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Motive control of unconscious inference: The limbic base of adaptive Bayes

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ABSTRACT

Current computational models of neocortical processing, described as predictive coding theory, are providing new ways of understanding Helmholtz's classical insight that perception cannot proceed in a data-driven fashion, but instead requires unconscious inference based on prior experience. Predictive coding is a Bayesian process, in which the operations at each lower level of the cortical hierarchy are predicted by prior projections of expectancies from a higher level, and are then updated by error-correction with lower level evidence. To generalize the predictive coding model to the human neocortex as a whole requires aligning the Bayesian negotiation of prior expectancies with sensory and motor evidence not only within the connectional architecture of the neocortex (primary sensory/motor, unimodal association areas, and heteromodal association areas) but also with the limbic cortex that forms the base for the adaptive control of the heteromodal areas and thereby the cerebral hemisphere as a whole. By reviewing the current evidence on the anatomy of the human corticolimbic connectivity (now formalized as the Structural Model) we address the problem of how limbic cortex resonates to the homeostatic, personal significance of events to provide Bayesian priors to organize the operations of predictive coding across the multiple levels of the neocortex. By reviewing both classical evidence and current models of control exerted between limbic and neocortical networks, we suggest a neuropsychological theory of human cognition, the *adaptive Bayes process model*, in which prior expectancies are not simply rationalized propositions, but rather affectively-charged expectancies that bias the interpretation of sensory data and action affordances to support *allostasis*, the motive control of expectancies for future events.

1. Introduction and overview

It has long been a challenge to understand how cognitive function can arise from neural tissue. This challenge remains daunting, even though an emerging scientific synthesis promises new and profound insight. In addition to the increasing advances in methods for imaging the activity of the brain, we are gaining an increasing understanding of the ordered network connectivity of the neocortex (García-Cabezas et al., 2019; Ugurbil et al., 2013), and this order apparently reflects evolutionary trends that gave rise to the cortex (Butler and Molnar, 2002; Luzzati, 2015; Sanides, 1970). In parallel, the development of artificial intelligence is rapidly advancing our knowledge of how neural network architectures might work, because we can simulate different connectivities and evaluate their properties for learning and for representing information (Friston, 2008; Sanda et al., 2019). This convergence of neuroscience and computational modeling is allowing a fresh look at psychological theory, recognizing the properties of mind that may be directly implied by the functional and computational properties of the neural architecture.

In this paper, we review several issues in attempting to understand the motive control of unconscious inference, drawing from both neurophysiological and computational approaches. Unconscious inference is the process that Helmholtz (1878/1971) inferred must occur in visual perception, because the data impinging on the retina are not interpretable as a functional perception without prior experience with vision. A century and a half after Helmholtz shared his insight, we are realizing that the inference we achieve through experience with retinal images is indeed automatic and unconscious, and it indeed reflects the implicit skill of experience. In the modern theory of predictive coding (Bastos et al., 2012; Friston, 2008; Rao and Ballard, 1999), the implicit perceptual inference is formulated in a Bayesian model closely aligned with the neocortical architecture of specific areas of the primate visual system. More than a theory of vision, predictive coding is becoming a theory of the brain, describing how the processing of information can be achieved by the operation of learned expectancies that shape the interpretation of sensory data. The Bayesian analysis also explains how

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prior expectancies are updated and modified when sensation is incompatible with familiar predictions. Extended to motor systems, predictive coding theory has allowed insight into how expectancies — formed in the cortex for the kinesthetic and proprioceptive results of actions — can be propagated to both brainstem and spinal pattern centers to regulate action (Adams et al., 2013).

If it could be extended to the neocortical architecture generally, predictive coding theory promises a formulation for how the linked areas of the cortex (primary sensory/motor, unimodal association, heteromodal association, and limbic areas) negotiate the sensory and motor contact with the world (at the sensory-motor extent of the cortical pathways) in relation to the homeostatic requirements of the organism (managed by the limbic base of the neocortical pathways). The question becomes the motive control of unconscious inference in this more general sense: how the process of cognition is regulated by the organism's personal homeostatic requirements, often in implicit ways that are not reflected in consciousness.

A leading proponent of predictive coding, Friston (2010) has, in fact, proposed that the Bayesian analysis of cognition can be extended from the neural systems level to a general, organismic process of cognition, a process described as *active inference*. The Bayesian individual attempts to predict the events in the world that are consistent with existing personal knowledge. Successful expectancies (predictions) are the main tasks for living, such that surprise (inaccurate prediction) is an adaptive threat. Friston points out that the requirements for successful prediction (and minimizing the error of the Bayesian process) are framed by the need for maintaining organismic homeostasis, such as imposed by hunger, or thirst, or pain. In order to be predictive, motive control is not just homeostatic, generated by adaptive needs, but *allostatic*, anticipating the multiple system adjustments required for coping with the future.

In understanding the neocortex, the obvious starting hypothesis is that the same principles — of expectancies arising in higher areas constraining the interpretation of evidence from lower areas — can be applied across the cortical hierarchy. However, modern anatomy shows that the neocortex is not uniform, but varies in its regional architecture in ways that are likely to affect the inter-laminar dynamics that define predictive coding. Although it is often assumed that the mammalian cortex comprises a uniform architecture of 6 layers, the anatomy of the cortical lamina is found to vary considerably from one area to the next, in a way that may seem unintuitive but is regular and consistent (García-Cabezas et al., 2019). Perhaps unexpectedly, the most differentiated 6-layer cytoarchitecture is found in a cognitively *lower* area (primary visual cortex), with increasingly *less* complex laminar differentiation at each *higher* area of the cortex (from unimodal association to heteromodal association to limbic areas).

Now, few neuroscientists and cognitive psychologists would consider limbic cortex as the *higher* area, given that it is the cortical source of personal emotions and motivations. An assumption of academic models of cognition is that higher mental function must be more purely objective. Yet, if the logic of predictive coding is to be generalized to the pathways of the neocortex as a whole, then the limbic cortex must apply expectancies to its adjacent heteromodal cortex in the same way that visual association applies expectancies to the incoming evidence of primary visual cortex. Limbic cortex is thus is in the position of the higher controller for the subordinated error-correction in the adjacent (in this case heteromodal association) linked network.

The task for a biological theory of predictive coding is then to understand the Bayesian neocortical logic, of prior expectancies shaping the interpretation of evidence, in a brain that is organized for allostatic self-regulation. The evidence is now clear that cognition is not limited to immediate conscious apprehension, but requires ongoing consolidation of its memory structures through unconscious processing, in both quiet reflective periods (Carr et al., 2011) and in the sequential organization of the stages of sleep (Diekelmann and Born, 2010). This is perhaps the most interesting challenge for predictive coding theory, to account for unconscious inference in light of the remarkable evidence that it unfolds over time through the ongoing yet largely unconscious dynamics of memory consolidation.

The theoretical perspective we bring to this account is the proposal that cognition should be understood as neural development itself, an ongoing ontogenesis of the neural architecture that continually adjusts neocortical connections to allow allostatic adaptation to the dynamic requirements of psychological development (Tucker and Luu, 2012). In the present paper, we attempt to formulate the motive control that the limbic system applies to the processing throughout the neocortical hierarchy. This formulation applies the terms of predictive coding theory, while drawing on both classical principles of cortical and limbic function from human neuropsychology and neurophysiology and the modern integration of anatomical evidence in the Structural Model. We propose a formulation of predictive coding described as the *adaptive Bayes process model.* The expectancies serving as priors on cognitive computations in cortical areas are proposed to be intrinsically charged with motive significance, thereby priming affect, cognition, and perception as a function of the current mood state, conditioned as it is by both homeostatic needs and recent success in prediction.

Similarly, the adaptive Bayes model proposes that ongoing, unconscious memory consolidation continues the process of negotiating new evidence with existing representations. This process is also charged by the emotional and motivational significance of expectancies that reflect the integral limbic control of memory. In both affective priming (cognition) and adaptive binding (memory consolidation), the inherent motive bias of Bayesian priors causes the negotiation between expectancy and evidence to proceed in line with the allostatic bias appropriate to the current state of the organism. In a good mood, things are looking up (and adaptive Bayes is confidently primed). In an anxious mood, threats loom large (uncertainty is palpable, and errors are salient).

Our theoretical proposition is congruent with recent extensions of predictive coding that emphasize the *affective charge* associated with prediction success and error, with corresponding adjustments in weighting the precision of sensory (and sensorimotor) evidence (Hesp et al., 2021). The unique perspective of our approach comes from recognizing the primacy of *motive control*: the adaptive influences from limbic networks that apply prior, feedforward control to cognitive expectancies throughout the neocortical hierarchy.

Given our goal of understanding the control mechanisms within the corticolimbic hierarchy, a major limitation in this paper will be our cursory treatment of subcortical regulatory systems. The subcortical control systems are essential to any description of how the neocortex self-regulates, but we can treat those systems only vaguely in this space, recognizing that the a full account of neocortical negotiation with limbic controls requires a more complete integration of hypothalamic, thalamic, striatal, midbrain, and brainstem regulatory systems.

