Obrela JOURNAL Dialogues in Clinical Neuroscience & Mental Health

e-ISSN: 2585-2795 • Printed-ISSN: 2654-1432
DOI: 10.26386/obrela.v4i1.146

Emotional intentionality and predictive processing

Orestis Giotakos

Abstract

The detection and the prediction of changes of the world incorporate processes enabling an automatic coding of sameness. Predictions can be formed in relation to stimulus timing, location, or content, while it is not yet clear whether they concern a unitary neural process or an array of independent, task-tailored mechanisms. Predictive activity is indispensable and vital for survival, and interoceptive inference remains influential in understanding the predictive processing, since it brings closer the phenomenology of consciousness with that of emotionality. On the other hand, intentionality seems to have essential relations with both consciousness and evolutionary selected functions. Phenomenologists suggested that affective intentionality is an embodied and enactive process that connect us to a shared world and guide our dealings with it. The present article poses the question whether the concepts of emotions, intentionality, and moreover emotional intentionality, can bridge the gaps in the predictive processing framework. Many relative concepts, theories and findings suggest that these dynamic domains, acting as hidden aspects in this framework, can give a thorough *understanding* of its functioning.

Key words: Emotions, intentionality, emotional intentionality, predictive processing, predictive coding, predictive brain

Corresponding Author: Orestis Giotakos (MD, MSc, PhD), Founder, npo "obrela", Immitou 112, Athens, 11634, Greece, e-mail: info@obrela.gr

DOI: 10.26386/obrela.v4i1.146

The perceptive brain

Our understanding of how consciousness is acquired seems to follow the logic of the *Cartesian theater*, where the information is presented and promoted to the audience **[1]**. It seems that we become aware of the representations after they are entered into a *conscious-awareness system*, which inscribes the information on some "scratch pad", to be able to be viewed by an internal scanning eye **[2, 3].** The brain extracts regularities from streams of sensory input, through passive sensory experience, self-generated actions, and exploration of the environment, while these expectations influence sensory processing **[4]**. Any new event initially activates all networks. The following events are likely to proceed simultaneously and iteratively as a rapid of succession of scripts, scenarios, and hypotheses: (1) perceptual identification, (2) deployment of spatial attention, (3) lexical labeling, (4) association with past experiences, (5) linkage to emotional and visceral patterns, (6) assessment of present context, (7) planning of options, and (8) prediction of consequences. The current goals and constraints of the individual are then expected to determine the most relevant ensembles or networks that will dominate the landscape of neural activity. Thus, the solution to a cognitive problem or task would require the settling of the entire system into a state of best fit.

The conscious awareness of some sensory experiences seems to be delayed by up to 500 ms beyond the time of the initial cortical response, during which the incoming impulses appear to acquire "neuronal adequacy". It appears that the accessibility of an event to explicit consciousness and introspective commentary is the byproduct of a special type of cortical activity and not an automatic consequence of sensory encoding **[5]**. The hippocampal formation serves an integrative function, linking together the various neuronal assemblies that are activated as a synchronized network. At this point, it is the thalamus and the 40-Hz gamma-band activity that binds, in real time, the various neuronal assemblies mentioned above, each oscillating at its own respective frequency **[6]**. It has been suggested that the brain is essentially a closed system capable of self-generated activity, based on the intrinsic electrical properties of both the component neurons and their connectivity. In such a view, the central nervous system is a *reality emulating system*, and the parameters of this inner reality are delineated by the outer senses. The hypothesis that the brain is a closed system follows from the observation that the thalamic input from the cortex is markedly larger than that from the peripheral sensory system. Thus, the majority of cells in the CNS are thalamo-cortical neurons, sensory or motor, which do not communicate with the outside world. Neurons with intrinsic oscillatory capabilities that reside in this complex synaptic network, allow the brain to self-generate dynamic oscillatory states, which shape the functional events elicited by senses, suggesting that the thalamo-cortical activity may be the basis for perception and consciousness **[7]**. Furthermore, perception can be regarded as providing veridical predictions about both exteroceptive and proprioceptive sensations. Perception is related through a common minimization of prediction error, while the subjective sense of reality depends on the successful suppression by top-down predictions of informative interoceptive signals **[8]**. When a stimulus is expected, beta-band activity (12–30 Hz) gradually builds in stimulus onset, and gamma activity (>30 Hz) is reduced when those expectations are realized. The violation of expectations is associated with increases in gamma activity, and beta oscillations are initially reduced before resynchronizing **[4]**.

The brain balances its ability to both *integrate* and *segregate* information. The optimal balance between functional integration and segregation is obtained at an intermediate structural connectivity between order and randomness. Lord et al **[9]** suggested that "as the structural connectivity gradually changes from an ordered lattice to a disordered graph, *perturbational integration* decreases because randomness shortens the length of the largest component in the network, while *perturbational segregation* follows the opposite trend, because randomness increases the capability to distinguish between two different external inputs". Similarly, *deep learning* and *neuromorphic engineering,* a relatively recent interdisciplinary research field, that attempts to simulate neurons and synapses directly on hardware, knows that executing trained neural networks on *neuromorphic platforms* comes

DOI: 10.26386/obrela.v4i1.146

with large energy savings and lower prediction latencies. The advantage of this approach is that because neurons are simulated in an asynchronous manner, the overall energy consumption is very low since neurons that do not participate in the computations consume nearly zero energy **[10]**.

Self-awareness monitoring

Multiple neuropsychological theories of *self-awareness* emphasize the role of an *error-monitoring system,* which consists of three parts: (a) An internal representation of the desired outcome, (b) a feedback related to the outcome and (c) a comparison between the desired and final outcome. *Anosognosia* or the overall *lack of insight* may result from fault in the mechanisms determining the desired outcome, or disturbances in the process of comparison. McGlynn & Schacter **[12]** suggested the presence of a *conscious awareness system* located in the inferio-parietal lobe, while Shallice **[2]** presented the case of the *supervisory attentional system.* The theoretical approaches regarding impaired insight include the disturbed perceptual input, the impaired linkage between thought and emotion and the breakdown of the process of self-monitoring and error checking. The inability to distinguish between internally and externally generated mental events has been described by the *meta-representation theory*. This theory includes the awareness of ones' goals, which leads to disorders of willed action, the awareness of intention, which leads to movement disorders, and the awareness of intentions of others, which leads to paranoid delusions.

