Mental distress through the prism of predictive processing theory

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Abstract

We review the predictive processing theory's take on goals and affect, to shed new light on mental distress and how it develops into psychopathology such as in affective and motivational disorders. This analysis recovers many of the classical factors known to be important in those disorders, like uncertainty and control, but integrates them in a mechanistic model of adaptive and maladaptive cognition and behavior. We derive implications for treatment that have so far remained underexposed in existing predictive processing accounts of mental disorder, specifically with regard to the model-dependent construction of value, the importance of model validation (evidence), and the introduction and learning of new, adaptive beliefs that relieve suffering.

Keywords: *predictive processing, mental distress, psychopathology, emotion, depression, anxiety, active inference, addiction, learning, psychotherapy, computational psychiatry*

Introduction

In recent decades, the influential theory of predictive processing (PP) (also called Bayesian brain or active inference) [1–3] has increasingly gained traction. It states that organisms do not merely respond to stimuli but proactively model and predict sensory inputs and the opportunities and challenges they represent. This view implements pioneering ideas of Jerome Bruner [4] and George Kelly [5] that describe mental processing in terms of the formation and testing of hypotheses. As true proto-scientists, we make predictions about the world and meet reality only in our failures [6], that is, when sensory input deviates from our predicted or constructed versions. Such deviations are called *prediction errors* in PP lingo. Because we perceive through our own

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constructs (in the form of predictions), perception is often described as ‘controlled hallucination’, which means that we de facto live in a constructed reality [7], kept in check by prediction errors.

The computational details of PP are beyond the current scope [8–10], but the central thesis is that the mental system realizes all its functions through prediction error minimization. Organisms embody an evolutionarily ‘discovered’ set of expected interoceptive (homeostatic) states that are required for continued existence but that they can only realize via action in their environment (e.g. foraging for food to increase blood glucose levels). This requires them to build a generative model about the structure of their environment and how to ‘generate’ their expected states through action. This model allows them to not only react on homeostatic disturbances, but also predict and so preempt those. It is shaped by prediction errors, which can be minimized either by acting to bring predicted events in line with the current model (cf. assimilation), or by perception and learning, that is, by changing our models and inferences so they correspond better with incoming evidence (cf. accommodation). In this way, the organism maximizes the evidence for the model it embodies, often called *self-evidencing* [11]. Importantly, our sense of self can be seen as just another inferred cause in the generative model, as the best explanation for the regularities in multimodal perceptual consequences created by one’s own actions, and as an efficient prediction of (regularities in) one’s future behavior, emotions, and thoughts (e.g. the narrative and social self). From this perspective, we construe and infer not only what is there in the world, but also who we are, what we can do, and what we want (i.e. our goals), as also described in self-perception theories [12].

The PP framework hence entails that a person’s behavior is canalized by the ways in which they construe and anticipate events and their own reactions to these (constructed) events [5]. Evidently, this holds for mental distress and disorder too. It is clear that people can end up with sometimes drastically diverging constructs (or beliefs) and mental models, first because of the vagaries of the environment that shape the models (i.e., their phylogenetic and ontogenetic learning history), second, because the updating of constructs using prediction errors is a fallible job that requires weighing evidence based on different types of uncertainty [**13], and third, because the very gathering of evidence is steered (biased) by the current constructs. Because of its unique capacity to account for the variety and idiosyncrasy of models, we will argue that the PP framework is well-placed to help us understand mental distress and, hopefully, its treatment.

