do to attenuate the precision of these threatening sensory signals?." An adaptive action will be to reassign the precision (redirect attention) to safety signals.

However, a socially anxious individual may find it hard to redirect attention and may also have learned from past experience that escape will lead to an immediate reduction of free energy (a preferred state), then avoidance behaviors naturally follow. This narrative can be formally modeled using a partially observable MDP, similar to how interoception and fear-learning have been successfully simulated in above mentioned study (e.g., Linson et al., 2020). This narrative is also in line with empirical findings showing that a failure to adaptively increase the precision assigned to the internal sensory signals is present in patients with anxiety among other disorders (Smith, Moutoussis, & Bilek, 2021).

Although the above model is an already-learned generative model, the learning process (as belief updating) itself can be conceptualized and formally modeled using active inference scheme. For example, reward-based learning has been successfully modeled using the current framework (FitzGerald et al., 2015). Broadly speaking, a diathesis of social anxiety, high neuroticism, can result in a high precision for threatening signals prior to the encounter of threatening signals. Given the same external environment, a neurotic individual with a maladaptively imbalanced precision assignment will learn threat-related information faster compared with individuals with a more balanced precision assignment, as the learning (i.e., belief updating) is largely driven by threatening information.

Together, anxiety, depression (see the online supplemental materials), and other internalizing disorders may be conceptualized as maladaptive precision assignment, both interoceptively (Paulus et al., 2019) as well as exteroceptively under PP. Notably, whereas depression has been hypothesized to be related to low precision assigned to internal sensory signals, anxiety maybe related to high precision assigned to internal and external sensory data indicating threats. This means that whereas individuals with pure depression are insensitive to (internal) sensory signals, individuals with pure anxiety are more likely to have belief update driven by threat-related sensory signals. Such predictions are in line with psychopathology models of the commonality and specificity of anxiety and depression (Kotov et al., 2017; Watson & Clark, 1984; Zinbarg & Barlow, 1996).

Conclusions

PP is a framework based in theoretical neurobiology that has received significant attention across several domains, including psychology, robotics, artificial intelligence and philosophy (Corlett et al., 2020). Recent development in psychopathology, and particularly clinical neuroscience, has witnessed an increasing interest

in applying the PP framework to understand pathological phenomena (Corlett et al., 2020; Friston et al., 2014; Smith, Badcock, et al., 2021). The PP framework is based on one simple principle—the free energy principle (Friston et al., 2006) and has demonstrated its power in explaining a range of basic phenomena including perception, action, cognition (including language) and emotion (Friston et al., 2015; Parr & Friston, 2017b; Seth & Friston, 2016). Conceptually, PP offers a system in which the interaction between intra- and extra-individual factors dynamically shapes the development of the generative model implemented by the brain, which given the right circumstances can manifest in psychopathology. Furthermore, it offers a system to explicitly integrate the neurobiological, psychological, and environmental factors into one framework. An appealing feature of PP is that it allows one to formalize the interaction of intra- and extra-individual factors, and the mapping between psychological constructs and their biologically plausible neural implementations. The present article stresses how PP framework can facilitate the integration of psychological and environmental factors and can be used to examine the psychological and neurobiological mechanisms of well-established psychosocial models of psychopathology.

Although the focus of this article has been to familiarize the audience with the conceptual aspects of PP, we will briefly discuss the formal modeling approach here (see the online supplemental materials for a sketch). PP provides a specific example of recent movement of computational psychiatry (Adams et al., 2016; Montague et al., 2012; Wang & Krystal, 2014). To use the PP framework to model a specific pathological phenomenon, researchers will need to construct an explicit mechanistic model that aims at producing output behavioral and/or neural responses that can be tested against empirical data. If the model generated data are undistinguishable from empirical data, then the face validity of the hypothetical mechanistic model is established. Researchers can then apply the model to empirical data to gain insights into the hidden pathological processes. Throughout this article, we have mentioned multiple examples where we see convincing face validity, as well as emerging empirical support. Specific model examples can be found in Statistical Parametric Mapping (SPM, https://www.fil.ion.ucl.ac.uk/spm/). This formalization provides clarity, precision, and rigor in psychopathology research (Teufel & Fletcher, 2020). Note that there are many modeling approaches that can be described as PP: many active inference studies use partially observable MDP with discrete variables, whereas predictive coding studies often use continuous variables.

Although the PP framework can unify and operationalize many theories of psychopathology, there are several challenges that remain to be addressed. There is considerable simulation-based research supporting PP; however, more experimental studies, particularly studies designed to integrate existing psychosocial factors and neurobiological factors, are needed. An increasing number of empirical studies involving real human data have applied conceptual ideas from the PP framework, such as the effect of prior information in guiding perception in anxiety (Sussman et al., 2016), autism (Lawson et al., 2017), and psychosis (Powers et al., 2017); however, few of these studies take advantage of the computational scheme. In addition, although a growing body of literature is demonstrating the utility of PP in explaining clinical symptoms/syndromes, most of the research focuses on interoception (in internalizing disorders) and sensory priors and PE (in psychoses). It is our hope that the present article draws attention to threat-related exteroception in anxiety and the role of emotion in development of psychotic symptoms (see the online supplemental materials).

Here we provide a couple of examples of hypotheses that can be tested using the PP framework. A high precision belief ("what I think I am sensing" or "what I think I will do") will lead to fast belief update when the individual enters a novel situation. Once the beliefs are formed (that is a strong prior belief about the environment), these beliefs will be difficult to update further. If a researcher hypothesizes that anxiety may be related to abnormally high precision beliefs, then using a threat-related associative learning task, one may predict that individuals with anxiety will show faster early belief updating and slower later belief updating. Also, given that visual attention can be formalized using precision assignment, one may hypothesize that in a task requiring sustained attention, performance of individuals with attention deficits such as in ADHD may best be predicted by a generative model with relatively nonprecise (flat) assignment about which part of the visual field the signals should be processed.

Future research also needs to address how the PP framework can help explain and inform nosology. Current genetic research suggests that genetic vulnerabilities act at a broad level of the hierarchy, predisposing individuals to psychopathology generally, rather than specific disorders and symptoms (Waszczuk et al., 2020). Future PP research could examine whether a genetic liability for anxiety, for example, is likely shaped into a phobic generative model through events that hone the precision of that belief. Also, it is yet to be examined how the current framework can explain the transition from one form of psychopathology to another, for example, mania to depression (Solomon et al., 2010). Furthermore, contemporary psychopathology models often stress the thought component, which intrinsically relies on human language. Recent advancement in PP, specifically active inference, has indeed started modeling language generation (Friston et al., 2020), but future experimental and empirical studies are needed to examine how maladaptive thoughts (e.g., excessive worries) are generated and maintained. Finally, although the temporal hierarchy of PP naturally incorporates neurodevelopment, there is a general lack of developmental and neurodevelopmental studies using PP, which warrants future research.

To conclude, the PP framework is very much in line with psychotherapy models such as cognitive behavior therapy with focus on internal schemas, stressing the roles of factors at various temporal scales that lead up to the present manifestation of psychopathology. This framework holds the promise to model and uncover the mechanisms of modifying prior beliefs that lead to therapeutic effects, but more intervention research is needed. Furthermore, given its strong base in neurobiology, the framework can naturally incorporate changes brought by pharmacological treatments.

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