The most prominent theoretical perspectives that attempt to account for the development of lack of self-awareness or lack of insight are summarized as follows: (1) The *perceptual input* is impaired, as occurs with the loss of hearing or vision, which may in turn lead to paranoid ideation or other abnormal perceptual experiences, such as hallucinations. (2) There is a disruption in the *inferential process*, where the patient fails to recognize the consequences or the results of actions. (3). The *process of self-monitoring* is disturbed, and the experience of consciousness altered, disturbing the distinction in the perception of internally and externally produced phenomena. For instance, a patient with auditory hallucinations may be engaged with inner speech, but he may not be aware of the fact that this speech is self-produced. (4). The processes of error checking may be disturbed, resulting in the disruption of the ability to doubt and discriminate what is indisputable from what may be possible or impossible. (5). The linkage between thought and affect is disrupted, leading to out of proportion affective reactions. (6) There are weaknesses in certain capacities, such as the capacity to hold on to a memory representation, organize a task and maintain the effort until successful completion **[13]**. In the case of *self-deception*, the lack of awareness tends to be global and incorporates large chunks of psychological material. Moreover, it is often associated with an individual's motivational components, something not evaluated in the cases of insight in schizophrenia**.** Sackeim & Gur (1978) **[14]** suggested the following criteria to describe the phenomenon of *self-deception*: (1). the person has two mental contents, that are conflicting when expressed as propositions, (2). these two mental contents occur simultaneously, (3). The individual is not aware of one of the two mental contents, (4). The process that defines which mental content is subject to awareness depends on the individual's motivation.

According to the long term studies by Petrides **[15]** in humans and monkeys, cortical lesions and damage in the medial part of the mid-dorsolateral prefrontal cortex (BA 46 & 9/46) lead to deficits in tasks of the monitoring of information in *working memory,* where the capacity for an *epoptic processing* of information is evaluated, while the architectonic areas 46 & 9/46 of the prefrontal cortex appear to be linked with specific segments of the inferior parietal lobe through the superior longitudinal fasciculus. The inferior part of the posterior parietal cortex seems to be a crucial area for the updating of information in the working memory and the BA *46 & 9/46* encode it into an "abstract/symbolic form", in order to achieve the controlled monitoring in the active mnemonic process. This system has the capacity to hold symbolically coded information in an active state, in order to supervise the between them relation and their relation with the intended programmed behavior.

DOI: 10.26386/obrela.v4i1.146

The *supervisory attentional system* postulated by Shallice **[2]** incorporates the prefrontal cortex as a core structure. Indeed, the prefrontal cortex seems to play a role in this system in various ways. For example, both the visual perceptual process and visual mental imagery seem to stimulate similar neural networks, such as the primary visual cortex, the posterior parietal cortex, temporal areas and the dorsolateral prefrontal cortex. Yet, an important distinction arises in the fact that neural networks involved in imagery result to the triggering of top-down processes, whereas neural networks in perception are associated with bottom-up processes **[3]**. Movement behavior begins with projections from the prefrontal cortex to the striatum, followed by the globus palidus and from there to the anteroventral and ventrolateral thalamic nuclei, which in turn project to the premotor area and the supplementary motor area and to the anterior cingulate **[16]**. Malfunction in any of these areas may lead to disorders such as Parkinson's disease, while the positive symptoms of schizophrenia have also been associated with a failure in monitoring movements, accounted for by deficits between premotor areas and the striatum. This is supported by the symptom of a sense of movement, as if controlled by external forces, in patients with *Metachromatic Leukodystrophy*, where the white matter connectivity of several cortical areas, and in particular the frontal cortex is upset **[17]**. Moreover, it seems that disruption in myelination and dysmyelination-induced delays in information processing can produce phenocopies of psychosis similar to schizophrenia **[18, 19].**

The predictive brain

Permanent predictive activity is indispensable and vital for survival. According to Friston **[20, 21]**, "the brain is a constructive or predictive organ that actively generates predictions of its sensory inputs using an internal or generative model". He suggest that the most important determinants of our behaviour, and their underlying predictions, are beliefs about the intentions and behaviour of others, which requires an internal model of self in relation to others and an implicit sense of agency. Craig's **[22]** model of *sentient self* places the insula at a central role for evaluating ongoing feeling states. According to Kirov **[23]**, *predictive coding* is continuously modulated by external environmental or internal mental information. He suggested: "predictive coding is a neurocognitive concept, according to which the brain does not process the whole qualia of external information, but only residual mismatches occurring between incoming information and an individual, inner model of the world, thus minimizing the free energy or brain entropy". Predictive coding appears as a universal evolutionary pathway. However, which neurophysiologic mechanisms support the formation, maintenance and consolidation of the inner model determining predictive coding?

First, the ultimate and sophisticated insular activity is often seen with prediction error coding, which refers to a discrepancy between an expectation and its occurrence and therefore the updating of expectations about the external and internal milieu and the corresponding modification of action. The ventral tegmental/substuntia nigra was found to have also an active participation in this process **[24]**. The insula plays a role in not only error evaluation but also updating the probabilities of an outcome **[25]**. Moreover, the insula has also been described as a "hub" for autonomic, affective, and cognitive integration, and it is associated with a wide array of stimuli, including cognitive, socio-emotional, olfactory-gustatory, interoceptive sensation, and pain processing. Therefore, the insula may be a contributing neural correlate of a fundamental awareness of reality. It seems to act in order to shift the mind from a state of self-referential perception of sensation to a state of all-inclusive, present-centered awareness. **[26]**.