2. The integral allostasis of neural development

In generalizing the predictive coding account, we begin with what could be described as a Bayesian neurodevelopmental model: human neural development first actualizes the genetic plan for forming the brain in utero, progressively specifying the vertebrate neural architecture in the rough phyletic sequence of the genetic plan for the species. For the child, growth then builds on this plan as the starting prior for synaptic differentiation and integration to organize learning and memory adaptively throughout the ontogenetic course of psychological development (Tucker and Luu, 2012). This evolutionary-developmental approach to human neural development can be seen as requiring Bayesian reasoning. This is because in each individual's development — starting with embryogenesis — the causality of development is embedded within the venerable priors of the species history. Each biological process can only be enacted within the causal momentum of evolutionary-developmental complexity. Ontogeny recapitulates phylogeny in embryogenesis, in a coherent outline, because the uniquely human genetic plan is progressively organized through the causal

momentum of vertebrate evolution as reflected in the unique ontogenetic adaptations of the human genetic plan (Gould, 1977). Ontogeny then continues the organizing process in individual neuropsychological development. Within neurodevelopmental theory (Tucker and Luu, 2012), each cognitive process is an extension of neural development: each thought can only emerge on the Bayesian foundation of the neural priors of personal ontogenetic history.[1]

The process of neural development must be continually guided by homeostasis, maintaining the organism's biological integrity from the early embryonic stages. Yet development is increasingly allostatic, anticipating the requirements not just of immediate needs but the preparation for each subsequent stage of ontogenesis (Luu and Tucker, 2004; Tucker et al., 2000). For mammals generally, and for humans particularly, allostatic development prepares for the ongoing requirements of socialization (Derryberry and Tucker, 2006; Luu and Tucker, 2004; Tucker et al., 2005). Classical accounts of plasticity in neural development have emphasized that neural plasticity is *experience-dependent*, in that synapses are only retained if they are functional. Yet other aspects of neural development are *experience-expectant*, in that they are regulated by the ontogenetic plan in anticipation of the next critical developmental stage of environmental experience (Greenough, 1975, 1984; Greenough and Black, 1992).

Because neural development with these properties of plasticity is inherently cumulative, with the mechanisms of each experience dependent on prior growth, a Bayesian approach to evolutionary-developmental theory is well-suited to an analysis that recognizes that cognitive function and neural structure are not just isomorphic but identical. The mechanisms of activity-dependent specification of synaptic connectivity first articulate the fine connectivity of neural networks in response to the endogenous rhythmic activity of the embryonic brain (Marin-Padilla, 1998b). The key scientific insight is then that the same mechanisms of activity-dependent synaptic plasticity continue to organize learning and memory throughout life (Von de Malsburg and Singer, 1988). From this perspective, the ontogenetic differentiation of neural connectivity must continue as the substrate for the daily and nightly developmental process of cognition, including not only conscious experience, but the ongoing mechanisms of unconscious memory consolidation. Each act of cognition is then an irreversible continuation of synaptic differentiation (Tucker and Luu, 2012).

We can begin our review by recognizing that the individual's behavior is shaped by homeostatic needs and urges operating to influence the cognitive apparatus. Yet exactly how allostasis emerges to anticipate future needs becomes difficult to specify in the traditional approach to motivation, which assumes that needs, and drives associated with needs, explain motive control. Instead, neurodevelopmental theory shows that the organization of increasing cognitive complexity is the integral process of growing the brain even as it is also the process of maintaining organismic coherence (minimizing free energy). The control of neural activity is not just need driven, but must be allostatic in a general sense, preparing for the likelihood of threats and opportunities even though these are fully uncertain. The fact that neural development is profoundly cumulative requires a Bayesian analysis, where the understanding of new information is dependent on the relevance of that information for the existing cognitive and neural representations encoded in the synaptic anatomy.

Our goal in this paper is to bring this neurodevelopmental approach to address the question of reentrant motive control across the cortical hierarchy, from limbic cortex to heteromodal association cortex, to unimodal association cortex to primary sensory and motor areas and back again. The central question is whether the relations between each cortical area and its adjacent networks in the cortical hierarchy can be described in the terms of predictive coding theory: forming expectancies and negotiating these with the evidence gained from interacting with the environment.

2.1. How expectancies shape unconscious inference

The modern computational theory of artificial neural networks is showing how Helmholtz's process of unconscious inference might be achieved by multi-leveled distributed neural architectures (Hinton, 2000; Hinton et al., 1995; Lee and Mumford, 2003; Mumford, 1992). Whereas the engineering of artificial intelligence has been successful by the unabashed stacking of many levels, important approaches to modeling brain activity have been successful by designing networks that are more similar to the connectivity between levels of the neocortex, such as between primary visual areas and unimodal visual association areas (Rao and Ballard, 1999). Rao and Ballard designed a computational model of the causes of the visual input (simulating association cortex) that predicted what should occur at primary visual cortex. The convergence of the predictive model weights with those influenced by the actual visual input pattern led to a process of error-correction, particularly at cortical layers 2 and 3 of their model. Remarkably, the input sensitivity of the primary visual neurons — after this predictive coding simulation was trained — showed a striking similarity to the receptive fields of actual neurons, including not only line/contour orientation sensitivity but distinct end-stop features.

The Rao and Ballard model had an important influence in stimulating interest in predictive coding theory (Friston, 2018). This predictive model was designed to compute just what Helmholtz concluded had to be done in order to interpret retinal inputs through unconscious inference. The model generated, through top-down control, the pattern of receptive fields in the input level neurons that was similar to the actual receptive fields of visual cortex neurons that has long been enigmatic. Receptive fields are not an intuitive mapping of visual space. The Rao and Ballard model showed that their unintuitive topography could be created by the top-down predictions (and inhibitions) from a higher cortical area (Friston, 2018).

[1] The increasing embryological evidence on the genetic blueprint (bauplan) for the mammalian brain is highly relevant to the evolutionary-developmental theory pursued here. Although reviewing of this evidence is beyond the scope of the present paper, it is important to point out that the organization of the telencephalon emerging from this genetic embryology is consistent with the Structural Model of the neocortex, even as it is clarifying the organization of the subcortical foundations of the limbic system at its base. Puelles and associates have recently analyzed the patterns of gene expression in embryogenesis of the mouse brain to clarify the prosomeric model of the differentiations of developing tissue that become elaborated into the neocortex (Puelles et al., 2013). The pallium or progenitor of cortex is anchored by the septal histogenetic domain at the dorsomedial extent, and by the amygdalar domain at the ventrolateral extent. In development, the medial pallium originates the hippocampal formation; the dorsal pallium originates most of the neocortex; the lateral pallium originates part of the mesocortex, specifically insular cortex; and the ventral pallium originates the primary olfactory cortex (Puelles et al., 2013). This emerging embryological evidence is largely consistent with the dual archicortical and paleocortical origins neocortex in the Sanides observations (Sanides, 1970) that have been incorporated within the Structural Model. Furthermore, this evidence provides additional clues to the subcortical control of the dual dorsal (archicortical) and ventral (paleocortical) divisions of the limbic system (Tucker and Luu, 2012). The nucleus accumbens, for example, is found to derive from the paraseptal domain (Puelles et al., 2013), providing a neural basis for the hedonic motive control for the dorsal division of the limbic system and neocortex (Tucker and Luu, 2012). Progress in modeling the mechanisms of embryogenesis has also led to an application of the free energy principle underlying predictive coding that has interesting implications for the neurodevelopmental theory of cognition we have adopted as the starting point for the present formulation. Following the genetic specification of the bauplan, the efficient organization of neural ensembles within the pallium can be modeled as oscillatory dynamics within entropic domains (Markov blankets) bounded by the free energy principle (Wright and Bourke, 2020). The proposal of this modeling is that the prenatal assembly of functional connectivity creates the organizational process that transitions directly to the learning and memory of child development, which is the core proposal of the neurodevelopmental theory of cognition (Tucker and Luu, 2012).

The utility of predictive coding theory in explaining cortical processing has been supported by analyses of the microcircuits of cortical columns that share considerable similarities with the neural network architectures that are able to generate, and error-correct, predictive coding. Bastos et al. (2012) reviewed both simulations and analyses of physiological activity to align the specific connectivity of cortical columns in the visual pathway with the operations of predictive coding that can be expressed in Bayesian equations. The basic connectivity of visual cortex is illustrated by the simplified cartoon in Fig. 1, showing the overall organization of visual areas in the top image, with primary visual cortex (shaded) at right, connected to unimodal visual association area (stippled), which is connected to the heteromodal association area (dashed), which is connected to limbic cortex (striped). The direction of processing traditionally described as "feedforward" is shown in the second row, with the six layers of the cortex indicated by the dots that denote the granular layer 4, with layers 1–3 positioned above and layers 5–6 below. The visual input from the thalamus to the granular layer 4 of primary visual cortex is indicated by the dark arrow at right. The major projection to the next (visual association) level arises in the supragranular layers 2/3 of primary cortex and projects primarily to layer 4 of the next level (dark arrow); this pattern continues across levels to the limbic cortex.

