BRAIN
REVIEW ARTICLE

A new science of emotion: implications for functional neurological disorder

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Functional neurological disorder reflects impairments in brain networks leading to distressing motor, sensory and/or cognitive symptoms that demonstrate positive clinical signs on examination incongruent with other conditions. A central issue in historical and contemporary formulations of functional neurological disorder has been the mechanistic and aetiological role of emotions. However, the debate has mostly omitted fundamental questions about the nature of emotions in the first place. In this perspective article, we first outline a set of relevant working principles of the brain (e.g. allostasis, predictive processing, interoception and affect), followed by a focused review of the theory of constructed emotion to introduce a new understanding of what emotions are. Building on this theoretical framework, we formulate how altered emotion category construction can be an integral component of the pathophysiology of functional neurological disorder and related functional somatic symptoms. In doing so, we address several themes for the functional neurological disorder field including: (i) how energy regulation and the process of emotion category construction relate to symptom generation, including revisiting alexithymia, 'panic attack without panic', dissociation, insecure attachment and the influential role of life experiences; (ii) re-interpret select neurobiological research findings in functional neurological disorder cohorts through the lens of the theory of constructed emotion to illustrate its potential mechanistic relevance; and (iii) discuss therapeutic implications. While we continue to support that functional neurological disorder is mechanistically and aetiologically heterogenous, consideration of how the theory of constructed emotion relates to the generation and maintenance of functional neurological and functional somatic symptoms offers an integrated viewpoint that cuts across neurology, psychiatry, psychology and cognitive-affective neuroscience.

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Abbreviations: FND = functional neurological disorder; HR = heart rate; HRV = heart rate variability

Introduction

Functional neurological disorder (FND), a condition at the intersection of neurology and psychiatry, reflects impairments in brain networks leading to distressing motor, sensory and/or cognitive symptoms that are diagnosed using positive clinical signs on examination incongruent with other conditions.[1,2] FND is commonly encountered, potentially disabling and results in significant healthcare costs, yet the underlying mechanisms for this condition remain incompletely understood.[3,4] People with FND can display a range of physical symptoms (e.g. abnormal movements, convulsions, speech output difficulties, dizziness, cognitive symptoms, pain, fatigue etc.), with a subset of individuals also endorsing co-occurring mood, anxiety and trauma-related mental health concerns.[5,6] Across ancient and modern times, FND has had a long and tumultuous history, with an evolving debate and understanding of how biopsychosocial factors (in early discussions also spiritual or supernatural elements) contribute to the manifestation of the disorder (see Fig. 1 for a timeline of FND conceptualizations).[7–29] A central issue in contemporary discussions has revolved around questioning the extent to which emotions play a mechanistic and aetiological role in FND.[23,28,30,31] Critical in this context, however, is that this ongoing debate has largely omitted the question of what emotions are in the first place.

Classically, emotions are understood as reactions to external or internal stimuli, whereby a dedicated neural circuitry coordinates a distinctive, recognizable pattern of physiological changes [e.g. heart rate (HR) fluctuations, hormone release, etc.], vocalizations, outwardly presenting motor activity such as facial expressions (Fig. 2), and mental features such as a subjective feeling and metacognitive awareness.[32] Different emotions are often understood to be biologically and psychologically distinct, universally present in humans and biologically innate. For example, instances of fear are assumed to be similar in their biological and psychological features across situations and people worldwide, because the assumed fear-circuitry in the brain is the same in all neurotypical humans.[33–35] Furthermore, while instances of emotion are thought to be associated with distinct bodily changes (e.g. the pitch of the voice, the tone of the muscles), they are largely understood to be different from physical symptoms, such as a headache, gastric distress or fatigue. This classical view of emotion derives from and is embedded in westernized sociocultural narratives, and its implications set a model for investigating how emotions develop (and go awry) in humans across the lifespan.[35–37] This includes research in FND (encompassing previous work from authors of this article), with task functional MRI (fMRI), neuroendocrine, autonomic and behavioural measurements attempting to probe emotions and their neurobiological underpinnings, guided (at least in part) by the classical view of emotion.[27,28,38–46]

However, accumulating cognitive-affective neuroscience evidence from almost every domain of measurement is strongly inconsistent with this classical view of emotion and its implications (Barrett and Lida, submitted for publication).[47–51] The notion that discrete emotion categories can be reliably identified by specific behavioural patterns and/or physiological features has been seriously called into doubt by the marked variability within instances of the same category as well as similarities across categories. Disconfirming evidence has been observed in research on facial movements,[52–57] vocalizations,[58,59] patterns of autonomic nervous system physiology,[34,60–62] brain activity profiles[63–68] and single neuron recordings.[69–71] Even the supposed prototypic brain 'biomarkers' for a given emotion category vary substantially across studies,[65,72–76] and across subjects within a given study.[34,55,63,65,69,77–83] Structured variation has been observed to be an intrinsic, instead of an epiphenomenal, property of emotions.[50] People also differ in their emotional granularity: the ability to construct precise, nuanced instances of emotion. Individuals who construct instances of emotion that are lower in granularity represent a range of negatively (or positively) valenced experiences using the same emotion word.[84–86] Emotional granularity is also linked to flexibility in the physiological motifs evidenced during an instance of emotion[86] and coping repertoires.[50,84,87] Lastly, new evidence over the last decade calls into question claims of the universality of emotion categories across cultures.[53] For example, the Himba from north-western Namibia do not label non-word vocalizations (e.g. growl, scream, sigh) in expected westernized emotional terms[88] and, in northern Tanzania, the Hadza use action words to describe facial configurations more frequently compared to their American counterparts.[54]

Advances in our understanding of fundamental brain structure–function principles in recent years contributed to the emergence of new formulations of consciousness in general and emotion specifically, including constructionist perspectives such as the theory of constructed emotion.[51] In parallel, a landmark paper by Edwards, Adams and colleagues in 2012 applied a hierarchical Bayesian inference model to account for functional sensorimotor symptoms,[23] with a cognitive framework supported by empiric research underscoring roles for altered sensory attenuation,[89] biased attentional allocation[90] and deficits in motor learning.[91] In this article, we revisit the role of emotion in the pathophysiology of FND.[49–51] First, we outline a set of inherent working principles of the brain, followed by a focused review of the theory of constructed emotion. Thereafter, we use this framework to posit how the predictive process of constructing emotion categories can be an integral component of the neurobiology of FND. In doing so, we address several important themes for the FND field including: (i) how energy regulation and altered emotion category construction relate to symptom generation, including revisiting the influential role of adverse life experiences; (ii) illustrate the potential mechanistic relevance of the theory of constructed emotion to FND by re-interpreting select findings in the literature; and (iii) discussing important therapeutic implications.