Second, the *default mode network* comprises an integrated system for autobiographical, self-centered, and social cognitive functions characterized by *ruminative*, often *subconscious*, self-referential *narrative thought* linking subjective experience across time—which generates one's concept of *self* or *identity*. It is a consistent pattern of deactivation across a network of brain regions during focused mental tasks. This brain network includes the *posterior cingulate cortex*, which is associated with autobiographical memory and self-referential processes, the *ventromedial prefrontal cortex*, involving social-cognitive processes related to self and oth-

DOI: 10.26386/obrela.v4i1.146

ers, the *medial temporal lobe*, involved in episodic memory, and the *angular gyrus*, involved in semantic processing **[27]**.

Third, the self-reference neural mode associated with the *salience network* acts as a "switch" between the activation/deactivation of the central executive network and default mode network, depending on the degree of mental engagement. The salience network is anchored in the *anterior insula* and the *anterior cingulate cortex*. The awareness of any object requires, first, a mental representation of oneself as a feeling entity; second, a mental representation of that object; and third, a mental representation of the salient interrelationship between oneself and that object in the immediate moment. These three aspects suggest that a primary role of the *salience network* is the integration of sensations, internally generated thoughts and information about goals and plans in order to update expectations about the internal and external milieu and, therefore, the initiation or modification of action **[27]**.

The detection of change incorporates processes enabling an automatic registration of "sameness". The *mismatch negativity* (MMN) is a response to a deviant within a sequence of otherwise regular stimuli. According to the MMN model, a prediction error signal occurs when the brain detects that the present state of the world violates a context-driven expectation about the environment **[28]**. The auditory MMN can occur in response to deviance in pitch, intensity, or duration, while the visual MMN can occur in response to deviance in such aspects as colour, size, or duration. The auditory MMN is created in the primary and non-primary auditory cortex and the visual MMN in the primary visual cortex, while both have a typical latency of 150-250 ms after the onset of the deviant stimulus. Many studies have suggested MMN as probably one of the most consistent electrophysiological signatures of schizophrenia. The duration of MMN has been found significantly reduced in at-risk subjects converting to first-episode psychosis, which means that it may contribute not only to the prediction of conversion but also to a more individualized risk estimation and thus risk-adapted prevention **[29]**.

Theories of self-monitoring and error-checking agree with theories concerning the function of a salience network in psychosis. According to Corlett et al **[30],** there are two *types of error prediction* associated with schizophrenia and the development of delusional beliefs, playing opposite roles: one that overweights the prediction versus one that underweights the prediction. The *over-weighting of the prediction* may be prioritized due to its pathogenetic nature, occurring first, and is followed by the *under-weighting of the prediction,* which bears as a result a state of fatigue and withdrawal. Neurobiologically, the hyperactivation of the salience network is likely followed by the hyper-activation of the default mode network and subsequently by the suppression of the salience and attention network. This initial hyperactivation seems to be normalized by antipsychotics **[31].**

The emotional brain

The *cognitive theory of emotions* **[32]** argues that the main function of emotions is to coordinate the architecture of the brain modules and that the emotions enhance adaptation to the continuous environmental challenges and opportunities presented throughout the evolution of the species. According to this theory, the emotions are triggered when the person feels that the progress of his / her current goals is threatened or that it requires an adjustment. The emotions appear in order to organize and redirect the activity of the individual. According to this approach, when a person faces the failure to achieve a basic goal or the loss of an active goal, this causes the emotion of sadness or unhappiness. Thus, depression may be related to a person's tendency to interrupt any movement or to withdraw, which gives him the significant benefit of taking care of themselves. Therefore, it seems that emotions are functional because they offer the individual the opportunity to evaluate their continuous and goal-related activity and then to guide their behavior in a way that it reacts to the meaning of these signals **[33, 34]**.

For Barrett **[35]**, emotions are constructions of the world, not reactions to it. The modern neuroscience approaches consider emotions as *super-ordinate mental programs that "orchestrate"* and prioritize the functioning of the entire set of mental programs when critical events arise. According to Cosmides & Tooby **[36]**, "each emotion entrains various other adaptive programs - deactivating some, activating others, and adjusting the modifiable parameters of still others - so that the whole system operates in a particularly harmonious and efficacious way when the individual is confronting certain kinds of triggering conditions or situations". Thus, the protective function of the emotions is supported, through their ability to orchestrate and organize the priority of brain programs.

Conceptualising emotions in terms of a *contextualisation* of bodily states has historical roots dating back to the James' theory of emotion and two-factor theory of emotion **[37]**. According to the "theory of constructed emotion" **[35]**, emotions are constructed in the same manner as percepts, where priors are recruited according to context to make a "best guess" at the hidden causes of (interoceptive) sensory signals. Interoceptive inference is experienced as emotion in service of producing allostatic action. Allostasis (predictively regulating the internal milieu) and interoception (representing the internal milieu) are at the functional basis of the nervous system. Emotions on this view are "constructions". There are no neural or physiological signatures that reliably discriminate *any* emotional state. Physiological reactions in the body occur in order to prepare it for action, and are categorised as emotions only contextually. For example, heart rate increases or decreases depending only on an anticipated action and given an emotional ascription only contextually. The same bodily state could be categorised as fear in one context and anger in another **[38]**.

Associative network models of emotion suggest that when an *emotion unit*, such as the unit representing "happiness", is triggered over a certain threshold, the activation is spread throughout the network of associative information, resulting in all the "happiness"-related reactions of the autonomic nervous system, expressive behaviors, emotionally charged events and personal memories being activated and entering more or less into consciousness. When a person feels *happy*, mnemonic "material" associated with happiness is activated, resulting in an increase in heartbeats and blood pressure, activation of the zygomaticus major muscle and increased accessibility, of cognitive type, to words or memories associated with *happiness*. In addition, nodes representing "opposing" emotional states such as "*sadness*" and "*happiness*" are linked to inhibitory interconnections, resulting in the activation of one emotion leading to the inhibition of the other **[39]**. Similarly, according to the semantics of emotion, the phenomenon of *affective agreement* can be observed when the perceptual stimuli are associated with the same, categorically distinct, affective state, such as that experienced by the participant in a perceptual task **[40]**. The *associative network models of emotion* can predict two different phenomena in retrieval of information from long-term memory. (a) *Mood state-dependent memory* refers to enhancing the recall of information whose affective meaning fits with the current affective state of the individual. Even if the information is neutral, it is important to match the emotional state during encoding and during recall. (b) *Mood-congruent memory* is characterized by an increase in the recall of information that the emotional meaning matches with the current emotional state **[41].**