An asymmetric pattern of projections describes the reverse (limbic-to-primary cortex) or *limbifugal* direction (traditionally described as the "feedback" direction), with a major projection arising from infragranular layers (5/6) and projecting to supragranular layers (2/3). This limbifugal projection provides the prediction or expectancy in predictive coding theory, predicting the input pattern that will be decoded at the next lower cortical level. The essential processing at each cortical level is the adjustment of the expectancy based on the actual input data, such that the "feedforward" or 2/3–4 projections of the second row (the *limbipetal* or toward-limbic direction) communicates the errors of prediction. When aligned with the asymmetric connection architecture of the neocortex illustrated in Fig. 1, predictive coding theory thus implements a Bayesian model in which each higher level of cognitive/neural representation is a prior expectancy for what happens at the adjacent lower level, and the errors of that expectancy are computed in relation to the lower level (eventually sensory input) data.

A key theoretical advantage of this interpretive model over traditional artificial neural networks is that it provides an explanation of control as well as representation. The predictive coding model incorporates the inherent control or cybernetics of the Bayesian model,[2] with the expectancy from the higher area as a starting point, and the error-correction of the prior model propagating in the limbipetal direction to update expectancies in relation to the environmental evidence.

When these cybernetic features are considered, it becomes clear that the terms "feedforward" and "feedback" are used incorrectly in the current neuroscience paradigm. The incoming processing direction starting from visual input is not feedforward control, as was initially assumed, but is actually feedback: it is error-correction for the starting position, clearly reflecting the function of feedback control in classical cybernetics (Hendler, 1995; Wiener, 1961). Similarly, the actual feedforward control is applied in the outgoing or limbifugal direction, in the form of the Bayesian prior expectancy.

It is not just in the application to predictive coding theory that the conventional usage of the feedforward and feedback terms in neuroscience reflects widespread assumptions that may have been reasonable at first but are now clearly incorrect. The psychological literature on visual perception provides considerable evidence that perception is not a process of successive interpretation of veridical input data, as initially assumed in experimental psychology (uncharitably described as the *dogma of immaculate perception*). Rather, perception begins with the operation of prior experience that generates a model of the visual world as the starting point, as Helmholtz surmised (Helmholtz, 1878/1971; Koenigsberger, 1902). From his extensive experiments on human visual perception, Roger Shepard (Shepard, 1984) summarized the cognitive process of perception as beginning with a generation of the possible percept, which is then adjusted in relation to the actual retinal input, a process that could be described as *hallucination constrained by the sensory data* (Tucker, 2007). As in predictive coding, and as in Helmholtz's unconscious inference, the starting (*feedforward*) process for perception is provided by memory, and the correction of this starting expectancy by sensory evidence is a process of error-correction (*feedback*).

A similar confusion is created when control theory terms are used incorrectly in the predictive coding model of action. It has long been apparent in neurology that motor regulation requires that higher brain regions, such as the neocortex and striatum, organize the semi-autonomous motor reflex arcs and pattern generators of lower areas. These include not only the midbrain and the reticular networks of the brainstem, but also the intrinsic pattern generating circuits of the spinal cord (Denny-Brown, 1966; Sherrington, 1913). Both neurological observations and systematic animal studies showed that motor control cannot be a simple plan that is formed at higher levels and then conveyed for point-by-point to execution by the muscles (as assumed incorrectly by early computational robotics). Rather, as now described by predictive coding theory (Adams et al., 2013), the cortical regulation of action begins with *feedforward* control, initially at the limbic level but linked through the neocortical hierarchy to primary motor cortex (Shipp, 2005). The control is exerted not by a motor command, but a neocortical expectancy for the results of action that envelopes and organizes the multiple and hierarchic pattern generation circuits of the lower neuraxis, with continual error-correction and *feedback* modification on the basis of evidence on the current states and environmental contingencies of the lower level executions (Adams et al., 2013).

Although the misuse of the terms *feedforward* and *feedback* is entrenched in the neuroscience literature and will be difficult to change, control theory is so important in understanding neural circuits that the terms should be used correctly. Table 1 summarizes the anatomical directions of information flow in the cortical hierarchy and the differing interpretations of feedforward and feedback control in the adaptive Bayes process model versus the current paradigm of neuroscience (Table 2).

2.2. Connectivity and structure of the neocortical architecture

The predictive coding theory of perception and action is aligned with the general anatomical evidence on the laminar inter-connectivity of cortical areas, as outlined in Fig. 1. This evidence reveals a regular pattern of increasing differentiation of cytoarchitecture proceeding from limbic toward primary sensory and motor areas (Barbas, 2015; García-Cabezas et al., 2019). Although it has often been assumed that the neocortex comprises a uniform web of cortical columns which is repeated in each cortical area, the anatomical studies of primate neocortex starting in the 1970s (Barbas and Pandya, 1989; Galaburda and Pandya, 1983; Mesulam et al., 1977; Pandya and Seltzer, 1982) revealed the increasing differentiation of cortical architectural complexity (cytoarchitectonics), proceeding from the simpler networks

[2] Bayes's Theorem could be said to be so remarkably useful for problem-solving because it arranges the conditional probability of two distributions in a way that captures the temporal order of what is known about these distributions, thereby structuring the solution. Of course, in computing the p (A|B) there is no requirement that B has already occurred. The logic follows the order of what is known (believed), not any temporal order of the evidence. But the expression orders what is known first (the prior belief) in a way that organizes how this knowledge must change with the additional evidence captured in the conditional probabilities. This formulation thus aligns well with the developmental analysis of neural networks, in which accumulated experience provides the starting point for how ongoing sensory and motor evidence must be evaluated.

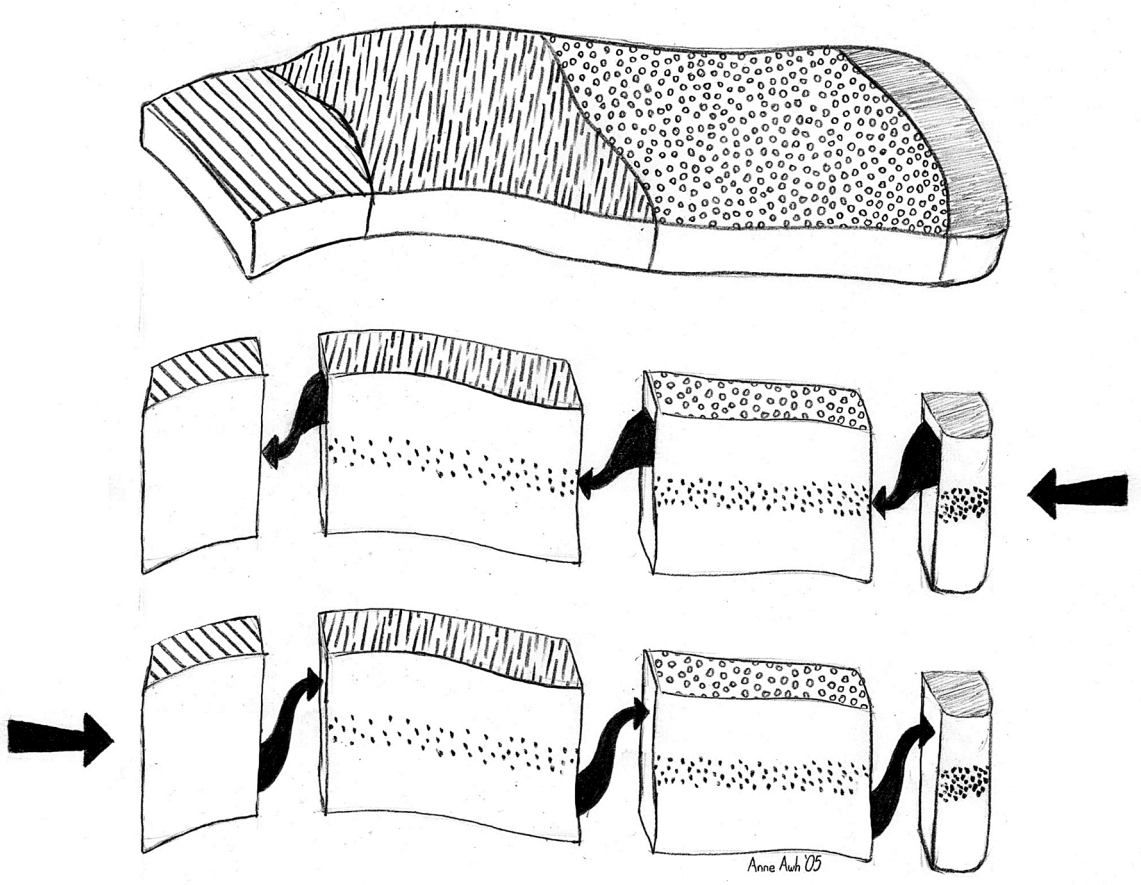

Fig. 1. Asymmetric connectivity of adjacent cortical areas within a sensory pathway, such as vision. From left to right the cortical areas are limbic, heteromodal association, unimodal association, and primary cortex. The top row illustrates the general cortical areas, and the bottom two rows show the pattern of connectivity across the cortical lamina of each area. The input (error-correction in predictive coding) such as from the thalamus in the second row (arrow at right) is to granular layer 4 (dots in the middle), and the primary projections are from supragranular layers to the layer 4 of the higher area. For completeness, the right-to-left or incoming direction is continued into the limbic cortex at left, even though this area has no granular layer. The reverse direction of connectivity (bottom row) begins with subcortical (particularly hypothalamic) input to the limbic area (arrow at left), and projections to the adjacent lower level are most dense for the infragranular to supragranular layers (from Tucker, 2007).