Allostasis and the predictive brain

Organisms need to obtain, store and use energy efficiently to survive. Small unicellular organisms like *Escherichia coli* can rely on cell membrane mechanisms to regulate energy intake from their direct environment,[92] while multicellular organisms with specialized tissues, organs and systems leverage a more orchestrated approach to execute energy balance.[92,93] In such organisms, energy

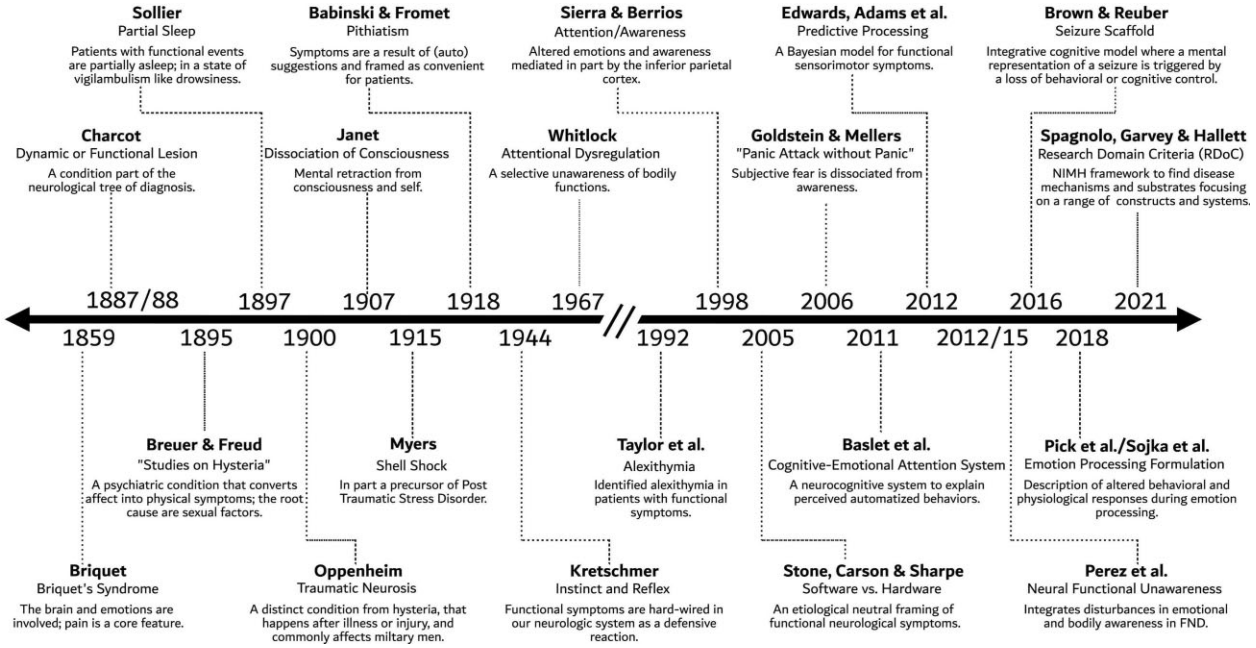

Figure 1 Examples of conceptual theories on FND in the modern medical literature. While not a comprehensive list, timeline depicts select historical and prevailing mechanistic theories for the development of FND.[7–29]

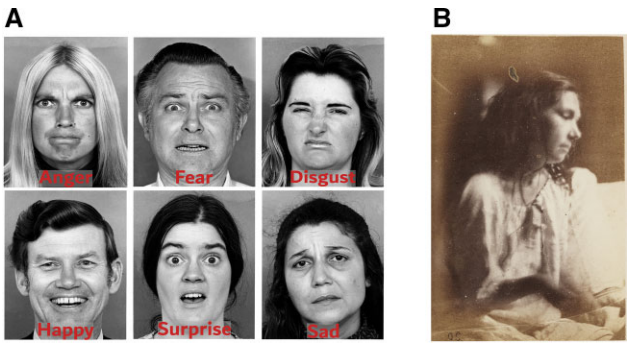

Figure 2 Classical (conventional) theories argue that internal emotional responses consistently relate to outwardly presenting facial and bodily expressions. (**A**) Photographs displaying stereotyped Ekman faces for emotion categories (replicated with permission). (**B**) Individual diagnosed with functional neurological disorder with a melancholic appearance (Delire Melancolique). Photograph in the public domain: commons.wikimedia.org.

management is performed by neural systems, which in some animals evolved into brains as new sensory organs and specialized motor features developed.[92,94] Without a dynamic energy budgeting system that manages the complex regulation of bodily systems in an ever-changing world, multicellular organisms could not survive. Therefore, although it would be a teleological claim to state that brains evolved to regulate the body's energy, it is a fact that energy regulation is a core brain function.[51]

The energetic supply-and-demand of the body is managed centrally through allostasis: the active process of forecasting the energetic needs of the body by modelling the body in the world and trying to meet those needs before they manifest.[95,96] Distinct from homeostasis, allostasis is concerned with metabolic efficiency instead of bringing a system back to a set point (or various set-points). With allostasis, a model to help prepare the body's next action plan based on past experiences is implemented within the CNS.[51] To understand how this is achieved, the foundational principles of predictive processing theories can be leveraged.[97,98]

Predictive processing frameworks represent a family of theoretical neuroscience approaches that explain how anticipatory calculations are generated and refined within the brain. These approaches are used to explain different features of brain function (such as motor action, belief and memory) within and across neural networks (including the neocortex, cerebellum and hippocampus),[99,100] and generally share three key principles. First, predictions of the future are continually generated within the brain: based on previous life experiences, an array of parallel predictive simulations are computed that all come with a certain probability.[96,101–112] If the prediction with the highest probability is confirmed by incoming sensory input, it becomes a perception, otherwise this results in a prediction error. Prediction errors, the second principle, are the mismatch between anticipated and actual sensory input, and are used to update and improve subsequent predictions. With a prediction error occurring, energy is either invested in refining the model or conserved by ignoring the prediction error in the instance that such information is not particularly relevant for the pursuit of metabolic efficiency.[96,98,108,112,113] Therefore, precision signals (the third principle) tune the different incoming predictions and prediction errors through engagement of the salience network.[113–115]

To perform allostasis efficiently, the system needs to anticipate, sense and integrate signals originating from within the body made available by interoception: the brain's moment-to-moment modelling of the internal physiological state of the body.[95,112,116–118] Interoception relies on comparing a predictive model to incoming sensory input arising within the body.[119] These interoceptive signals are then integrated within the brain to ultimately maintain allostasis efficiently. For example, interoceptive signals regarding the blood glucose level are compared against interoceptive predictions to update the in-the-moment index of the energetic state of the body to regulate the body accordingly. Therefore, the interoceptive model works in the service of allostasis.[95,112]