Emotions and interoceptive predictive processing

Interoceptive sensitivity is a characterological trait that reflects individual sensitivity to interoceptive signals, while active *interoceptive inference* depends on the selective attenuation of attention to interoceptive prediction errors. The *interoceptive concept of emotion* was first described by James, who argued that emotions arise from perception of changes in the body. This basic idea remains influential more than a century later, underpinning frameworks for understanding neural substrates of emotion, such as the "somatic marker hypothesis" **[42]** and the "sentient self" model **[22]**, both linked to the notion of "interoceptive sensitivity" or "interoceptive awareness" **[43]**. The *theory of constructed emotion* **[35]**, posits that the primary purpose of a brain is to predictively regulate physiological resources to coordinate the body's motor activity and learning in the short term, and to meet the body's needs for growth, survival, and reproduction in the long term. Barrett suggests that "In this view, all mental events —cognition, emotion, perception, and action— are shaped by allostasis, and thus all decision making is embodied, predictive, and concerned with balancing

DOI: 10.26386/obrela.v4i1.146

energy needs… We also posit a key role for the autonomic nervous system (ANS) in regulating short-term energy expenditures, such that the ANS influences experience and behavior under stressful circumstances" [**35**].

The model of "interoceptive predictive coding" **[43]** suggests a new view of emotional feelings as interoceptive inference. Chronic anxiety has been suggested to result from heightened interoceptive prediction error signals, while disrupted interoceptive predictive coding may causally account for many other psychiatric disorders. By analogy with comparator models of schizophrenia **[16],** Seth & Critchley **[43]** suggested that dissociative symptoms, notably depersonalization and derealization arise from imprecise interoceptive prediction error signals. According to Friston **[21]**, "prediction errors are simply the difference between the representations encoded at any level in the hierarchy and the top-down predictions generated by the brain's internal model… prediction errors are not just suppressed by optimising top-down or descending predictions but can also be reduced by changing sensory input. This does not necessarily mean visual or auditory input but the proprioceptive input responding to bodily movements".

The initial assessment of the amygdala is sent to the anterior circulate and to the orbitofrontal cortex for further evaluation **[44]**. Other information that can also be passed on is the facial expression or the gaze direction of others or even aspects of non-verbal behavior that reveal the intentions of others. These areas also record the bodily state and directly affect its activation state. Information from these areas is also transmitted to the hippocampus for ''cognitive mapping'' and in some cases, it is transferred to explicit memory. The orbitofrontal cortex plays an important role in coordinating these assessment procedures with more complex representations of the social context and the symbols in the neocortex **[45]**. During this process, it is understandable that the initial undifferentiated emotional states in the limbic system, through the mediation of feelings, are transformed into *nuanced emotions***.**

Converging clinical and neuroimaging findings suggest that the a*nterior cingulate* mediates modulation of emotion, cognition, sensation, and movement, mobilizes the appropriate responses to internal and external stimuli, integrates the emotional-cognitive responses and coordinates motor preparation, and conflict monitoring. The *anterior cingulate* carries out these functions by activating somatic states that focus attention on internal and external demands and motivate appropriate action through its projections to *autonomic*, *visceromotor*, and *endocrine* systems **[43]**. One of the main functions of the *anterior cingulate* is the regulation of bodily states of *arousal* to meet concurrent behavioral demands expressed by the social context with either threatening or benign stimuli. Representations of others' facial and bodily movements appear to involve the *anterior cingulate* and the *insula*, which together form a "salience network" (aberrant salience) that is capable to segregate the most important internal and distinct personal stimuli in order to guide behavior. Additionally, the *anterior* and posterior *insula* appear to interact as a hub, integrating "salient" *stimuli* and *events* with *visceral* and *autonomic* nervous system activity. Together they appear to help to generate a heightened physiological awareness of salient stimuli and appropriate behavioral responses **[46]**.

Anterior insular cortex provides a natural locus for comparator mechanisms underlying interoceptive predictive coding, through its demonstrated importance for interoceptive representation **[22]**. Anterior insular cortex is also rich in Von Economo neurons (VENs), large projection neurons which are circumstantially associated with self-consciousness and complex social emotions. In Seth Critchley model **[43],** fast VEN-mediated connections may enable the rapid registration of visceromotor and viscerosensory signals needed for efficient updating of generative models underlying interoceptive predictive coding. According to Porges's *polyvagal theory* **[47]**, the neurological basis of social engagement is evolutionarily linked to the autonomic nervous system and how it relays emotional experience. Porges proposes three parts of the autonomic nervous system: (1) vagal *visceral* unmyelinated afferents that decrease metabolism in response to environmental threats and that contribute to somatic feelings associated with emotional distress, including behavioral immobilization seen in certain animals feigning

DOI: 10.26386/obrela.v4i1.146

death, 2) the *sympathetic* nervous system, which increases heart rate and motor activity for the "fight or flight" response and (3) the *parasympathetic* nervous system involving the myelinated vagus nerve regulating cardiac activity with discriminating responsiveness to social approach or avoidance by downregulating cardiovagal tone. The myelinated vagus nerve is also linked to adaptive social behavior by relay mechanisms to the cranial nerves that regulate facial expression, vocalizations, and listening. These three parts of the autonomic nervous system are without a doubt the critical components of interpersonal engagement.

Emotional Intentionality

For *affective intentionality*, phenomenologists proposed that it is an *embodied* and *enactive* process that connect us to a shared world and guide our dealings with it **[48]**. For Heidegger, *moods* set up our encounter with the world by constituting our sense of belonging to it. They reveal the world as a space of practical purposes, values, goals, and activities —a space of meaning— and in this sense they are primordial phenomena presupposed by the intelligibility of our thoughts, experiences, and actions **[49]**. Ratcliffe **[50]** pointed on the disagreement concerning the nature of *emotional intentionality* and he presented ways that could distinguish emotional intentionality from other forms of intentionality. *Intentionality* seems to have essential connections with both consciousness and evolutionary selected functions, comprising the endogenous initiation, construction, and direction of behavior into the world. Affective intentionality is an *embodied* and *enactive* processing, while a *what-matters* model, according to Turner **[51]**, would be useful, employing a combination of the principles of intentionality and causality. Having in mind the rich philosophical and neuroscientific research, we can suggest that the field of *emotional intentionality* is a prominent neuroscience area, helping to better understanding of consciousness, emotions, emotion-cognition interplay, emotion (dys)regulation, human behavior, and even psychopathology **[52]**.