Table 1
Brief definitions of major concepts.

predictive coding	a Bayesian computational model in which higher cortical areas provide predictions for patterns of information to lower areas, and lower areas provide sensory evidence that may correct the errors of these predictions.
allostasis	preparatory homeostasis: adjusting multiple bodily systems in advance of coping efforts that require those systems.
homeostasis	physiological regulation to maintain adaptive set points required for organismic integrity.
adaptive Bayes	the present theoretical model that expectancies (priors) may be charged with motive significance, thereby biasing the balance of prediction with evidence (precision) throughout the neocortical hierarchy.
active inference	the Bayesian process of negotiating expectancies integral to organismic integrity (minimizing free energy) in relation to the evidence of reality accessed by perception and action.
affective inference	an extension of active inference in which the success of predictions modifies both the precision weighting of evidence and the affective state associated with success and error.
adaptive control	cybernetic influence on neural operations based in organismic homeostasis
limbifugal	direction of connections and processing *away* from limbic areas
limbipetal	direction of connections and processing *toward* limbic areas

of limbic areas toward the more complex local architecture of primary sensory and motor neocortex.

Barbas and associates (Barbas, 1995a, 2015; García-Cabezas et al., 2019) have described the differentiated laminar pattern of neocortex as *eulaminate*, the absence of layer 4 in limbic cortex as *agranular*, and the intermediate regions as *dysgranular*.[3] An obvious feature of the increasing architectural differentiation from limbic to neocortical areas, as illustrated in Fig. 1, is the greater density of the granular layer in primary sensory neocortex, with progressively less density in unimodal and heteromodal association areas, particularly for the supragranular layers, and the absence of a granular layer 4 in limbic cortex.

Once the increasing architectonic differentiation of cortical layers was recognized, it also became apparent that the pattern of projections

[3] Broca's term *limbic* referred to the limbus or border between the medial cortex and subcortical structures (Pessoa and Hof, 2015). This border of the cortex includes both agranular areas and dysgranular areas (Barbas, 2015). Although some authors refer to only the agranular cortex (periallocortex) as limbic, and the adjacent dysgranular cortex (proisocortex) as paralimbic (Mesulam, 2000), we retain Broca's more general description, because we rely heavily on human clinical evidence that is based largely on this more general description of limbic cortex. Nonetheless, the Structural Model provides clear guidelines for the unique connectivity for agranular and dysgranular areas, and making this distinction is important when possible in interpreting specific evidence.

Table 2
Nature of Control in Adaptive Bayes Process Model vs Current Neuroscience Paradigm.

Anatomical Direction	Function	New Interpretation	Current Neuroscience Paradigm
Limbifugal	Expectancy Prediciton	Feedforward	Feedback
Limbipetal	Actual Input, Error Correction	Feedback	Feedforward

differs for the relatively undifferentiated limbic areas (which project to frontal heteromodal areas primarily from infragranular layers) compared to more differentiated primary sensory and motor areas (which project from supragranular layers) (Barbas, 1986). As more evidence was gathered and interpreted, the Structural Model generalized these early observations to summarize a coherent pattern for the projections from the limbic core of the hemisphere toward the more differentiated sensory and motor neocortices, and the asymmetric reciprocal projections from more differentiated to less differentiated cortical areas (Barbas and Rempel-Clower, 1997). The pathways from areas of simpler laminar elaboration to areas of more complex laminar elaboration originate in deep layers (5–6) and terminate in superficial layers (1–3); it is these limbifugal pathways that are mistakenly defined as "feedback" (sic) in the conventional neuroscience paradigm. In the reverse (limbipetal) direction, pathways from areas of complex laminar elaboration to areas of simpler laminar elaboration originate in superficial layers (2–3) and terminate in middle-deep layers (4–6); these pathways are usually defined as "feedforward" (sic) in the neuroscience literature. Finally, when two areas of comparable laminar elaboration are connected, pathways originate in superficial (2–3) and deep (5–6) layers and terminate through all layers (1–4).

As neuronal density decreases in limbic cortex, dendritic arbors of pyramidal neurons are more ramified (Barbas and García-Cabezas, 2016), apparently supporting less regional differentiation. Of particular significance is the proposal that in agranular cortex, relative to eulaminate cortex, the canonical cortical column circuitry reflects less interlaminar inhibitory control (Beul and Hilgetag, 2015). Thus, with the absence of layer 4 in agranular cortex, error feedback, according to predictive coding theory, must be conveyed to deep layer 3 and upper layer 5. Clearly the inter-areal, cross-laminar communications as well as intralaminar integration described by predictive coding theory must change in some way as these changes in neocortical dynamics are propagated to the cortical columns of limbic areas.

In their review of differences in cytoarchitectonic organization across cortical regions, Beul and Hilgetag (2015) emphasize that the model of canonical cortical columns derived from the highly differentiated primary visual cortex in primates may not generalize well to the varied cortical laminar cytoarchitectonics across multiple cortical areas. If predictive coding theory is to be generalized to account for the mammalian (and human) brain generally, then the architectural differences must be considered to create variations in predictive coding. How can the primitive limbic cortex, with its architecture reflecting the primordial pallium in both archicortical (hippocampal/cingulate) and paleocortical (olfactory/insular/amygdalar) moieties, provide the expectancies for higher control of the neocortex? Limbic influences must take shape within the progression of increasing differentiation from agranular/dysgranular limbic areas to granular neocortical areas. In a functional analysis, we must think that expectancies and evidence take different forms at different levels of the neocortical hierarchy.

As often occurs in biological theory, the most comprehensive interpretation of the increasing complexity of neocortical networks emanating from the limbic base was in terms of evolution. Sanides (1970) reviewed several classical studies of vertebrate cortical architecture (Abbie, 1942; Dart, 1934) that suggested increasing evolution of neocortical complexity from two points of limbic origin, the archicortex (including the hippocampus and related cingulate cortex) and the paleocortex (centered on the olfactory and piriform cortices adjacent to the amygdala). The progressive increase in cytoarchitectonic differentiation of cortical lamina from the both these limbic bases of the hemisphere, as is now clear from studies of primate neocortical connectivity, supports the Sanides hypothesis that neocortical evolution has progressed through *growth rings* that formed as more differentiated cortical lamina evolved from simpler architectures. Mammalian, and specifically primate, evolution has resulted in an increasingly complex neocortical architecture for sensory and motor function, while — for some remarkable reason — retaining the limbic base in more or less the same primitive 3-layered functional form of the premammalian vertebrate pallium (Abbie, 1940; Dart, 1934; Goulas et al., 2019; Sanides, 1970).

Importantly, the major functional divisions of the hemisphere for spatial and object cognition and memory (Aggleton and Brown, 1999; Ungerleider and Mishkin, 1982) can be seen to align with these dual limbic roots, with the dorsal neocortex differentiating from the archicortical base to specialize for spatial cognition, in contrast to the ventral neocortex differentiating from the paleocortical base to specialize for object cognition (Tucker and Luu, 2012). The progression of the dorsal and ventral streams for cognition and memory across the four levels or cortical types is illustrated in Fig. 2, including the sensory pathways into the limbic evaluative areas (here illustrated for vision) and the motor pathways with their limbic motive roots.

Barbas and associates point to another important finding from the studies of primate cortical connectivity: connections arising from areas of a given level of architectural differentiation (described as cortical *type*) tend to terminate in other areas of a similar level or type, rather than crossing levels (Barbas, 2015; García-Cabezas et al., 2019; Pandya and Yeterian, 1984). Thus heteromodal cortex for vision connects preferentially to other heteromodal areas rather than to unimodal or other limbic regions. The implication is that — because connectivity implies function — the four cortical levels or types (limbic, heteromodal, unimodal, primary) form four global divisions of the hemisphere, crossing the specific sensory or motor modalities.

A schematic for visualizing these patterns of connection is shown in Fig. 3. Keeping in mind the separation of the dorsal, archicortical division of neocortex from the ventral, paleocortical division, with only a few points of convergence of their unique cytoarchitectonic features (Eidelberg and Galaburda, 1984), the three top flaps of Fig. 3 may be considered as illustrating the dorsal, archicortical derivation and the three bottom flaps as the ventral, paleocortical derivation. A more accurate depiction of the levels of cortical type in the human cerebral cortex, and the regular patterns of interconnection that follow from cortical types in the Structural Model, can be found in Garcia-Cabezas et al. (2020).

Another observation from the primate cortical connectivity evidence is that the density of cross-pathway interconnections is greatest for limbic areas and decreases progressively with the increased cytoarchitectonic differentiation toward the primary sensory and motor areas (Barbas and Pandya, 1989; Zikopoulos et al., 2018). Fig. 3 provides a schematic for how this might be visualized, with more lines drawn between pathway levels closer to the limbic core. Thus the limbic areas are richly interconnected; the interconnections among heteromodal areas are less dense but still supported by extensive and well known fiber tracts; the interconnections of unimodal areas are less dense; and the primary cortices have only sparse long distance cortico-cortical connections (with the exception of sensorimotor cortex).