The process of interoception is mostly outside of awareness, but provides a low-dimensional moment-to-moment characterization of the state of the body that is experienced by the individual as affect.[95] Affect is the ever present awake-moment low-dimensional feature of consciousness that can be described as simultaneously having two core properties: valence (i.e. pleasant or unpleasant feeling) and arousal (i.e. feeling high or low in energy); furthermore, affect often has other descriptive qualities such as effort (i.e. feeling energized versus tired).[120] Affect serves as a subjective barometer of the in-the-moment estimation of the energetic state of the body, and influences the moment-to-moment perception of incoming information.[121]

To understand how, on a neuroanatomical level, predictive processing models are implemented, it is important to first recapitulate what a brain is: a massive collection of multipurpose neurons where many neurons synapse to one and a single neuron synapses with many.[51] Therefore, a single brain structure (made from many neurons) has a range of active connectivity pattern possibilities in a single moment in time.[51] This complexity allows that brain functions are not constrained to distinct sets of neurons but rather have degeneracy (many to one functions)[122–124]: engagement of a different set of brain areas (activation patterns) can produce the same outcome.[125,126] Similarly, brain areas (and sets of brain regions organized into discrete networks) are also domain-general (one to many)—meaning that one brain region/network can be involved in several functions (such as nociception and cognitive control).[51,127] For example, people with Urbach–Wiethe disease resulting in extensive bilateral amygdala damage demonstrate degeneracy; some individuals show preserved novelty signalling indicating that other brain regions can compensate for functions typically attributed to the amygdala.[82] Likewise, the amygdala is domain-general in that it is involved in various functions including arousal, selective attention, salience detection/attribution, fear responses and activation of defensive behaviours.[80,81]

Predictive processing to guide the performance of allostasis is rooted in the brain's cortical cytoarchitecture. Predictions in the cortex run from less laminated structures (i.e. four-layered agranular tissue referred to as limbic cortices including: ventral anterior insula, cingulate and posterior orbitofrontal/ventromedial prefrontal cortex; and the primary motor cortex with a small granular layer) to higher laminated structures (i.e. six-layered eulaminate areas such as the mid-to-posterior insula as the primary interoceptive cortex, or koniocortex areas such as primary somatosensory, auditory and visual areas).[51,103,123,128,129] Prediction errors start at the sensory input level (i.e. primary sensory cortices), run in the opposite direction (koniocortex-to-limbic) and are integrated at each level (see Fig. 3 and caption for additional neuroanatomical details). In this cytoarchitectural gradient supporting predictive processing mechanisms, the most abstracted multimodal features of the brain's internal model initiate within the default mode network, which are then decompressed as prediction signals for sensory, visceromotor and skeletomotor functions along a gradient.[51,131–134] Prediction errors, on the other hand, are weighted for their potential allostatic relevance (i.e. the need to encode the prediction error) by the salience network.[51,135]

The constructionist brain and instances of emotion

The theory of constructed emotion offers a constructionist approach to understanding what emotions are by leveraging the brain's primary role in energy regulation and predictive processing. In the brain, incoming sensory information from the body and world is compared to features that have already been classified and can be used to give meaning to the current input. Without direct access to what is causing advent sensory information, meaning is derived from past events that seem similar to the current state of the body and world.[136] Based on past experiences that share features of equivalence with the present, our brain uses the concepts (i.e. abstract mental representations) that were relevant then, with all the possible futures and associated action plans to deal with and understand the current incoming sensory signals. In doing so, an individual is using an embodied concept as a prediction.[50,98,103,110,123,129,137]

If a prediction (i.e. the embodied concept) matches the incoming sensory information and the prediction error is minimized, similar features from the past are pieced together to give meaning to the present: constructing a situated or *ad hoc* category (Fig. 4).[50,51,110,138,139] When an experience of emotion is used as a reference for the present constructed category, we are constructing a conceptual category for emotion.[58,143] If the same set of incoming sensory information matched with a 'non-emotional' concept (e.g. 'exhaustion' instead of 'sadness') under different circumstances or by someone else, a conceptual category for emotion would not be constructed.

The primary purpose of the brain's constructed category, whether for an instance of emotion or otherwise, is allostasis. Newly constructed categories help sort, understand and deal with the incoming sensory data, and will later help inform future situations.[58,110,143] Categories provide meaning to incoming sensory input because they represent a grouping of features that have similarities or share a goal.[51] Prediction signals come to include visceromotor and motor action plans, affective properties and more, as they move along the cortical cytoarchitectural gradient preparing the body for the expected sensory consequences (called an efference copy or corollary discharge).[50,51,98,103,123,128,129,137,140,144] Thus, when an *ad hoc* category is constructed, the individual is primed to deal with the present.[51] In this way, the dynamics of predictive processing suggest that action preparation gives rise to experience, not the other way around. Therefore, *ad hoc* conceptual categories of emotion are compressed summaries that categorize physical signals, give them emotional meaning and render the experience an instance of that emotion category (Fig. 5).

Importantly, each constructed conceptual category for an emotion is situation-specific; therefore, the category itself encompasses varying features (i.e. we can construct happiness with the touch of warm water in a winter day or cold water in a summer day) and there are no two identical instances of an emotion (i.e. the happiness felt on the summer day is different from that of the winter day).[50] Similarly, the complete set of neuronal activation patterns and networks involved in constructing each conceptual category for emotion are also unique to that moment, meaning that the neurocircuitry involved in an instance of emotion is not fixed (albeit different instances of the same emotion category in a given individual do at times have some overlapping features). Furthermore, since conceptual categories for emotions are constructed based on self-referential events, they are perceiver-dependent, indicating that there is variability in the experience of a given emotion both between and within humans.[51] By implication, every category implemented in any human brain is an *ad hoc* event[139,145–147]: its features of equivalence are always constructed in a particular individual for a particular function in a particular situation.