In neuroanatomical level, Frith's **[16],** proposal of a *cybernetic model* put emphasis on *intention* and on the *monitoring system.* Frith supported that psychosis can be seen as *a disorder of meta-representation,* which plays an important role in awareness processes. This model involves (a) the awareness of one's goals, (b) the awareness of one's intentions, and (c) the awareness of the intentions of others. Frith hypothesized that (a) the lack of awareness of one's goals leads to disorders of willed action characterized by negative symptoms, such as apathy, (b) the lack of awareness of intentions leads to self-monitoring disorders and anomalies in the experience of action, such as motor movement deficits, while limitations in social interaction leads to delusions of persecution and reference, and (c) the hallucinations are the result of a person's failure to recognize the self-generated nature of some actions or of inner speech, attributing it to an external source. Most positive symptoms can, according to Frith, be explained as a deficit in the capacity to distinguish between changes resulting from actions of the individual himself and those resulting from external events. Although these views emphasize *output mechanisms,* most neurobehavioral theories on delusions and hallucinations attribute them to deficits in perceptual *input mechanisms.* Input mechanisms are mostly associated with posterior brain areas, such as the parietal lobe, while output mechanisms are mostly associated with frontal areas, including the frontal cortex. Finally, Frith **[16]** suggested that brain areas involved in the disorders of willed action, such as the *dorsolateral prefrontal cortex*, the *supplementary motor area* and the *anterior cingulate gyrus* are responsible for the positive symptoms in schizophrenia, while the monitoring system seems to be primarily linked with the *hippocampal* system.

Millikan's **[53]** theory explains i*ntentionality* in terms that are broadly 'biological' or *teleological,* using the explanatory resources of natural selection: what thoughts and sentences and desires are 'about' is ultimately elucidated by reference to what has been selected and what it has been selected for, i.e., what advantage it conferred on ancestors who possessed it. Millikan's famous example of the *bee dance* illustrates that the internal device that leads the bee to perform its dance has the *proper function* to create a relational structure between the pattern of the dance and the nectar location. As a result, the internal interpretative device

DOI: 10.26386/obrela.v4i1.146

of the bees observing the dance fulfills its own relational proper function ["proper functions," meaning by "proper" a thing's "own" functions - Latin proprius, as in "property"]. In *teleosemantics*, the representational capacities of an organism are explained by reference to the teleofunction of a certain type of representational device inherited from its ancestors. In Millikan's model these *teleological functions* are *proper functions* of representational devices, and the mental representations they generate are the result of intentional signs respectively produced and consumed according to the proper functions of such devices, tuned to one another through an evolutionary process of mutual adjustment **[54].**

In a parallel way and extending his *intentionality theory*, Searle **[55]** suggested that *collective intentionality* is a biologically primitive phenomenon that humans share with other social animals. He argues that not *collective intentionality* itself, but the underlying capacity for collective intentionality is biologically innate. "The selectional advantage of co-operative behavior is obvious. Inclusive fitness is increased by cooperating with conspecifics.". Thus, he seems to hold that underlying *collective intentionality* there is a capacity that is innate, rather than culturally acquired, and that has been selected in processes of biological evolution. Without *collective intentionality* there could not have been *social reality* and without a *pre-intentional sense of community* there could not have been *collective intentionality*. Taken together this implies that *social reality* would not have been possible without a *pre-intentional sense of community* **[56].**

Emotions, intentionality, and emotional intentionality: hidden aspects in the predictive processing framework

There is probably no neural correlate of consciousness, since there is probably no area of the brain that is specifically dedicated to consciousness as opposed to vision, memory, learning, and so on **[57]**. As already mentioned, the anterior insular cortex plays a major role in such a process by encoding 'a meta-representation of the primary interoceptive activity' **[22]**. There is a widespread tendency to conceptualize the influence of predictions exclusively in terms of 'top-down' processes, although this excludes from consideration the predictive information embedded in the 'bottom-up' stream of information processing **[58]**. The incoming sensory evidence, in the form of prediction error, helps to shape the distributions of predictions that best fit the sensory array, thereby minimizing prediction error, which results in a *categorization* of the incoming sensory information in terms of past experiences **[35].**

Predictive processing is a theoretical framework that posits that the brain's overall function is to minimize long-term average prediction error. The objective of predictive processing is for the organism to maintain itself in its expected homeostatic states **[59]**. According to Seth **[60]** **"**predictive processing involve predictive modelling of internal physiological states (*interoceptive inference*), and integration with "enactive" and "embodied" approaches to cognitive science (*predictive perception of sensorimotor contingencies*)…This way of thinking leads to a new view of emotion as active interoceptive inference". *Predictive coding* is a concrete 'message passing' process where representations in higher levels generate predictions of representations in lower levels and the top-down predictions are compared with representations at the lower level to form a prediction error. These predictions are sent cascading down the processing hierarchy, suppressing congruent incoming sensory signals, such that only the residual, unexplained components of sensory information remain to be fed forward to higher levels in the form of "prediction error". The mismatch signal is passed back up the hierarchy, to update higher level representations following the loop of: *top-down predictions > expected precisions > endogenous precision modulation > bottom-up precision-weighted prediction error > precision prediction errors* **[4].**

In their recent review Walsh et al **[4]** concluded: "Although many studies report the expectation-related neural modulations that are predicted by predictive processing across the full range of neurophysiological recording techniques, a significant contingent failed to replicate these effects… It is not clear whether the formation of sensory predictions is a unitary neural process or an array of independent, task-tailored

DOI: 10.26386/obrela.v4i1.146

mechanisms… Expectations can arise from arbitrary stimulus pairings, predictive cues, or higher order regularities… Expectations can be formed in relation to stimulus timing, stimulus location, or stimulus content". Optimising the learning rate, and in-so-doing minimizing prediction error over time, is a major challenge for the brain. Failing to accommodate new evidence leads to *underfitting*, a failure to update predictions. According to Hohwy **[61]**, the more certain we are that the priors are correct, the less we should be influenced by the prediction error, which means that the learning rate is low. Conversely, the better the precision on the prediction error, the higher the learning rate; that is, the more we trust the quality of the evidence the more we should learn from it. That means, as Deane **[38]** pointed: the lower the learning rate, the greater the influence of top-down modulation from priors, and the higher the learning rate, the greater the influence of the sensory evidence on the resulting posterior.