2.3. Neuropsychological interpretations of the structural model

The several features of primate cortical anatomy outlined here, including the regular increase in cytoarchitectonic differentiation moving away from the limbic core, the regular and asymmetric pattern of cross-laminar projections within each sensory and motor pathway, and the tendency for cross-pathway projections to be received by an area

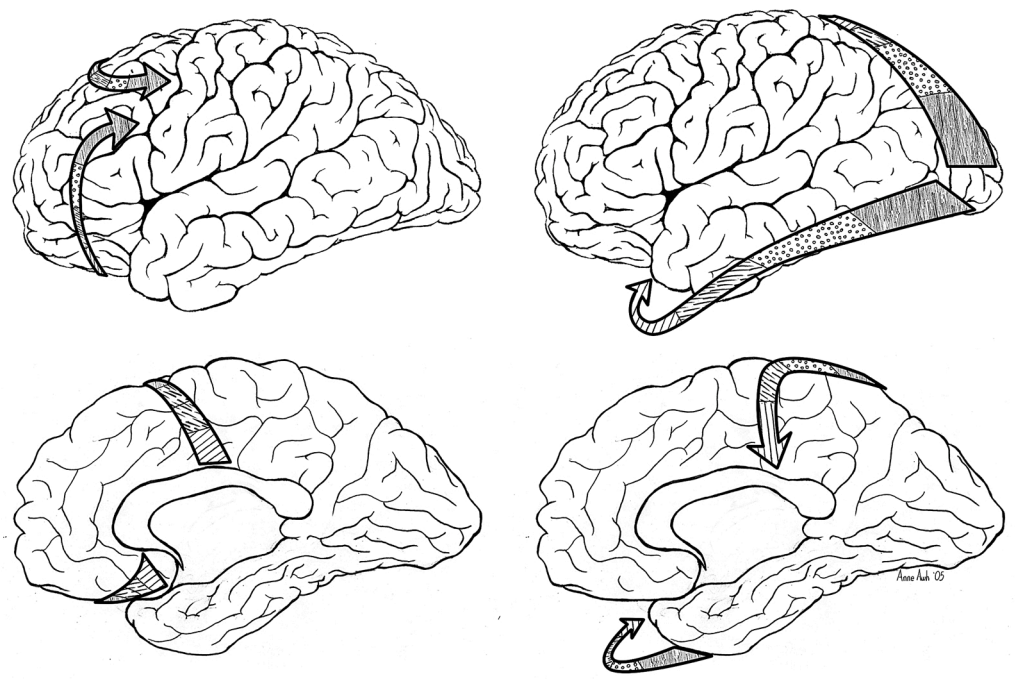

Fig. 2. The alignment of the levels or types of neocortical differentiation with the dorsal and ventral streams for visual processing (right) and motor control (left). Using the same graphic for each level as in Fig. 1 (shaded, stippled, dashed, striped), the "feedforward" direction of visual processing begins in the most differentiated primary visual cortex and progresses toward the least differentiated limbic cortex at the medial border of the hemisphere. The reverse direction of processing, from least to more differentiated, would then be followed in the processing of action control in the frontal brain (left). The important exception to this cartoon generalization is that the granular layer remains poorly developed even in premotor and motor cortex in the dorsal division (Shipp, 2005). Figure from Tucker (2007).

of the same level or type as the sender, have been summarized in series of papers by Barbas and associates as the Structural Model (Barbas, 1995b, 2015; García-Cabezas et al., 2019). Although the tendency in neuroscience is often to focus on a local neural system or function, the Structural Model codifies the modern findings on primate cortical anatomy to provide a framework for the global organization of the primate brain.

This insight into primate connectional anatomy invites theoretical attempts to align models of human experience and behavior with the architecture of connectivity within the cerebral hemisphere. Perhaps the most influential model of human neuropsychology that was consistent with the overall organization of connectional anatomy from the primate studies of cortical anatomy was that of Mesulam (1992, 2000). Drawing from classical neurology (Yakovlev, 1948) Mesulam emphasized that the architecture of the cerebral hemisphere in primates including humans implies a functional model. The limbic base of the hemisphere interacts with the hypothalamus and other subcortical structures in the interoception of the signals for the homeostatic requirements of the organism, and it must communicate these requirements throughout the cerebral hemisphere. At the lateral surface of the hemisphere, the sensory and motor cortices provide the interface with the environment, such that the traffic between these — in the extensive association cortices — must be understood to mediate between the internal biological requirements and the specific sensory and motor capacities of the organism in interfacing with the world. Even with the extensive interest in *functional connectivity* (hemodynamic and electrophysiological correlation) studies in recent neuroimaging research, the need for considering the structure of anatomical connections remains important in a neurocognitive analysis (Mesulam, 2012).

In an effort to align this general architecture of the cerebral hemisphere shown by the primate studies with the neurodevelopmental process of activity-dependent selection of synaptic connections, Tucker proposed that as the neocortex becomes increasingly differentiated, both in embryogenesis and in the continuing ontogenesis of childhood, the connectional architecture of the hemisphere implies that adaptive control of neural activity from limbic areas must provide the regulatory substrate for activity-dependent specification of the neocortex (Tucker, 1992). The process of activity-dependent specification of the global cortical architecture in embryogenesis appears to continue for the activity-dependent specification for cognition throughout ontogenesis. Following this line of reasoning, Tucker (2001) proposed that the connectional architecture of the cerebral hemisphere could be described in traditional neurophysiological terms. The limbic core of the hemisphere reflects the *visceral nervous system* — organizing cognition through elaborating the signals of hypothalamic control of homeostasis. This perspective was first articulated by Yakovlev's observation that the neural control of motility (behavior) must emerge from the *sphere of visceration* (Yakovlev, 1948). With this motive control as the inner boundary, the neocortex's external primary sensory and motor cortices (the shell) reflect the *somatic nervous system* that interfaces the brain's boundary with the world. Even though certain networks are specialized for core visceral versus shell somatic operations (such as emotional interoception at the core and sensory exteroception at the shell), cognition at the psychological level (concepts, reasoning) must span both domains. Cognition appears to be embodied at both core and shell boundaries of the hemispheric networks, not only anchored in sensory and motor referents, but continually guided by the core limbic processes of emotional and motivational control (Johnson and Tucker, in press; Tucker, 2001, 2007; Tucker and Luu, 2012).

More recently, Barrett has also emphasized the implications for psychological theory that are found through considering the functional architecture of limbic and neocortical association areas as clarified by the Structural Model (Barrett, 2016, 2017). Whereas emotions have often been categorized as distinct entities, Barrett emphasizes that the process of interoception provides more general signals to the cognitive process. Barrett proposes that this process can be described as one of *active inference,* in which the function of cognition is to generate predictions about the world that support the allostatic anticipation of homeostatic needs. Because it appears integral to working memory, the limbic level of the hemisphere may be particularly important to consciousness (Barrett, 2017; Chanes and Barrett, 2016).

2.4. Generating affective bias for active inference

Recent theoretical work in active inference has integrated the notion of affective evaluation within the Bayesian equations that structure the relation between prediction and error correction (Hesp et al., 2021). The inference process in this model is stated in terms of the organism's