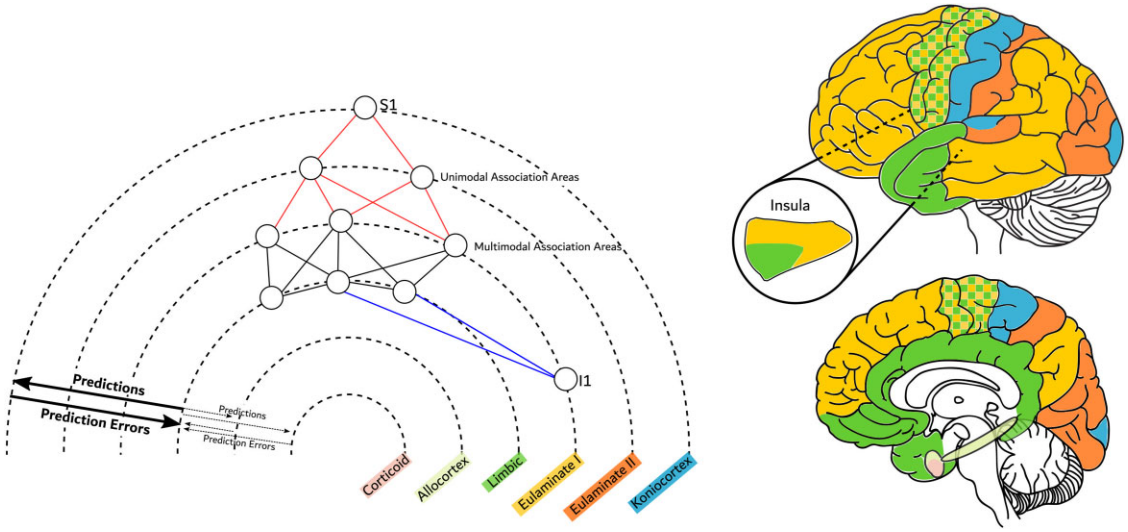

Figure 3 Relationships between cytoarchitectural complexity and predictive processing. Schematic representation of cortical laminar gradient in relation to the directionality of predictions and prediction error detection. According to their cytoarchitecture (lamination profiles), neuronal collections can be divided into networks (from least to most complex) as follows: corticoid [e.g. part of the amygdala, substantia innominata (not shown)]; allocortex (e.g. hippocampus, olfactory cortex, part of the amygdala); limbic cortices (e.g. cingulate cortex, ventral anterior insula, posterior orbitofrontal/ventromedial prefrontal cortex, parahippocampus and the temporal pole, with projections to the hypothalamus, PAG area, etc.); primary motor/premotor/supplementary motor areas; eulaminate areas (eulaminate I represent multimodal association areas, e.g. dorsolateral prefrontal cortex, lateral temporal areas, posterior parietal areas; eulaminate II areas are unimodal association cortices, e.g. superior parietal lobule of the somatosensory system) and the konicortex (primary somatosensory, auditory and visual areas). Predictions arise from lower-level structures and gain granularity as they are processed towards more complex areas. Prediction errors are received from sensory input in more laminated cortical structures and compressed towards simpler structures. Note, similar predictive processing approaches are described in the cerebellum and hippocampus. Thus, abstract brain-based concepts are used to predictively organize internal (bodily) and external (environmental) signals, with the goal of supporting allostasis. In this setting, areas defined as visceromotor regions (e.g. anterior cingulate cortex, orbitofrontal cortex, insula, amygdala, ventral striatum) implement allostasis by issuing predictions about the energetic state of the body and the relevance/salience of anticipated sensations.[51] A subset of visceromotor areas overlaps with the salience network.[115] In their role of guiding the re-allocation of attentional resources for personally and/or environmentally relevant information, salience network (cinguloinsular) areas are involved in interoception.[114] More broadly, visceromotor regions are interconnected to brainstem structures coordinating autonomic, immune and endocrine systems in the service of allostasis.[88] Additionally, this display only shows the predictive processing of somatosensory and interoceptive signals; other sensory modalities (e.g. visual) have similar processes. Note that there is debate on how to characterize the lamination profiles of the motor cortices, with conflicting evidence supporting an agranular/dysgranular ('limbic like') versus rudimentary layer IV ('Eulaminate I like') profile.[130] Thus, we display motor cortices in chequered colours representative of this intersection. I1 = interoceptive cortex; S1 = primary somatosensory cortex.

Incoming information that cannot be categorized is ambiguous, and its implications for allostasis remain unclear (thus, we are 'experientially blind' to such information).[148] To start making sense of the world, young children have a highly plastic brain and quickly learn to categorize and grow their concept repertoire.[50,141,149,150] Rapidly, they also learn to categorize the emotional meaning of facial movements based on the context.[151,152] Yet, because there are allostatic implications to categorizing, some concepts can be more 'adaptive' than others. Therefore, a critical function of early-life experiences and development, including interactions with parents, caregivers, educators and peers, is to aid the child to curate relevant information and build concepts related to their bodies' needs and the world that will enable them to perform allostasis more efficiently on their own.[153] In this way, sociocultural and environmental factors transmit emotion concepts to the new generations as a tool for regulating the body and each other.[50,51,154–156] Thus, 'supervised category learning' such as word learning is an important source of emotional development and cultural inheritance.[58,141,157,158] During development and the processes referred to as 'socialization via words'[157] and actions of others,[158–160] culture creates recurrent situations that allow one to learn specific, situated meanings of particular signals in the natural and cultural ecology of a person's environment.

The theory of constructed emotion has broad implications.[135] Neurotypical adults regulate their body by continuously updating well-fitting prediction models (including those related to emotion concepts) to efficiently meet the metabolic demands of the body. Therefore, the process of creating and revising emotion concepts occurs across the life span. Such continuous revisions are adaptive—the ability to take in prediction error, learn it, update the action plan (i.e. recategorize) and ultimately modify that model to issue better predictions next time. Early-life development plays an important role in guiding the acquisition and initial refinement of increasingly efficient (adaptive) concepts. For individuals who did not have a supportive and stable environment during development to help them discern important from unimportant signals for allostasis, this task might be performed less efficiently and distinctly across instances. Thus, not only might the efficiency of the predictive process be disrupted, but the mental representation of emotion concepts (and the real-time construction of an emotion category) may not be well-tailored to the immediate environment. For example, caregivers with active mental health conditions are less synchronous with children, and synchrony is important for the development of emotion concepts.[161] Early-life adversity can also bias concept creation to aid allostasis during hardship. Consequentially, limitations in updating predictions (e.g. prediction error learning) can sustain the longer-term use of a particular