On the other hand, the embodied predictive mind is an emotional mind. Within the areas of philosophy of mind and emotion, *vehicles* of knowledge are generally understood to be some kind of *vehicle of representation,* although a vehicle of knowledge simply makes it possible for the content of the vehicle to be known **[62]**. Millikan **[53]** refers to such vehicles of representation as "infosign vehicles" arguing that although emotions can "tell" us about the world, what they tell us is not something that is known since emotions are not justified and since emotions are not conscious. de Sousa's **[63]** puts *emotional salience* at the heart of how emotions solve this problem, supporting that "emotions are species of determinate patterns of salience among objects of attention, lines of inquiry, and inferential strategies". Although *emotion rationality* is independent *of* the rationality of judgments, perceptions, and desires, emotions work in conjunction with judgments, perceptions, and functional desires **[62]**, while predictive processing renders emotion to include even the very subtlest of these, ever-present in cognition **[64].** In their recent paper, Ransom et al **[65]** concluded that "prediction error minimization is not sufficient to explain all mental phenomena" and that "affectively salient stimuli can capture our attention even when precision expectations are low".

A number of 'enactive' approaches have been suggested, which support that conscious perception is closely entwined with agency, and intention **[43]**, while a focussing on interoceptive inference can better explain conscious phenomenology, especially in relation to affect, emotion, and self **[59]**. Seth et al **[66]** have suggested that "depersonalization and derealisation, that are associated with alexithymia and a general loss of 'emotional colour', may arise from imprecise (as opposed to inaccurate) interoceptive predictions, as part of a model of conscious 'presence'"**.** Emotions are taken as forming an anticipatory structure of experiencing the world, a structure which makes intentional, mental and bodily, acts possible. They have been characterized as background feelings, or as possibility structures, or as styles of anticipation of experience **[50]**. They open up or foreclose certain ranges of possible experience, allowing one to be attuned to the world and to one's own self, in some unthematized manner **[67]**.

Among psychologists is common the misconception that intention is a mental state of goal-directedness or purposiveness. Lawyers distinguish between intent and motive, since intent is a forthcoming action, while motive is the reason. Existing intentionalist views of emotions suggest that while some times they appear not to be directed at anything, upon closer examination, they are in fact directed at special kinds of objects, like bodily states or unusual external objects, such as the world as a whole, indeterminate intentional objects, or frequently changing objects **[68]**. For Panksepp **[69]** emotion is the "stretching forth" of intentionality, which is seen in primitive animals preparing to attack in order to gain food, territory, or resources to reproduce. This primitive form of emotion is called "motivation" or "drive" by behaviorists. But, how do intentional behaviors, all of which are emotive, whether or not they are conscious, emerge through the self-organization of neural activity in even the most primitive brains? The term *intentionality* is often used in association with the multivalent term of consciousness. For pragmatists most intentional actions and perceptions are unconscious or pre-conscious. For Heidegger the subject is structured intentionally within itself. Intentionality, also, is neither objective nor subjective in the usual sense, although it is certainly both **[70]**.

DOI: 10.26386/obrela.v4i1.146

Concluding, moods seem to be a pre-intentional state, constituting the background in the context of which intentionally directed emotions target their objects. In this opinion article I pose the question whether emotions, intentionality, as well as emotional intentionality, can bridge the gaps in the predictive processing framework. Most of above mentioned concepts, theories and findings can add useful information in understanding the priorities of this framework. I hope to see in a refined predictive processing framework a bridging of the gap that will include the intentional feature of emotions, a dynamic aspect that currently exist in isolation.