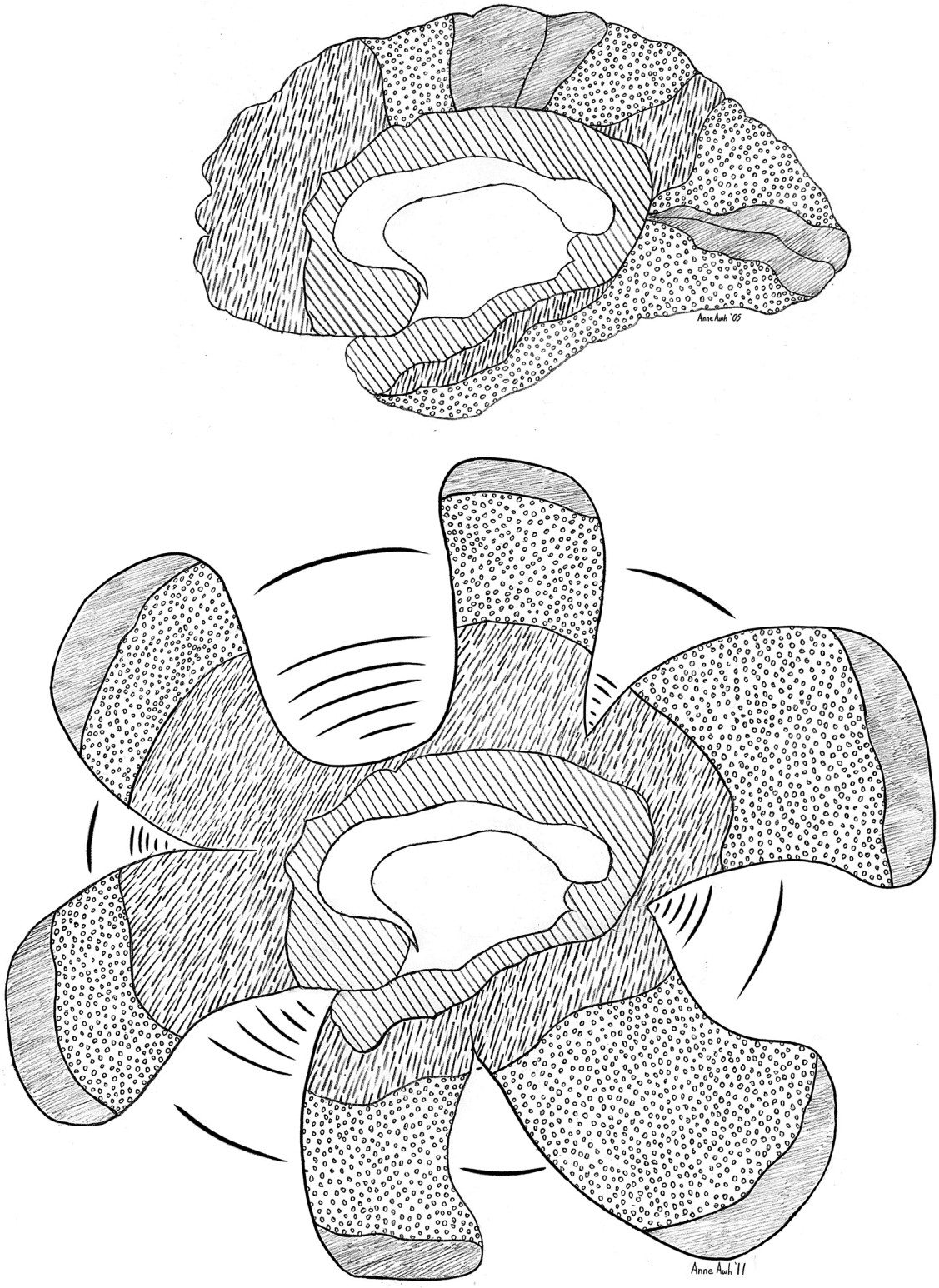

Fig. 3. Because of the extensive folding of the human cortex, the topology of connections within sensory and motor pathways, and the pattern of connections between pathways, is difficult to visualize. This cartoon suggests how multiple pathways (from the limbic base to the primary neocortical area) might be unfolded. Three dorsal pathways and three ventral pathways are illustrated. The lines between pathways serve to show that connections from one level or cortical type tend to target that same level or cortical type, and that these inter-pathway connections are dense for limbic areas, somewhat less dense for heteromodal areas, lesser still for unimodal areas, and virtually absent for primary areas (with the important exception of motor and somatosensory areas) see Zikopoulos et al., 2018 and Garcia--Cabezas et al., 2020. Figure from Tucker and Luu, 2012).

general cognition, rather than the specific negotiations of predictions and errors within the specific regions of the neocortex. Yet the formulation of the model provides a way to integrate affective evaluation in a way that is directly relevant to the monitoring of prediction errors, and thus the ongoing success of the cognitive process.

Hesp et al. (2021) propose that the *precision* term of traditional Bayesian analysis can be reinterpreted to reflect not just the quality of new evidence (which of course is initially unknown) but the affective valence (good or bad) to be associated with the inference process. Successful predictions of valued (phenotypic-relevant) outcomes can build positive affective valence (affective charge) that then provides confidence in subsequent predictions.

Although this model is not related to specific cortical areas, it does illustrate the computational power of higher level (more abstract) representations in regulating behavior. Hesp and associates illustrate the role of affective valence, together with context representation, in a simulation of a rat learning the direction of turning in a maze. Both representations of valence and context were provided in higher-order network nodes, in addition to the lower-order level of the model, thereby simulating the effects of "deep" or more abstract computation. The major effects of the higher level parameters were seen in the ability of the simulated rat to change its behavior (expectations) under conditions of changes in contingencies (position of the reward in the maze). When operating with only lower level control representations, the simulated rat adapted only slowly (mechanistically) to new contingencies. When operating with the higher level affective valence and context priors, the simulation adapted more quickly (we might say insightfully) to change.

Although it is not obvious how principles from a Bayesian simulation can be mapped on to the mechanisms of actual brains, we propose that Hesp et al. (2021) are proposing an important advance to include *affective charge* to bias the organism's confidence in prediction — versus sensitivity to changing the prediction on the basis of error feedback. Furthermore, in developing a neuropsychological theory to describe the adaptive control of the neocortex by limbic networks, this notion of affective charge can help to explain the unique role of the limbic networks at the "top" of the neocortical hierarchy, with their relatively undifferentiated cellular architecture yet apparently influential role in organize the cognitive process organizing the linked networks of the Structural Model. In the next section, we outline the adaptive Bayes process model, suggesting how limbic networks may provide relatively diffuse but affectively charged expectations that orient the neocortical hierarchy to achieve knowledge that is significant for the organism's homeostatic needs. We propose that this model can integrate classical neuropsychological evidence on both motive control and memory control functions of the limbic system, as elementary representations of value in limbic areas become combined with abstract, multileveled cognitive representations across the neocortical hierarchy.

3. Active inference through reciprocal control from higher and lower areas of the cerebral hemisphere

The predictive coding model ascribes specific functions to the regular, asymmetric pattern of connections between adjacent areas of the neocortex, as outlined in Fig. 1. The input projections from the thalamus to sensory cortex primarily target granular layer 4. The adjacent connections to sensory association cortex proceed from superficial layers of sensory cortex toward layer 4 of sensory association cortex, as if the sensory cortex is sending its information as (now processed) sensory input to the adjacent higher association area (Fig. 1). The sensory association cortex then sends trial patterns (*expectancies*) through projections from infragranular layers to the supragranular layers of lower cortex. Predictive coding theory suggests that this architecture serves a Bayesian predictive model, where prior experience in perception allows the higher area of association cortex to send learned, expectant (indeed *feedforward)* control projections to the lower area of sensory cortex, such that the sensory data serves as evidence (indeed *feedback*) to correct the expectancies.

This canonical pattern of cross-laminar connectivity links each sensory area to its association cortex; the same general pattern then links sensory association cortex to heteromodal association cortex; and heteromodal association cortex is then connected to limbic cortex at the core of the cerebral hemisphere (Fig. 1). Although this general outline of mostly sequential connectivity among cortical areas has been known for some time (Jones and Powell, 1970), the tract tracing studies of the 1970s and 1980s revealed the overall connectional architecture of the primate cerebral hemisphere in remarkable detail for the first time, as now outlined clearly by the Structural Model. The intriguing theoretical question is the extent to which the predictive coding model can be generalized to explain the full corticolimbic hierarchy of the cerebral hemisphere. If sensory association cortex provides starting expectancies for the patterns to be decoded in the primary sensory area, does the higher-level heteromodal cortex then provide more general, conceptual, expectancies for sensory association cortex? How are we then to consider the limbic control of its adjacent heteromodal association cortex? Is the limbic lobe a still higher level expectancy constraining incoming evidence? Are there limbic expectancies in the form of homeostatic needs or motives? And how would these be error-corrected by the patterns of neural activity in heteromodal areas?

A specific challenge for understanding the nature of limbic control in terms of predictive coding is to account for the less differentiated nature of cortical lamina in limbic areas that are either dysgranular or agranular (Beul and Hilgetag, 2015). Indication that limbic areas are more dynamic and plastic than eulaminate neocortical areas may be provided by findings that these areas have fewer parvalbumin expressing inhibitory neurons than eulaminate areas (Garcia-Cabezas et al., 2017; Joyce et al., 2020). Is the well-developed inhibitory control in more articulated (eulaminate) neocortex then absent in the more undifferentiated limbic cortex? Does this imply different dynamics of predictive coding, if the inhibitory control for error-correction is absent? Is affective charge more significant for limbic areas with their absence of integral inhibitory control?

By the logic of predictive coding, the higher cortical area provides the prior expectancies for perception or behavior that are then negotiated against the posterior evidence in sensory or motor areas: does this mean that the limbic areas are the highest level of the neocortical hierarchy? If so, then what are the prior expectancies that they generate, and what is the evidence in the adjacent heteromodal area that provides corrective feedback?

These questions arise if we extend the logic of the predictive coding model to the limbic cortex, which — as next in line — would reflect the highest cortical level of the network hierarchy of the cerebral hemisphere. Of course, the traditional assumption in neuropsychology is that the neocortex provides the higher level of (cognitive) control over the (primitive motivational) limbic system. Furthermore, the primary regulatory input to limbic cortex is not in the laminar cortical form (it is indeed the end of the corticolimbic hierarchy) but rather direct projections from the amygdala (Ghashghaei et al., 2007) and specific nuclei of the hypothalamus (Risold et al., 1997). Yet, in spite of these obvious facts, the Structural Model shows the connectivity between limbic cortex and heteromodal cortex follows roughly the same asymmetric cross-laminar pattern as between heteromodal cortex and sensory or motor association areas (Barbas, 2015). How can the undifferentiated (3-layer) limbic cortex — with an architecture little changed from the primordial vertebrate pallium — then provide regulatory control over the extensive heteromodal cortex through decoding the non-laminar amygdalar and hypothalamic signals in order to project them in the laminar-specific form required for predicting prior expectancies?

3.1. Adaptive visceral urges corrected by the evidence of experience

Given their positions adjacent to the heteromodal association areas,

the logic of predictive coding theory would propose that the limbic areas must provide some form of expectancy, a Bayesian prior that predicts the pattern in the adjacent cortical area, presented in the limbifugal (infragranular to supragranular) projections. At their base, the limbic networks include the poorly differentiated, dysgranular allocortex and periallocortex without the full 6 layers of neocortex (Barbas, 2015). Furthermore, there are no adjacent cortical networks "above" the limbic networks in the control hierarchy; rather the input to infralimbic and prelimbic cortex includes direct projections from the hypothalamic nuclei that signal the homeostatic state (Risold et al., 1997). The hypothalamus integrates a wide range of internal homeostatic controls, including neuroendocrine function, temperature regulation, and regulation of organismic states of mood, sleep, and arousal (Risold et al., 1997). Recent embryological studies have shown that the hypothalamus is the developmental base of the telencephalon, consistent with a fundamental role in regulating ontogenesis both structurally and functionally (Wells et al., 2020).