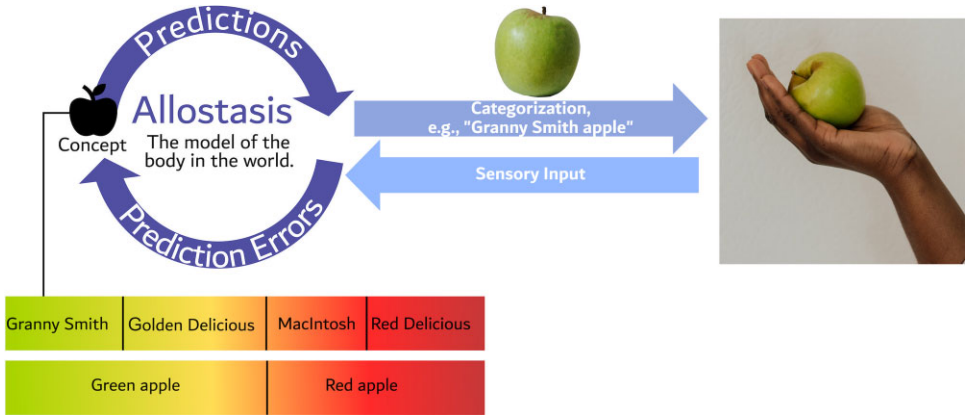

Figure 4 Concepts, predictions and the construction of categories. Concepts are abstract mental representations of features or instances that repeatedly occur together; the more granular a concept is, the more detailed and better defined the representation (e.g. knowing the difference between a Granny Smith, Golden Delicious, MacIntosh or Red Delicious apple, versus only knowing green and red apples). Individuals engage in allostasis by issuing brain-based situation-specific predictions based on available concepts. If the prediction matches the incoming sensory input, a category is constructed to reflect that instance (e.g. a Granny Smith apple). The incoming sensory input will then help improve future predictions through learning of prediction errors. If the instances that are grouped together share physical features, they are called concrete or perceptual categories (e.g. apples), otherwise they share mental features and are referred to as abstract, conceptual or functional categories (e.g. food). Moreover, the same object or event can be categorized as abstract or concrete depending on the situation: a grasshopper can be categorized as food in certain cultures, and as only an insect in other cultures. Thus, the category depends on the individual's goal in a particular situation.[138] Abstract, conceptual categories are easily mistaken for concrete ones. For example, a facial movement, such as smiling, is abstract because movements that look identical to the naked eye are variable under the skin,[57] and the same action can vary at the muscular level within one individual.[140] Similarly, scents where different chemicals produce the same smell, or phonemes where the sound of a letter changes in each word but serves the same phonematic function, are all abstract categories. In fact, categories that are assumed to be concrete or perceptual, such as dogs, flowers or weeds, can be understood as abstract, conceptual categories.[50,51] The ability to construct abstract categories is determined by the degree of compression in the features that the brain can support. The expansion of the human brain allows for compression and dimensionality reduction, suggesting that we can assemble multimodal summaries (i.e. features) in early infancy and later in life.[129,141,142] The pictures are in the public domain and taken from: https://www.pexels.com.

concept even after environmental demands change. At one end of the spectrum, differences in the mental representation/conceptualization of emotions can provide the basis for emotional granularity. At the other end of the spectrum, some individuals might not develop a given emotion concept at all. The long-term by-product of performing allostasis less efficiently is that the body's budget is in a chronic deficit: a general predisposing vulnerability for the development of psychopathology.[135,137]

Reformulating FND using the theory of constructed emotion

Using the theory of constructed emotion, we propose that functional neurological and functional somatic symptoms can emerge as a result of disruptions occurring across several points of predictive processing in the emotion category construction stream (Fig. 6). Note that the six proposals detailed next are inter-related and should not be interpreted as mutually exclusive. Their respective roles across individuals with FND may also vary.

Proposal 1: There is chronic energy mismanagement in FND

We posit that FND symptoms in some people reflect chronic difficulties in allostatic energy management through inefficient use of emotion concepts. This does not imply that affected individuals suffer from objective energy deficits, but rather that the inherent and continual allostatic processes are marked by suboptimal emotion construction. As less granular and efficient concepts such as 'fatigue' and 'unwell' supplant more fine-tuned (i.e. predictive) emotion concepts, this chronic inefficiency becomes perpetuated.[135,162] Despite the clinical heterogeneity of FND, fatigue is the most common shared symptom, reported by 93% of 1048 individuals surveyed across 16 countries.[6] Fatigue is highly prevalent and a chief predictor of quality of life in motor FND, more so than motor symptom severity.[6,163] Inefficient emotion construction can also lead to hypervigilance and hyperarousal. Hyperarousal is common in FND; this is exemplified by the observation of 'panic attack without panic' in people with functional seizures, as well as increased sympathetic tone and abnormal startle responses in FND populations.[21,46,164,165] Individuals with FND also have high rates of comorbid post-traumatic stress disorder where hyperarousal is a core symptom.[5] Elevated arousal, particularly when paired with negative valence, is known to trigger increased muscle tension that may relate to chronic pain[166,167]; similar associations are also well accepted for gastrointestinal distress.[168] Additionally, it has been proposed that mood disorders including major depression also relate to allostatic mismanagement.[50,86,113,135] Thus, the high co-morbidity between FND, select psychiatric conditions and functional somatic disorders suggest shared inefficiencies in allostatic energy management.[169,170]

Proposal 2: FND can occur in the context of aberrant emotion construction

In some people with FND, we posit that functional neurological symptoms occur due to deficits in the repertoire of available emotion concepts and/or impairments in constructing a conceptual category for an emotion in a particular instance. High arousal states that are frequently although not exclusively coupled with a negative valence (e.g. during an altercation or in the immediate aftermath of a car crash) are recognized to be associated with paroxysmal motor phenomena (e.g. tremulous movements, stuttering speech), perceptual distortions (e.g. dizziness, derealization)

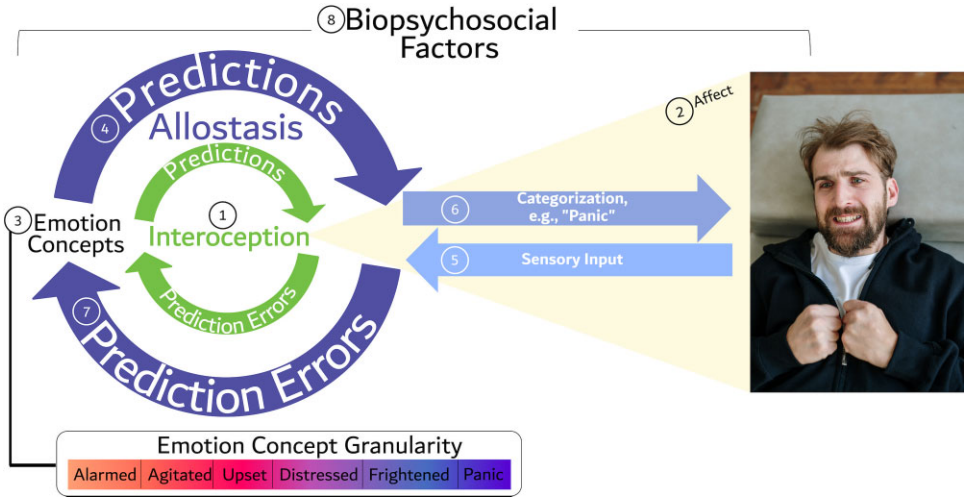

Figure 5 The theory of constructed emotion. Schematic representation of predictive processing and emotion category construction. (1) Interoception is the brain's modelling of the physiological state of the body with the goal of efficient regulation through allostasis. (2) This model of the bodily state is experienced as affect, which is a quality of consciousness that 'colours' predictions, concepts, perceptions etc., regarding the allostatic needs of the body. (3) The available repertoire and detailed granularity of emotion concepts depends on lived experiences. (4) The concepts that were relevant in similar past experiences will be used to issue predictions by preparing a motor action plan and a sensory experience. Concepts may differ in their granularity. (5) Incoming sensory input will be compared to the predictions: (6) if there is a match, a category that represents that instance can be constructed; (7) if there is not a match, the sensory input can be used to improve future predictions through learning prediction errors. Precision signals (not depicted here) tune predictions and prediction errors. (8) Biopsychosocial factors are relevant across the emotion construction process, and predictive processing more broadly. The picture used is in the public domain and taken from: https://www.pexels.com.

and cognitive symptoms (e.g. clouded thinking, word finding difficulties). In such instances, individuals may make sense of these physical symptoms by constructing a conceptual category of 'fear' or 'being shocked'. Symptoms such as the tremor, stutter and cognitive difficulties in such situations are both physiological and involuntary. If an individual lacks a relevant emotion concept or does not construct a conceptual category for an emotion in that instance, such experiences could be represented as a non-emotion category (e.g. 'shaking').