References

1. Dennett DC, Kinsbourne M. Time and the observer: The where and when of consciousness in the brain. *Behavioral and Brain Sciences* 1992*, 15:*183-247. DOI: 10.1017/S0140525X00068229
2. Shallice T. (1988). *From neuropsychology to mental structure.* Cambridge: Cambridge University Press.
3. Kosslyn SM, Koenig O. (1992). *Wet mind: The new cognitive neuroscience.* New York: Free Press.
4. Walsh K et al. Evaluating the neurophysiological evidence for predictive processing as a model of perception. Ann N Y Acad Sci. 2020 Mar; 1464(1): 242–268. doi: 10.1111/nyas.14321
5. Mesulam MM. (2000). *Principles of Behavioral and cognitive neurology*. Oxford University Press, NY.
6. Llinas RR. (2001). *I of the Vortex: f*rom Neurons to Self. Cambridge, MA: MIT Press.
7. Llinas RR, Ribary U. Consciousness and the brain: The thalamo-cortical dialogue in health and disease. *Annals of the New York Academy of Sciences* 2001*, 929:*166-175. doi.org/10.1111/j.1749-6632.2001.tb05715.x
8. Seth, A. K. From Unconscious Inference to the Beholder's Share: Predictive Perception and Human Experience. *European Review* 2019, 27:(3) 378- 410. DOI: https://doi.org/10.1017/S1062798719000061
9. Lord LD, Stevner AB, Deco G, Kringelbach L. Understanding principles of integration and segregation using whole-brain computational connectomics: implications for neuropsychiatric disorders. *Philos Trans A Math Phys Eng Sci*. 2017, 28;375(2096):20160283. doi: 10.1098/rsta.2016.0283.
10. Stromatias E. State-of-the-art deep learning has a carbon emission problem. Can neuromorphic engineering help? *Dialogues in Clinical Neuroscience & Mental Health* 2020, 3(3),143-149. DOI: 10.26386/obrela.v3i3.166
11. Goldberg, E., & Barr, W. B. (1991). Three possible mechanisms of unawareness of de!cit. In G. P. Prigatano & D. L. Schacter (Eds.), Awareness of de!cit after brain injury: Clinical and theoretical issues (pp. 152-175). New York: Oxford University Press.
12. McGlynn, S. M., & Schacter, D. L. (1989). Unawareness of de!cits in neuropsychological syndromes. Journal of Clinical Experimental Neuropsychology, 11, 143-205
13. Giotakos O. Poor insight in psychosis and meta-representation models. *Dialogues in Clinical Neuroscience & Mental Health* 2018, 1:12-24. DOI: https://doi.org / 10. 26386/obrela.v1i1.5
14. Sackeim, H. A., & Gur, R. C. (1978). Self-deception, self-confrontation, and self-consciousness. In G. E. Schwartz, & D. Shapiro (Eds.), Consciousness and self-regulation (pp. 139-197). New York: Plenum Press.
15. Petrides, M. (2013). The mid-dorsolateral prefronto-parietal network and the epoptic process. In D.T. Stuss & R.T. Knight (eds), Principles of Frontal Lobe Function, Chapter 7. New York: Oxford University Press, 79-89.
16. Frith, C. D., Friston, K., Liddle, P. F., & Frackow iak, R. S. J. Willed action and the prefrontal cortex in man: A study with PET. *Proceedings of the Royal Society of London* 1991, 244: 241-246.
17. Hyde, T. M., Ziegler, J. C., & Weinberger, D. R. Psychiatric disturbances in metachromatic leukodystrophy: Insight into the neurobiology of psychosis. *Archives of Neurology* 1992, 49: 401-406
18. Giotakos O (a). Is psychosis, at least in part, an immune-related dysmyelination disease? *Dialogues in Clinical Neuroscience & Mental Health* 2019, 2(2):16-129. DOI: 10.26386/obrela.v2i2.118
19. Giotakos O. (b). Is psychosis a dysmyelination-related information-processing disorder? *Psychiatriki* 2019, 30:245–255. doi: 10.22365/jpsych.2019.303.245
20. Friston K, Moran R, Seth AK. Analysing connectivity with Granger causality and dynamic causal modelling. *Curr Opin Neurobiol.* 2013 23(2):172–178. doi: 10.1016/j.conb.2012.11.010

DOI: 10.26386/obrela.v4i1.146

21. Friston K. Prediction, perception and agency. *International Journal of Psychophysiology* 2012, 83: 248-252. https://doi.org/10.1016/j.ijpsycho.2011.11.014

22. Craig AD. How do you feel—now? The anterior insula and human awareness. *Nat Rev Neurosci* 2009, 10:59-70. doi.org/10.1038/nrn2555

23. Kirov R Editorial: Brain Oscillations and Predictive Coding in the Context of Different Conscious States and Sleep-Wake Cycle: Implications for Decision Making and Psychopathology. *Front. Psychol*. 2016, 7:1768**.** doi: 10.3389/fpsyg.2016.01768

24. Murray GK, Corlett PR, Clark L, et al. Substantia nigra/ventral tegmental reward prediction error disruption in psychosis. *Mol Psychiatry* 2008, 13:239, 267-76. doi: 10.1038/sj.mp.4002058

25. Xue G, Lu Z, Levin IP, et al. The impact of prior risk experiences on subsequent risky decision-making: the role of e insula. *Neuroimage* 2010, 50:709-16. DOI: 10.1016/j.neuroimage.2009.12.097

26. Kurth F, Zilles K, Fox PT, Laird AR, Eickhoff SB. A link between the systems: functional differentiation and integration within the human insula revealed by meta-analysis. *Brain Struct Funct* 2010, 214:519-34. DOI: 10.1007/s00429-010-0255-z

27. Bressler SL, Menon V. Large-scale brain networks in cognition: emerging methods and principles. *Trends Cogn Sci* 2010, 14: 277-90. doi: 10.1016/j.tics.2010.04.004

28. Todd J, Michie PT, Schall U, Ward PB, Catts SV. Mismatch negativity (MMN) reduction in schizophrenia—Impaired prediction-error generation, estimation or salience? *International Journal of Psychophysiology* 2012, 83:222-231. https://doi.org/10.1016/j.ijpsycho.2011.10.003

29. Bodatsch M, Ruhrmann S, Wagner M, Müller R, Schultze-Lutter F, Frommann I, et al. "Prediction of psychosis by mismatch negativity". *Biological Psychiatry* 2011, 69(10): 959–66. *doi*: 10.1016/j.biopsych.2010.09.057

30. Corlett PR, Taylor J, Wang X, et al. Toward a neurobiology of delusions. *Prog Neurobiol*. 2010; 92:345-69. doi: 10.1016/j.pneurobio.2010.06.007

31. Kapur S. Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *Am J Psychiatry* 2003, 160(1): 13-23. doi.org/10.1176/appi.ajp.160.1.13

32. Oatley K, Johnson-Laird PN. Towards a cognitive theory of emotions. *Cognition and Emotion* 1987, *1:*29-50. DOI: 10.1080/02699938708408362

33. Giotakos O. (a). (2020) Emotional Trauma in Emotional Brain,(e-Book), iWrite Publications.

34. Giotakos O. (b) Neurobiology of emotional trauma. *Psychiatriki* 2020, 31:162– 171. DOI: 10.22365/jpsych.2020.312.162

35. Barrett LF. The theory of constructed emotion: an active inference account of interoception and categorization. *Social Cognitive and Affective Neuroscience*, 2017, 1–23. doi.org/10.1093/scan/nsw154

36. Cosmides L, Tooby J. (2000). Evolutionary psychology and the emotions. In M. Lewis, & J. M. Haviland-Jones (Eds.), *Handbook of Emotions* (2nd ed.). New York: Guilford Press. pp. 91-115.