Recognizing these unique foundations of limbic adaptive control from subcortical projections, we suggest that the predictive coding framework may still be instructive for understanding the nature of limbic control negotiated with adjacent heteromodal neocortex, particularly when a mechanism such as affective charge is integrated with the model of information processing. The Bayesian expectancies from limbic cortex may be in the form of elementary urges, perhaps described as *adaptive implications*, taking the form of incipient directions for action and perception, and defined more by their motive significance than prediction of specific sensations or behavioral actions. When linked to perceptual and behavioral options through predictive coding, an adaptive implication may form the motive base for Gibson's concept of *affordance:* an option within the organism's behavioral repertoire with implicit homeostatic significance (Gibson, 1970). The implicit nature of an organismic affordance may be expressed in affective inference theory by the notion of *phenotypic expectancy* (Hesp et al., 2021). The primitive limbic urge then adaptively primes the formation of an expectancy in more differentiated neocortical regions through the homeostatic anticipation of the affordance.

Through a similar line of reasoning, the *evidence* that is presented from heteromodal cortex to limbic cortex through the canonical cross-laminar projections is not simply derived from sensory and motor feedback. Rather, this evidence must be a more fully organized form of conceptual representation. Forged primarily at the level of heteromodal cortex, the representation of evidence organizes the organism's integrated knowledge of the world, assembled from many experiences now consolidated in synaptic memory. Given the lifelong connectivity of the heteromodal area with its limbic foundation, this evidence may take the form of indications — from the assembled integration of prior history — that the limbic need or urge is likely to be satisfied or not in the present, currently activated, environmental context.

This formulation of corticolimbic dynamics within the logic of predictive coding simultaneously addresses the two key questions of neocortical exchange with the limbic system: (1) how homeostatic controls act to motivate the organization of adaptive experience and behavior within neocortical networks, and (2) how the organismic experience represented in the neocortex acts to constrain limbic urges so that they are manifested in contextually appropriate ways. This corticolimbic negotiation remains a Bayesian conditional dynamic of testing evidence on the basis of expectancy, but here the expectancy *prior* is not the prior knowledge, but rather the emergent homeostatic need (in perception) or urge (in action). Furthermore, the evidence indeed affords error-correction, but one that represents the evidence of organismic experience as it recursively consolidates the current (multileveled Bayesian) evaluation of the environment in the context of the developmental experience that has wired the neocortical architecture.

These dynamics of motive control must be integral not just to immediate perception, but to limbic consolidation of memory generally. As a result, the apparent conclusion from this line of reasoning is that the limbic regulation of the neocortex is not limited to instances that are clearly defined as motivational and emotional. Rather, some activity within limbic networks must continually regulate the ongoing consolidation of activity throughout the neocortical pathways, including during quiet waking and during the specific stages of sleep (Diekelmann and Born, 2010). Understanding the nature of limbic organization, including the effective recruitment and control of hypothalamic and other subcortical systems, is thus critical to understanding the ongoing nature of implicit and unconscious inference in memory consolidation, and thus the personal, historical nature of the adaptive foundations of cognition.

3.2. Classical neuropsychology of limbic control

Important context for considering questions of limbic control in relation to human behavior is provided by the clinical evidence on the role of limbic cortex in psychological function and dysfunction. Classically, the two functional roles of the limbic system are motivational control, on the one hand, and memory consolidation, on the other. Although these functions would naturally seem related in a biological theory, the academic and cultural biases in modern science seem to have caused motivation and memory to be considered as separate functions when applied to humans (Johnson and Tucker, in press).

Papez (1937) first identified his "proposed circuit for emotion" through observing that seizures induced in one component of the limbic lobe tended to propagate throughout a limbic circuit, including not only cingulate cortex, but the septal area, mammillary bodies of the hypothalamus, hippocampus, fornix, before returning to the cingulate area. As we will study in the sections that follow, this coincidence of emotional and motivational control with neurophysiological excitability was an important clue that continues to be relevant to understanding the adaptive control of memory in cortical tissue. Building on Papez's neurophysiological analysis, the concept of a limbic system was advanced by MacLean (MacLean, 1958), who emphasized the integral role of limbic circuits in uniquely mammalian behavior, including play and social attachment.

The clinical evidence of limbic control of motivation and emotion came from consistent observations of a loss of both motive control and emotional sensitivity with limbic lesions. Sufficiently extensive (typically bilateral) cingulate cortex lesions, for example, lead to the syndrome of *akinetic mutism* (Barris and Schuman, 1953; Blumer and Benson, 1975; Damasio et al., 1980) in which the patient appears mute and unresponsive but is fully able to speak and act in response to external stimulation (such as being asked a question in a loud and challenging voice). What seems lost is not the capacity to speak and act but the motive initiative. The importance of limbic function to emotional sensitivity is shown by the effect of neurosurgical lesions of cingulate cortex for relief from intractable pain. Following recovery from surgery, the patient can still describe the pain accurately, but no longer reports the associated emotional distress (Flor-Henry, 1977; Knight, 1965; Strom-Olsen and Carlisle, 1971).

Classical evidence on the regulatory dynamics between limbic and neocortical areas includes the observation of increased impulsiveness and loss of self-control with certain neocortical (particularly frontal lobe) lesions. These lesions appear to leave limbic areas without neocortical regulation, producing what are called the disinhibition syndromes (Anderson et al., 1999; Blumer and Benson, 1975; Luria, 1973; Starkstein et al., 1988). Other clinical observations show that facial expressions of emotion may become exaggerated, apparently disinhibited, following neocortical lesions that impair voluntary control of the facial musculature (Monrad-Krohn, 1924). The traditional interpretation is that limbic cortex generates motive impulses — apparently reflecting the immediate influence of hypothalamic drives — that are normally regulated and integrated with the cognitive control of socially appropriate behavior by the neocortex. In the connectivity of the Structural Model, the most important regulating neocortex would be the large heteromodal areas of the human neocortex adjacent to limbic

areas.

A separate literature, with extensive clinical as well as experimental animal evidence, implicates the same limbic circuits and networks in the consolidation of memory (Squire, 1987). The neocortex appears to store memory, allowing sufficiently normal cognition without limbic contribution that even dense amnesia following limbic lesions is often not detected in the clinical setting without explicit testing (Lezak, 1983). However, the patent with a sufficient limbic lesion may be unable to form new declarative memories (Corkin, 2002). Not only hippocampal or cingulate lesions may cause amnesia; equally dense amnesias may follow lesions of the amygdala and associated ventral limbic cortex (insula and anterior temporal pole) (Aggleton and Brown, 1999; Squire, 1986; Yonelinas, 2006). The implication is that ventral (amygdala, paleocortical) as well as dorsal (hippocampal, archicortical) limbic areas are essential for the process of memory consolidation through which new memories are retained in a short term store to allow eventual consolidation within the synaptic architecture of the neocortex (Buzsáki and Chrobak, 2005). Furthermore, this corticolimbic consolidation of memory can only be achieved by the paleocortical and archicortical limbic divisions in concert with their respective ventral and dorsal neocortical hierarchies (Tucker and Luu, 2012).

Even aside from our immediate theoretical effort to extend predictive coding and active inference to explain limbic regulation of the neocortex, the evidence on the essential role of limbic cortex in memory consolidation presents a remarkable mystery to the scientific understanding of the brain and human nature. Apparently, the extensive and complex cognitive operations of the entire human cerebral hemisphere are dependent on the ongoing neurophysiological consolidation of memory provided by the most primitive, and least differentiated, limbic regions of mammalian cortex. It is as if these primitive regions provide some essential cybernetic mechanism for information processing that has been maintained since the elementary 3-layered pallium first appeared in simpler vertebrates.

In his pioneering studies of the architecture of the hippocampus, Ramon y Cajal pointed out that the pyramidal architecture of the amphibian pallium (pyramidal cells on the edge of the lamina) can be observed in the dentate gyrus of the mammalian hippocampus (Harrington, 1991). Furthermore, the structure of the reptilian pallium (pyramidal cells in the center) can be seen in Ammon's horn (Martin-Padilla, 1998a). Certain forms of neurophysiological control of memory function seem to have been irreplaceable through the remarkable variations of vertebrate neurobehavioral evolution that led to our current capacities in organizing the experiences of daily living. As we will see below, in a similar fashion, the archaic neural foundation of the ventral limbic division, rooted in the networks of the extended amygdala, may also continue to be integral to the continuing adaptive control of mammalian, and human, cognitive architecture.