Goals in predictive processing

The overarching principle of prediction error minimization means that the mental system is geared towards *model validation*, maximizing the evidence for one's model or, equivalently, reducing uncertainty relative to this model. At first glance, this conflicts with established reward-based or goal-directed theories [14] that see reward maximization or goal-realization as the core organizing principle for the mind. But, as pointed out above, both biological needs and more personal goals are folded into the model. Goals are expected states (desired outcomes) that can be attained

('generated') by our own actions. Some (core) goals are defined at a higher level (longer temporal horizon) and are persistent against obstacles, i.e. these expectations will *not* be readily updated by momentary prediction errors. Evidently, this resistance is crucial for homeostatic expected states, where 'updates' (e.g., lowering glucose expectation) would lead to loss of organismic integrity. However, humans construe a myriad of other goals and values in (putative) support of those interoceptive expectations. We infer our goals and values from the history of our choices and actions (e.g., approach and avoidance) [15], as well as from what people like us (parents, peers, and mentors) do and want. What makes these expectations resistant to updates is the *precision* they have accumulated through experience. Precision is the technical term in PP for the estimate of expected uncertainty associated with expected sensory states, in this particular case the expected outcomes of actions. In the literature on goal-directed behavior [16], this expected uncertainty about mapping between actions and outcomes is known as 'feasibility' or control(lability).

Given that any behavior is driven by expected (interoceptive or exteroceptive) states, habits in the sense of automatic stimulus-response mappings have no place in PP. Instead, habit-like behavior can be accounted for as actions that are based on strong stimulus-stimulus predictions (one sensory input predicts another proprioceptive and exteroceptive input) maintained on hierarchically lower levels. While these actions were originally driven by higher level goals, through experience, the lower level expectations involved (sub-goals) accumulated enough precision (reduced uncertainty) so the behavior is now sustained by 'shallow models' [17,18]. In contrast, behavior commonly referred to as 'goal-directed behavior' depends on higher level goals and involves lower level expectations that are contextualized by hierarchically deep models. These higher level goals are only activated when there is uncertainty about actions and whether those lead to desired outcomes. For instance, when sensory input activates different alternative actions, deliberation will involve weighing whether one already has reliable (precise) actions to attain desired outcomes, or whether one needs to gather more information about the environment (including the consequences of one's actions) through so-called epistemic actions. This deliberation may remain implicit, but will include counterfactual beliefs: "What if I do this, instead of that, how well does that reduce my expected prediction errors (uncertainty)?". Hence, whether some behavior is goal-directed or habit-like, is a matter of degree, dependent on how shallow or (counterfactually) 'deep' the model is that led to this behavior.

This conceptualization of goals casts a new light on clinically relevant cases where verbally reported goals conflict with (possibly maladaptive) actual behavior that is more habit-like but possibly still driven by lower level "goals" or desired outcomes [18]. Indeed, here we essentially have two different models within one agent applied to the same behavior: The model that supports the behavior of self-reporting our explicit, conscious goals, and the model that supports the habit-like behavior. For example, in addiction, a particular 'eliciting' context would evoke high precision beliefs about interoceptive expected consequences of substance use (the habit-like behavior). But such beliefs are distinct from the (more explicit) beliefs that underlie statements about this

behavior and its consequences in a different context (e.g., during a therapy session). Note that the terms 'beliefs' and models are used in the Bayesian sense here, as implicit, inferred networks of hidden causes (technically: probability distributions over parameter values) or constructs that explain input and sustain behaviors [19]. Part of the work in clinical therapy, as psychotherapists can attest, is therefore to articulate these constructs underlying habit-like (pathological) behavior, not so much to be able to directly confront them with the clients' conscious goals, but to confront them with inconsistent evidence directed toward their own logic. This will boost the expected uncertainty of unwanted beliefs, enabling new learning and epistemic actions to establish constructs that support effective behavior in line with one's explicit goals. While this view confirms received views of cognitive behavioral therapy, it also acknowledges the problem of fragmented models: The fact that some (parts of) models that support problematic behavior are so 'inferentially isolated' that the task of un/relearning models cannot be undertaken right away, but should be preceded by a reconstruction of underlying beliefs and desires. This quest to understand the subject's web of constructs (their generative model) from within is akin to an anthropologist's task [20], but it is required to target the right 'wrong' beliefs in therapy.