Additionally, heightened arousal is linked to defensive behaviours driven by the periaqueductal grey, such as tonic immobility and fight or flight responses—behavioural programmes that share overlapping semiological features with functional seizures.[45,171–174] By extension, some forms of functional weakness can be conceptualized as either a derivative of tonic immobility occurring during high arousal, or potentially a manifestation of a low arousal (and probably non-conscious low motivational) state. High arousal states—that are associated with increased muscle tension and chronic pain disorders—may also activate some of the motor programmes recruited in functional dystonia.[175] Indeed, most studies on FND onset have identified highly distressing physical events as triggers.[176–178]

We would also like to point out that not all forms of aberrant emotion construction are relevant to the development and maintenance of functional neurological and functional somatic symptoms. Rather, we posit that the availability and reliance on non-emotion, bodily/health-focused concepts (and the related pervasive tendency to categorize incoming afferent sensory information as non-emotion instances) is specific to functional disorders. Within this formulation, functional motor symptoms are postulated to be experienced along the spectrum of arousal and valence as detailed previously. However, aberrant emotion construction in general might be understood as a transdiagnostic vulnerability to the development of neuropsychiatric disorders.[135]

Proposal 3: There is altered prediction error learning in FND

We also put forward that some individuals with FND might be using inefficient allostatic and/or interoceptive models due in part to altered prediction error learning (including the influence of precision signals originating in the salience network). While individual differences are important, data indicate that some people with FND show deficits in sensory processing, interoceptive accuracy, biased attention and impairments in motor learning.[90,91,179–184] Thus, in addition to themes related to available emotion concepts and potential deficits in constructing a conceptual category for an emotion, chronic deficits in performing allostasis efficiently may be facilitated by a range of neuropsychological constructs that can interfere with prediction error learning. Of note, such a proposal for aberrant prediction error learning has also been suggested by previously developed Bayesian accounts of FND.[23]

Proposal 4: Alexithymia, 'panic attack without panic' and dissociation in FND can be reframed

Aberrant emotion construction can be used to re-conceptualize three longstanding mechanistic themes in the FND literature: alexithymia, 'panic attack without panic' and dissociation. Alexithymia, a characteristic described as difficulties identifying/describing feelings and having externally oriented thinking, is found in many people with FND.[18,185–187] Here, we suggest that in alexithymia there is a limited availability of granular emotion concepts, promoting inefficiencies in adaptively contextualizing sensory input.[50,51,148,188] 'Panic attack without panic' was characterized by Goldstein and Mellers in people with functional seizures to describe individuals who endorsed the autonomic symptoms that commonly accompany panic attacks, yet lacked an associated subjective emotional perception.[21] Interestingly, there is a similar paradoxical relationship between the perception

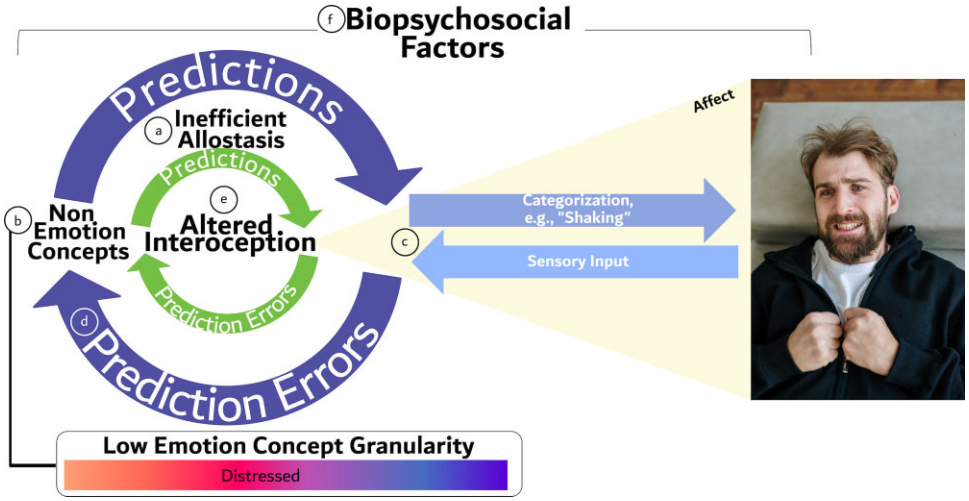

Figure 6 The theory of constructed emotion applied to FND. Illustration of posited points for aberrant emotion construction in individuals with FND. (**A**) Chronic energy mismanagement is present in some people with FND, leading to chronic fatigue and hyperarousal states among other symptoms. (**B**) Adverse life experiences may lead to a limited or lack of granular emotion concepts. Additionally, childhood maltreatment may aid the development of more bodily and health/illness related non-emotion concepts. (**C**) Incoming sensory input can match a prediction that does not have emotion content, and a bodily/illness category is constructed (e.g. 'shaking'). (**D**) Deficits in sensory processing, interoceptive accuracy, biased attention and impairments in motor learning among other constructs limit the use of precision signals and predictive errors to improve future predictions. (**E**) The momentary uncoupling of the brain's interoceptive and allostatic models may play a role in dissociative mechanisms. (**F**) Biopsychosocial factors can predispose, precipitate and/or perpetuate the deficits at each stage of the predictive processing stream. The picture used is in the public domain and taken from: https://www.pexels.com.

reported by people with functional seizures and their physiological response: individuals who reported a subjective emotional response to certain images had reduced sympathetic responses compared to those who did not endorse intense emotional responses but exhibited large autonomic changes.[41] Given that individuals with functional seizures frequently also experience panic attacks[189] (suggesting the potential availability of the 'panic' emotion concept), 'panic attack without panic' can be understood as an instance where a conceptual category for an emotion was not constructed.