37. Schachter, S., & Singer, J. Cognitive, social, and physiological determinants of emotional state. *Psychological Review 1962, 69*(5):379–399. https://doi.org/10.1037/h0046234

38. Deane, G. Dissolving the self: Active inference, psychedelics, and ego-dissolution. *Philosophy and the Mind Sciences* 2020, *1*(I), 2. https://doi.org/10.33735/phimisci.2020.I.39

39. Bower GH. Mood and memory. *American Psychologist 1981,* 36:129-148. doi.org/10.1037/0003-066X.36.2.129

40. Niederthal PM, Krauth-Gruber S, Ric F. (2006). *Psychology of emotion, Interpersonal, experiential and cognitive approaches*. Psychology Press.

41. Gross, J. Emotion regulation: Past, present, future. *Cognition and Emotion* 1999*, 13:*551-573. doi.org/10.1080/026999399379186

42. Damasio AR. (2012). *Self comes to mind: Constructing the conscious mind*. Vintage London.

43. Seth AK & Critchley HD. Extending predictive processing to the body: Emotion as interoceptive inference. *Behavioral and Brain Sciences* 2013, 36:3 doi:10.1017/S0140525X12002270

44. Allman J, Brothers L. Neuropsychology. Faces, fear and the amygdala. *Nature.*1994, 15: 613-4. doi: 10.1038/372613a0

45. Price JL, Carmichael ST, Drevets WC. Networks related to the orbital and medial prefrontal cortex: A substrate for emotional behavior? *Progress in Brain Research.* 1994, 107:523- 536. doi.org/10.1016/S0079-6123(08)61885-3

46. Menon V, Uddin LQ. Saliency, switching, attention and control: a network model of insula function. *Brain Struct Funct* 2010, 214:655-67. doi: 10.1007/s00429-010-0262-0

47. Porges SW. The polyvagal theory: phylogenetic contributions to social behavior. *Physiol Behav* 2003, 79:503-13. doi: 10.1016/s0031-9384(03)00156-2

DOI: 10.26386/obrela.v4i1.146

48. Colombetti, G. (2014). The Feeling Body: Affective Science Meets the Enactive Mind. Cambridge, MA: MIT Press

49. Ratcliffe, M. (2008). International perspectives in philosophy and psychiatry. Feelings of being: Phenomenology, psychiatry and the sense of reality. Oxford University Press. https://doi.org/10.1093/med/9780199206469.001.0001

50. Ratcliffe M. Emotional Intentionality. Royal Institute of Philosophy Supplement 2019, 85:251-269 DOI: 10.1017/S1358246118000784

51. Turner CK. A Principle of Intentionality. *Front. Psychol*. 2017, 8:137 https://doi.org/10.3389/fpsyg.2017.00137

52. Giotakos O. (c) Intentionality and Emotions. *Dialogues in Clinical Neuroscience & Mental Health* 2020, 3(3):133-142. DOI: 10.26386/obrela.v3i3.167

53. Millikan, Ruth G. 2017. *Beyond Concepts: Unicepts, Language, and Natural Information*. Oxford: Oxford University Press.

54. Faye Pierre. (2011) In Favor of Teleosemantics: A Millikanian Treatment of the Intentional Content of Mental Representation. A dissertation submitted to the Graduate Faculty in Philosophy in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York.

55. Searle, J.R, (1995), The Construction of Social Reality. NY Free Press

56. Vromen J. Collective Intentionality, Evolutionary Biology and Social Reality, *Philosophical Explorations: An International Journal for the Philosophy of Mind and Action* 2003, 6:3, 251-265, DOI: 10.1080/10002003098538753

57. Güzeldere, G. 1999. There is no neural correlate of consciousness. Paper presented at “Toward a Science of Consciousness: Fundamental Approaches”, Tokyo, May 25-28, 1999.

58. Teufel and Fletcher. Forms of prediction in the nervous system. *Nature reviews Neuroscience* 2020, 21(4):1-12. DOI: 10.1038/s41583-020-0275-5

59. Hohwy, J & Seth A. Predictive processing as a systematic basis for identifying the neural correlates of consciousness, June 2020. Preprint DOI 10.31234/osf.io/nd82g

60. Seth, A. K. (2015). The Cybernetic Bayesian Brain - From Interoceptive Inference to Sensorimotor Contingencies. In T. Metzinger & J. M. Windt (Eds). *Open MIND:* 35(T). Frankfurt am Main: MIND Group. doi: 10.15502/9783958570108

61. Hohwy, J. New directions in predictive processing. *Mind & Language 2020,* 35(2): 209-223. doi: 10.1111/mila.12281

62. Candiotto L. (2019). The Value of Emotions for Knowledge. Palgrave Macmillan.

63. de Sousa R. (1987). *The Rationality of Emotion.* Cambridge: MIT Press. de Sousa. 2011. *Emotional Truth*. New York: Oxford University Press.

64. Wilkinson S, Deane G, Nave K and Clark A. (2019). *Getting Warmer: Predictive Processing and the Nature of Emotion*. In Laura Candiotto. The Value of Emotions for Knowledge. Palgrave Macmillan

65. Ransom M, Fazelpour S, Markovic J, Kryklywy J, Thompson ET, Todd RM. Affect-biased attention and predictive processing. Cognition 203 (2020) 104370. doi.org/10.1016/j.cognition.2020.10437

66. Seth, A.K. et al. (2011) An interoceptive predictive coding model of conscious presence. Front. Psychol. 2, 395

67. Hatzimoysis A. (2019). *In Search for the Rationality of Moods*. In Laura Candiotto. The Value of Emotions for Knowledge. Palgrave Macmillan.

68. Mendelovici A. Intentionalism about moods. Thought: A Journal of Philosophy 2013. Nov 18. DOI:10.1002/tht3.81

69. Panksepp, J. (1998). *Series in affective science. Affective neuroscience: The foundations of human and animal emotions.* Oxford University Press.

70. Freeman WJ, (2000). *Emotion is Essential to All Intentional Behaviors*. in: Emotion, Development, and Self-Organization Dynamic Systems Approaches to Emotional Development, Marc D. Lewis & Isabel Granic (Eds). Cambridge U.K.: Cambridge University Press, p 209-235.