Mental distress in predictive processing

The PP framework has already been applied to several psychopathologies, such as autism spectrum disorder [21], schizophrenia [22], and obsessive-compulsive disorder [23], usually by characterizing these conditions as stemming from a general deficiency in prediction updating (technically: precision weighting). However, to explain mental distress in affective and motivational disorders with their strong link to life events, we first need to look at the way PP understands emotions.

Dominant views of affect cast it as a continuous "neurophysiological barometer of the individual's relationship to an environment at a given point in time" [24]. Similarly, Frijda [25] noted: "pleasure is the positive outcome of constantly monitoring one's functioning". Translated to PP, this kind of monitoring of one's own performance in predicting internal and external disturbances, needs to be an operation on prediction errors. However, given that prediction error minimization is really all the brain does (when perceiving, acting, and thinking), any momentary prediction error cannot be very informative or 'affective'. Instead, meta-expectations about the temporal average of prediction errors and how that changes —whether uncertainty increases or decreases relative to one's models— may fulfill this meta-monitoring function that we can associate with emotional valence [26–28]. This aligns with the well-known role of increasing uncertainty in negative affect such as anxiety [29], and of decreasing uncertainty in pleasure, such as aesthetic appreciation [30]. Specifically, given that, as described in the previous section, we absorb goals into the agent's model, decreasing/increasing uncertainties relative to that model are precisely informative about our progress/regress relative to our goals and the aptness of our models to deal with our

surroundings.

This sets the stage for an understanding of distress as rooted in persistent breaches against predictive models, especially concerning high-level, high precision expected states (goals), because those predictions structure major parts of one's behavior, and are the basis for interpretation of much of the perceptual inputs the person encounters (past, present, and future). Persistent and increasing prediction errors signal the subversion of a person's constructed identity: the regularities from the world that they have absorbed but that no longer hold. It is here that we situate normal and pathological distress, be it grief, depression, or anxiety.

Usually, prediction errors (even with regard to core goals) can be neutralized by changing one's auxiliary assumptions, and so keeping the core of a person's models intact (sometimes called cognitive immunization). That is just run-of-the-mill model updating which will take place first for lower-level, less precise predictions, rather than for higher-level, precise predictions. However, persistent model disconfirmations can incite a number of different responses to reduce uncertainty (increase model evidence). A first approach might be to tighten up on one's constructs, and insist on their validity. This is often expressed as a more selective data gathering to file off the sharp edges of disconfirmations, in other words: avoiding particular (e.g., social) situations and retreat to more reliably predictable environments, as we often see in the rituals of anxious people. Another outcome could be hostility, to be seen as extreme efforts to force the circumstances to conform to one's constructs again. Whether avoidant action or aggressive action is taken may depend on perceived control, a well-known factor in emotion and psychopathology, which, in PP, can be conceptualized as uncertainty about (our prediction of) action consequences [31].

Crucially, persistent prediction errors in reaching desired action outcomes can lead to model updates that increase the expected uncertainty or, simply put, the perceived powerlessness of actions. The generalized loss of confidence (certainty) that one can attain the expected consequences of one's own actions (cf. learned helplessness) can then lead to withdrawal and other depressive symptoms. This resonates with the fact that unpredictable, unresponsive environments, especially early on in development, are a key risk factor for (depressive) psychopathology [32].

The 'system-wide' generalization of loss of confidence in the efficacy of all actions (as in depression) might seem unwarranted but it may actually be internally-rational, given one's models and the experiences that shaped them. Usually, the reverse generalization, a certain generalized overconfidence (known as the optimism bias or the overestimation of control) keeps us motivated and is often seen as a mark of good mental health [33]. However, a generalized *loss* of confidence in actions shifts one's mental model from trying to fit the world to their mental model (by actions) to fitting the mental model to the world and may therefore be a successful way to more easily achieve prediction error minimization. This might relate to depressive rumination (searching for better models without seeking out new evidence) as well as depressive realism, the finding that people with depression often have a more accurate perception of their capacities and control (i.e., a more accurate model of the present).