Dissociation is a dimensional construct seen across a range of trauma-related disorders that can be defined in part as the compartmentalization of perceptions with detachment from oneself (depersonalization) or the outer world (derealization), or the hyperregulation of negative emotion (note, a comprehensive review of dissociation is beyond the scope of this article and is provided elsewhere[190]). FND ranks fourth among 19 common psychiatric conditions in terms of the highest self-reported dissociation scores. In FND populations, elevated dissociation has been related to anxiety levels,[21] lower interoceptive accuracy,[184,191] maternal disfunction,[192] other psychiatric comorbidities[169] and childhood trauma.[169] While dissociation is probably mechanistically heterogeneous, dissociation within the theory of constructed emotion framework could be conceptualized in part as the momentary uncoupling of the brain's higher-level generative models of the internal state of the body (interoception) and of the body in the world. This uncoupling could then be experienced as a dissociative event, where the conscious feature of interoception, affect, does not 'match' the constructed experience in the service of allostasis.

Proposal 5: The theory of constructed emotion helps to contextualize the debate on emotion in FND

Individual differences in emotion construction (including the tendency to use bodily and health/illness concepts where others would construct instances of emotion) helps explain the paradox that some people with FND can be evaluated by clinicians as appearing 'anxious' while the individual themselves denies the presence of negative emotions. Here, the clinician is responding to outward cues reflecting a state of heightened sympathetic arousal and constructing a conceptual category for emotion, inferring the emotional state of the individual with FND. Conversely, the predictions and pattern completion of incoming sensations by the person with FND does not construct a conceptual category for emotion, and thus the high arousal and (possibly) negatively valenced affect are not experienced as an instance of emotion. Importantly, this paradox gives insight into an important feature of emotion communication: synchrony of conceptual meanings and non-verbal communication between people is what is important, not accuracy in guessing someone else's instances of emotion.[53,193,194] In fact, an implication of the theory of constructed emotion is that humans cannot know the subjective emotional experience of someone else.[50]

Proposal 6: The repertoire of concepts and their refinement is negatively affected by adverse life experiences

Stress-diathesis and neurodevelopmental perspectives have important roles in aetiological formulations for FND.[195] Within this context, life experiences feature prominently into the relevance of the theory of constructed emotion for FND: while not all individuals with FND report a history of early-life adversity or antecedent trauma, adverse life experiences are ~3-fold more common in FND versus controls.[196] Importantly, cohort studies have identified positive associations between the magnitude of previously experienced adverse life experiences and functional neurological symptom severity, underscoring the aetiological importance of adverse life experiences in this population[169,192,197–199]; relatedly, abuse is associated with early symptom onset in people with FND.[200] Here, we theorize that aberrant emotion construction and related factors contributing to inefficient allostatic modelling (e.g. insecure

attachment) may mediate correlations between FND symptom severity and adverse life event burden.

Individuals use concepts generated in the default mode network that are challenged or confirmed throughout the processing stream where top-down signals are compared to incoming information to predictively complete patterns.[50,51] The content of predictions begins with visceromotor plans that gain skeletomotor qualities as the predictions run through the cytoarchitectural gradient.[50,51,123] The sensory predictions and the experiences that arise from them are a consequence of the motor predictions.[50,51] Thus, disruptions in the development, availability and usage of concepts may also potentially lead to motor and/or sensory disturbances. Relatedly, life experiences, especially those in critical developmental stages, form the basis of concept formation, which might be updated throughout life to enable a more fine-grained repertoire of (emotion) concepts. We support the notion that adverse life experiences (particularly childhood abuse and neglect) prime concept formation that can have long-term disadvantages as environmental conditions change, leading to maladaptive predictions. In other words, concepts developed in the face of early-life adversity that were adaptive in that context, might prove inefficient later in life. For example, if an individual grows up in a highly physically threatening environment (e.g. experiencing physical or sexual abuse), a focus on predictive models favouring a strong motor component would be advantageous (metabolically efficient); in exchange, categorizing the same sensory input as a self-referential emotional state would probably be less adaptive at that instance. As conditions change, the visceromotor, skeletomotor and psychological qualities of the constructed concepts would (hopefully) also change. Relatedly, the human brain is most neuroplastic earlier in life, suggesting that the qualities of certain acquired concepts may be carried forward and less malleable to modification later in life. Within this context, an individual with FND who experienced previous post-traumatic stress disorder symptoms may indeed report that such experiences are seemingly no longer consciously relevant, yet the predictive processing consequences of these past experiences can persist. Additionally, there are a range of other biopsychosocial factors that could influence the repertoire of emotion concepts and the process of constructing a conceptual category for an emotion in a given instance. For example, intellectual disability and autism spectrum disorder are often associated with alexithymia and are known risk factors for FND[201,202]; we speculate that developmentally mediated disruptions in emotion construction play a role in the increased propensity for functional neurological symptoms in these populations.

Furthermore, caregivers early in life perform allostasis for newborns by managing their entire energetic state (e.g. their sleep, food, temperature).[50] Then, as children grow, caregivers curate the world for them so they can start learning concepts through life experiences.[50,159] Gradually, the child starts increasing the availability and accuracy of their concepts to slowly start performing allostasis on their own.[50] Therefore, interpersonal interactions are foundational to the way that humans learn to efficiently perform allostasis and thus regulate interoception, and therefore affect.[159,203] Thus, insecure attachment is evidence of the inefficient allostatic model a person has been running to regulate their body's budget with others. While not always present, insecure attachment is commonly identified in paediatric and adult populations with FND[204,205]; in this population, insecure attachment has also been linked to increased symptom severity, childhood maltreatment and poor clinical outcomes.[206]

Exploring potential reinterpretations of select FND findings using the theory of constructed emotion

While a retrospective reinterpretation of published findings in the field is fraught with potential biases, this next section revisits select results to illustrate the use of the theory of constructed emotion in providing an alternative viewpoint on the neurobiological understanding of FND.

Neuroimaging studies in FND cohorts using a range of imaging modalities have detected subtle functional and structural alterations compared to healthy controls, as well as correlates of illness severity and predisposing vulnerabilities.[8,207,208] While findings have not been entirely consistent across studies and FND sub-populations, between-group differences in default mode, salience and sensorimotor network areas have been identified in a range FND populations compared to controls.[45,207,209–212] These findings speak to the previously mentioned mechanisms of concept generation (default mode network) and precision signalling (salience network), as well as the importance of interoceptive and allostasis-related predictions and prediction errors that might be altered in FND. For example, findings of amygdala hyperreactivity and enhanced amygdala/cinguloinsular—motor control network connectivity[45,213–216] in people with FND could be interpreted as an indication that there is too much ambiguity/uncertainty, either because the brain was wired in a chaotic environment and so has a model of the world as unpredictable, or because the brain cannot adjust to a novel situation/environment due to some metabolic deficit that inhibits encoding prediction error. Increased amygdala/cinguloinsular—motor control network connectivity may also represent biased predictions for the need to engage a motor action plan (as would be expected in the setting of previously experienced physical abuse[217]). Additionally, several functional and structural neuroimaging studies have related self-reported symptom severity and/or FND risk factors to alterations in default mode and/or salience network brain areas.[172,199,217–220] While principles of degeneracy and domain-generality suggest that there is probably not one universal neural signature for FND, the propensity to develop and maintain functional neurological and functional somatic symptoms may occur in the setting of inefficient emotion construction driven by perturbations within and across the default mode, salience and/or sensorimotor networks.