Finally, another effective strategy to deal with persistent prediction errors is to form predictions on a more global, less granular level [34]. Simply stated, categorizing something as merely 'fruit' or 'vegetable', rather than at a more specific level, allows one to be correct more often. Applied to psychopathologies such as anxiety disorder, over-general threat-related priors (e.g., "social situations are threatening") may start to dominate thought and behavior because they 'worked' (i.e., they were effective in reducing errors) and have therefore accumulated great (perceived) precision, hence becoming less sensitive to new contradicting evidence [*35]. In addition, by putting all social situations under one umbrella, the lack of confidence associated with an action in one particular social situation easily propagates to lack of confidence in the ability of actions for social situations across the board. The one action that remains is avoidance, which of course prevents the confrontation with new evidence altogether.

Interestingly, none of these mechanisms to cope with persistent prediction errors can be labeled as pathological per se (they are part of the conspiracy theorist's mind, and even the 'normal' mind). They are valid, adaptive ways of curbing prediction errors, not specific to psychopathologies. Hence our focus on distress in general. However, the salience of prediction errors can create an increasing pressure to actively shape one's environment (or 'niche') to reduce or prevent these errors [36], following the idea of active (selective) evidence sampling, central to PP. As the avoidance generalizes, following the principles above, one's world narrows, which in turn will be mirrored in a narrowing of cognition (one's models). In a progressive, reciprocal fashion [**37], this will again lead to a further shrinking of one's world, and so on in a vicious mutually reinforcing feedback cycle. It is this gradual reciprocal narrowing of one's goals and environments (cognition) that creates suffering and the need for psychotherapeutic support. Here, terms such as 'disorder' or 'maladaptive behavior/beliefs' will apply. Addiction, as well as anxiety and mood disorders can be usefully described in this fashion, where particular concerns come to dominate one's world and collapse other goals and predictions. PP is uniquely suited to describe this developmental process, thereby reconceptualizing mental disorders as a possible, pernicious outcome of normal environmentally-embedded learning, instead of as brain diseases [37]. This may cast a new light on the fact that no discrete biomarkers have been found (e.g., for affective disorders or addiction), and that many mental disorders share genetic risk factors [38,39] as well as a common psychological vulnerability factor [40]. It also helps us understand why pathogenetic reciprocal narrowing is more likely when living conditions have already started to narrow one's paths available in the world (cf. the role of early adversity in life or of prior physical illness in mental disorder).

Toward more effective treatments

Current psychotherapeutic practice of affective disorders predominantly involves trying to uncover and modify a client's maladaptive beliefs (CBT). The PP account fits well in this tradition [**41]

because it provides a neurocognitive process theory about how these beliefs are formed and how they can be changed (using scaffolded learning environments). Correspondingly, most papers that cast psychopathology in PP terms emphasize belief updating [*42] and, because this precision-based updating is thought to be mediated by neuromodulators, they express the hope for better pharmacological treatments in the future [43]. However, PP's capacity to help us understand and improve psychotherapy is less explored so far. Here, we can only briefly touch on three important elements.

Firstly, clinical practice should give attention to specifying the generative model of the client, to determine what the client construes as valuable (or 'expected') instead of relying on objective definitions or normative ideas of what is rewarding or unpleasurable (e.g., my idea [expectation] of what a mutually beneficial [valuable] social interaction entails might be very different from yours, which gives me significant anxiety when our pattern of interaction deviates from it). Failing to do so may make us either blind to the attractiveness of "what should objectively be" aversive situations ("Why does a person sometimes persist in 'unrewarding' behavior?" [44]), or puzzled about someone's avoidance of "what should objectively be" neutral or even positive situations. For instance, harmless situations (e.g., asking a barista for a coffee) can, through the workings of our predictive mind, be construed as (near) catastrophes (e.g., the fact that she didn't understand me the first time is proof that I am totally insignificant, which brings me physical anguish, which de facto makes it a dreadful experience instead of a harmless one). This of course precludes the updating of maladaptive avoidance behavior in exposure therapy [**41].