Relatedly, interoception is a construct of high theoretical interest to the pathophysiology of FND.[26] In the only published neuroimaging study to date probing brain–interoception relations in FND, interoceptive trait prediction error correlated with reduced white matter integrity of fibre bundles originating from the bilateral insula and temporoparietal junction among other areas.[182] Functional and structural alterations of the insula (found in several FND neuroimaging studies) might lead to imprecise predictive interoceptive signal modelling and error detection, contributing to inefficiencies in performing allostasis.[95,104,112,135] Such 'miscalculations' could predispose to chronic negative affect and altered predictions, which offers an explanatory framework for behavioural and physiological findings of altered responses to 'emotional' stimuli in people with FND.[27,28,44] To date, interoceptive abilities in FND cohorts using heartbeat-related detection and counting tasks have had mixed results—with reports of decreased interoceptive accuracy at baseline or following induction procedures, or with no differences compared to healthy controls.[182,184,191,221] This inconsistency may relate in part to the notion that the brain's modelling of the condition of the body is mostly not accessible to awareness. Therefore, awareness measures only assess

one small facet of interoception. Nonetheless, interoceptive accuracy deficits are relevant when present, such as interoceptive trait prediction error positively correlating with dissociation severity in individuals with functional seizures.[184]

Other research has investigated autonomic and neuroendocrine profiles in FND.[46] There is evidence that children and adolescents with FND exhibit increased HR and lower heart rate variability (HRV) compared to healthy controls or normative data.[222–224] While HR and HRV findings are more heterogeneous in adults, people with functional motor symptoms have exhibited increased resting HR and decreased HRV compared to healthy controls.[225] In terms of endocrine findings, cortisol levels correlated with trauma burden[226,227] and attentional bias to threat stimuli[39] in people with FND. While these findings, particularly those related to the 'stress hormone' cortisol, are commonly framed as markers of 'emotion dysregulation'—the theory of constructed emotion would reframe this as representing inefficient energy management.[113] Cortisol, with a primary role in stimulating gluconeogenesis, might be understood to be a metabolic rather than a 'stress' hormone.[228,229]

Therapeutic implications, future research directions and limitations

Our proposals have several implications. (i) We argue that the altered emotion construction in some individuals is sufficient to mechanistically explain the development and maintenance of FND and related functional somatic symptoms. (ii) When people with FND state that they are not anxious or depressed, despite externally presenting information seemingly to the contrary, they are correct. (iii) From a treatment perspective, an important therapeutic element may be guiding the individual with FND to re-attribute bodily symptoms to newly developed or more granular emotion concepts that aid more efficient performance of allostasis. This contrasts with certain psychodynamic theories arguing that a discrete emotion concept has been formed but is repressed or 'converted'. From a constructionist's perspective, the treatment setting may be the time when a given emotion concept is acquired in the first place. (iv) Furthermore, therapies that focus on learning how to detect, regulate and fulfil the body's needs (i.e. to perform 'allostatic maintenance' through behavioural and lifestyle changes) warrant research in FND. Dual use of bottom-up (e.g. sensorimotor psychotherapy) and top-down (e.g. cognitive behavioural therapy) approaches may also have complementary therapeutic benefit.

From a research perspective, there are several lines of investigation that can help support or refute the role of aberrant emotion construction in FND and related conditions including: (i) experimentally quantifying emotional granularity levels through multiple experience sampling in people with FND compared to healthy and neuropsychiatric controls, and relating individual differences in emotional granularity to clinically relevant characteristics[230]; (ii) comprehensively interrogating interoceptive abilities in individuals with FND using experimental paradigms beyond heartbeat counting tasks, such as peripheral and/or central perturbations (e.g. inspiratory breathing loads, pharmacological adrenergic stimulation, placebo/sham interventions etc.) to test both the afferent and efferent components of interoception within the context of multisystem assessments[231]; (iii) given the central role of cytoarchitecture to emotion construction and predictive processing more broadly, high field strength brain imaging (i.e. 7T MRI) could be used to investigate cytoarchitectural profiles in people with FND compared to controls, as well as probing potential relationships between age of onset, illness duration, symptom severity, treatment responsiveness and hierarchical neurocomputational gradients[232,233]; and (iv) from a treatment perspective, prospective qualitative and quantitative research could investigate if people with FND that fully recover (or significantly improve) do so in the context of refining their repertoire and use of emotion concepts (e.g. does treatment responsiveness correlate with changes in emotional granularity and a reconceptualization of physical symptoms).

Importantly, the wide clinical heterogeneity of FND suggests that any pathomechanistic principle outlined here might not be universally relevant to symptom production across all cases. Thus, the theory of constructed emotion framework provides insight into potential mechanisms most relevant for people with FND who report alexithymia, 'panic attack without panic', comorbid psychopathology, fatigue and/or a high burden of early-life adversity. Furthermore, we think that other proposed emotion processing mechanisms for FND are not incorrect, but rather incomplete—with a constructionist perspective addressing an important gap.[27,28] It is noteworthy to also point out that among cognitive-affective neuroscientists, there remains active debate regarding the relevance of neuroconstruction to emotion[47,234]—and there are other constructionist perspectives on emotion apart from the theory of constructed emotion that warrant further consideration.[235,236] Additionally, since this article offers a framework for investigating the role of emotion construction in FND and related functional somatic disorders, specific pathomechanistic evidence in these conditions is still needed. Lastly, since FND is mechanistically and aetiologically heterogenous, a range of other mechanisms articulated in the field remain relevant.[8,22,23,26–29]

Conclusions

In conclusion, the theory of constructed emotion offers a framework for reconceptualizing and integrating the relevance of emotion (and its aberrant construction) in the pathophysiology of FND. Research efforts are needed to test the theory of constructed emotion framework for FND.

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Competing interests

D.L.P. has received honoraria for continuing medical education lectures in functional neurological disorder, royalties from Springer for a book on functional movement disorder and is on the editorial board of *Epilepsy & Behavior*. L.F.B. receives royalties from her book *How Emotions Are Made*. S.P. receives royalties from Springer for a book on functional neurological disorders, and has received speaking fees from Novartis and Esai.

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