Secondly, 'model validation' will be an important, recurrent step in psychotherapy. This means providing support (in dry PP terms: model evidence) for core constructs such as adaptive beliefs, values and goals (cf. acceptance and commitment therapy). In fact, model validation is what we colloquially mean by 'consolation' (by a therapist or friend), that is, the reassurance that one's emotional dynamics are to be expected ('normal') given the reference frame of the person (their beliefs, experienced events, and goals). It renders their experiences *predictable* and meaningful, rather than aberrant, irrational or unpredictable. Earlier, we alluded to the 'protective belt' that models build for themselves by explaining away prediction errors using auxiliary hypotheses or by downregulating the salience of conflicting evidence by lowering our trust (expected confidence or certainty) in particular information sources. To alter these models, it is important to cultivate (renewed) epistemic trust in suppressed information sources and in one's own actions, evoking a generalized sense of initiative beyond the therapeutic setting [45,46]. In PP terms, this constitutes regaining confidence that one's actions will minimize prediction errors (that one's planning works, that a future can be produced). The model validation stage is crucial for regaining this trust/confidence.

Thirdly, the model validation provides a safe basis for the introduction of new, more adaptive constructs and inferences in mental models. Here too, taking into account automatic predictions has shown to provide an advance beyond traditional association-formation and behavioral change

(CBT) ideas [47]. Eliciting a change in generative models requires us to set-up the environment to evoke prediction errors and to aid accommodation of these errors in an adaptive, sustainable manner. Simply put, therapists should create learning environments that allow clients to confidently predict that they will perform alternative behavior (e.g. drinking water in case of alcohol addiction) in relevant contexts (e.g. after dinner) and infer precise supporting beliefs for this behavior, most notably that this alternative behavior aligns better with their goals [48]. Together, the above three elements should help to widen (instead of narrowing) one's prospects again —the diversity of one's predictions, goals and 'action radius' in the world.

Conclusion

The emerging description of mental distress in PP terms dovetails with insights from several psychotherapeutic traditions, as well as with dimensional (non-essentialist) and transdiagnostic views in psychopathology. The PP approach proposes plausible (inferential) mechanisms not only for pharmacological intervention, but also for the science of psychotherapy, showing that the 'soft' hermeneutic skills of psychotherapy [49] lend themselves to a 'dry' Bayesian PP analysis. It can do so because it is decidedly *not* biologically reductionist, through its emphasis on how the environment shapes inferences and constructed values [50]. Much more than a PP gloss on old ideas, the framework should continue to show great heuristic power for psychopathology.

Conflict of interest statement

Nothing declared.

Author note

This work is supported by the Flemish Government under Grant METH/14/02 and by the Scientific Research Foundation Flanders under Grant FWO19/PDS/041 awarded to Pieter van Dessel.

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[**13] The authors review evidence that estimation of uncertainty plays an important role in learning and explain how misestimation of uncertainty can lead to symptoms of anxiety and depression and might underlie affective disorders.

[*35] The authors explain how a person's general strategy to process prediction error in low detail can give rise to weakly informed prior beliefs that might underlie (affective) mental disorders.

[**37] The author reviews neurobiological alterations in addiction and concludes they cannot be labelled as pathological, let alone specific to the disorder. He presents an alternative learning-based account of addiction based on the concept of reciprocal narrowing that may fruitfully be applied to mental disorders more generally.

[**41] The authors describe how recent advances in PP and computational modeling can help formalize and test hypotheses regarding the maladaptive inferences that underlie mental disorder and can improve learning-based therapies such as CBT.

[*42] The authors review evidence about how healthy people and people with mental disorders update their beliefs after receiving new information that supports or challenges their views. They argue that the strategy to afford very high or very low precision to prior beliefs may underlie psychopathology.