3.3. Adaptive binding theory

In the neuropsychological analysis of motive control of cognition (Derryberry and Tucker, 1990; Luu et al., 1998; Tucker and Luu, 2006; Tucker and Williamson, 1984), an important question has been how to explain motivation and memory as inextricably related functions. Furthermore, the subcortical and limbic regulation of mammalian cognition is found to be not only homeostatic, reflecting current visceral requirements, but allostatic, biasing the cognitive anticipation of events systematically. In studying both normal and pathological emotional influences on cognitive appraisal in humans, the integral alignment of motive control with the control of memory and cognition soon becomes apparent, indicating that the binding of information in memory is often guided by generic affective influences that shape the quality of expectancy. *Adaptive binding theory* recognizes that evolution has created human memory systems to integrate emotion and motivation as the regulatory basis of the cognitive process (Tucker and Luu, 2006, 2012). In the present formulation of the adaptive Bayes process model, we are attempting to extend this fundamental alignment of motive and memory functions from adaptive binding theory to the operations of active inference within the explicit architecture of the Structural Model.

One implication of the evidence on adaptive binding is that *affective charge*, or the bias of expectancies to favor confidence or uncertainty (Hesp et al., 2021), is not a metacognitive construct in the sense of higher level awareness, but a primitive bias that often operates unconsciously. The unique roles of the dorsal archicortical and ventral paleocortical limbic divisions, characterized by Tucker and Luu (2012) as *impulse* and *constraint*, respectively, may be integral to explaining the familiar syndromes of psychopathology (Giaccio, 2006) as well as more normal cognition. These motive controls clearly operate to shape conscious experience, but often without awareness of the shaping. The manic individual shows excessive optimism that quickly becomes maladaptive, leading to uncontrolled spending, impulsive sexual advances, and unwarranted confidence in personal abilities. When the manic episode is followed by depression, the same person loses self-confidence, as well as all hope for the future (APA., 2013; Tucker and Luu, 2007). The implication is that the depression-elation dimension of mood applies inherent controls on cognition (mediated primarily through the dorsal corticolimbic division) that support the normal impulses for hedonic gratification.

The unconscious nature of such mood state biases in cognition are readily apparent in clinical work: the depressed person typically has no ability to reflect that he was manic and grandiose the week before. Mood states of depression and elation vary in severity in a continuous fashion in the normal population (Akiskal, 1986; Akiskal et al., 2003). As a result, mood-dependent biases in cognitive appraisal are not just pathological but may be an integral component of all decisions (Isen, 1987). It is these motive influences on ongoing cognition that we propose to reflect the limbic controls, and perhaps the affective charge, that organize active inference throughout the neocortical hierarchy.

A similar, but qualitatively unique, allostatic bias is seen in anxiety disorders, where the person is not only sensitive to perception of threat but often perceives unthreatening circumstances as dangerous. The loss of self-confidence may appear similar to that in depression, but it is specific to the anxiety disorder. For example, rather than losing hope (as in depression), the anxious person avoids social contact to avoid threat (APA., 2013; Tucker and Luu, 2007). The lack of normal positive affect in depression indeed distorts cognition, but in ways distinct from the active engagement of negative affect in anxiety (Tellegen, 1995; Tucker and Luu, 2007). These exaggerated effects are not just pathological anomalies; normal anxiety may be essential for effective risk perception (Johnson and Tversky, 1983; Tucker, 2007).

Thus the mood dimensions of elation and anxiety appear to be integral motive controls in normal cognitive function, and they constrain cognitive appraisal in ways that are directly relevant to the control of of predictive coding. In mania, uncertainty is minimal, personal expectancies are followed with confidence, and discrepant evidence has little capacity in error-correction. In anxiety, uncertainty dominates expectancy, leading the person to search for relevant evidence. However, the exaggerated affective charge of expectancies can lead the anxious person to be insensitive to evidence even here. Expectancies of the highly anxious person may be unresponsive to error-correction when the evidence indicates that reality is safer and more pleasant than expected, and thus is incongruent with the mood state bias that primes expectancies for threat.

In cognitive theory, the priming of concepts in memory can be described as a process of *spreading activation*, as access to one domain may result in sequential activation of related semantic domains (Meyer and Schvaneveldt, 1971; Neely, 1977; Yaniv and Meyer, 1987). However, the affective quality of events may contribute to a more diffuse, generic form of affective priming than is typical for less affectively charged spreading activation. Bringing attention to a medical risk, for example, not only increases perception of risk for other medical dangers (as would be predicted by local spreading activation), but it also primes

semantically unrelated negative events such as risk from criminal acts (Johnson and Tversky, 1983).

Thus spreading affective priming may be more diffuse than spreading semantic activation, perhaps reflecting the more diffuse connectional organization of limbic areas in the Structural Model. Nonetheless, psychometric evidence indicates that affective priming may form a basis for semantic priming, global and diffuse as it is. For example, there are two major dimensions of affective structure that emerge in factor analysis of self-description with trait questionnaires, Positive Affect and Negative Affect (Tellegen, 1985; Thayer, 1989). These correspond directly to the depression-elation and calm-anxious dimensions of mood seen in clinical settings (Tucker and Luu, 2012). Although psychometric factor analyses of emotion questionnaires were traditionally interpreted with a different rotation of the dimensions (to produce an *arousal* factor separate from an *valence* factor (good/bad), Tellegen's extensive analyses suggested that normal variations in mood were more consistent with the PA/NA rotation. This interpretation is congruent with the dimensionality of the exaggerated mood states in both personality disorders and severe psychopathology (APA., 2013), and with the depression-elation and calm-anxiety dimensions of neural arousal (Tucker and Luu, 2012). Given this evidence, the affective charge of predictive coding must have not one allostatic valence control, but two: the Positive Affect of depression-elation and the Negative Affect of anxiety.

The integral affective priming of meaning in observed in the psychometric (self-report) literature appears to apply to lexical semantics generally. Osgood and associates used the method of factor analysis to examine the dimensionality of cognitive appraisal in a wide variety of applications with the semantic differential method (Osgood et al., 1957). This method involves reporting on the connotative (rather than denotative) meaning of words. An example might be rating the architecture of a new building on dimensions anchored by the terms *simple vs complex*, or *loud vs soft*. The fact that people make these ratings easily, with good agreement across individuals, emphasizes the generality of connotative as well as denotative meaning (Osgood et al., 1957). In factor analysis of these ratings in many studies, a common dimensionality emerges, reflecting factors labeled as *evaluation, activity,* and *potency*. Because evaluation describes the majority of the variance, a similar rotation as Tellegen advocated would be consistent with the two-factor, Positive Affect and Negative Affect, structure in self-report, indicating that the dimensions of affective priming of cognition arise from the dimensions of human mood states, and these are fundamental to linguistic meaning generally, such that their influence becomes apparent in the global semantics of connotative meaning.

Within adaptive binding theory, these psychometric insights are combined with the neuropsychological evidence on the limbic basis of both affect and memory (Derryberry and Tucker, 1992; Luu et al., 1998; Tucker and Luu, 2006). Meaning is anchored in reality through both cultural convention and personal experience in contacting the world at the somatic (sensory and motor) interface, in the process of gaining knowledge that is normally considered as the cognitive process. In addition, however, cognition is anchored in feelings at the visceral limbic base of the cerebral hemisphere, such that adaptive cognition requires a negotiation of both visceral limbic and somatic neocortical boundaries of the corticolimbic hierarchy. The psychometric evidence shows that meaning — as reflected in the structure of verbal semantics — is grounded in the allostatic mood controls of elation and anxiety (PA and NA). These several lines of reasoning point to the limbic basis for the *feeling of what happens* (Damasio, 1999): the affective state that serves as the integral monitor for the process of experience.

The basic influences of affective charge in unconscious inference are likely to be concrete, primitively exciting or depleting the hedonic valence of an expectancy and thereby biasing its negotiation with evidence. If these influences reach awareness, the result may be the feeling of confidence in the knowledge and its current fit with the world. Although motive controls (expressions) and affective charges (feelings) seem inherently concrete, they may be combined with the personal experience assembled in heteromodal cortex to achieve abstract concepts that compose the higher levels of the neuropsychological network hierarchy. The Hesp et al. (2021) simulation of Bayesian affective inference is instructive here. The nodes reflecting affective charge and context information in the simulation of the rat's inferential engine are not only operational at a basic level, but are also incorporated at a higher (hidden or abstract) level. In contrast to when only the basic level nodes were operative, the representation of valence at a structurally higher (more abstract?) level gave the rat the capacity for rapidly adjusting actions in response to a changed environment. If limbic contributions to motivation and emotion can themselves become more abstract in guiding behavior, then we might understand how adaptive conceptualizations in limbic-heteromodal networks may contribute to optimal levels of human thought and self-regulation, indeed representing the highest level of the neocortical hierarchy.

Our effort here is to more fully articulate the notions of adaptive binding in terms of the architecture of the Structural Model and the process of active inference within that architecture. From the classical neuropsychological evidence we can see that is important to understand not only how limbic areas regulate neocortical networks but also how they are regulated by them. The connections of the Structural Model go both ways (although asymmetric across cortical lamina), as do the causal influences between primitive limbic motive influences and more articulated neocortical representations of heteromodal neocortex.

When cognition becomes overly constrained by the mood state, in excessive depression or anxiety, it is impressive how effective Cognitive Behavior Therapy has proven to be in helping the person use rational appraisal and deliberate cognitive control to manage their personal affective bias (Tucker and Luu, 2007). In our extension of predictive coding theory to the limbic base of the neocortical hierarchy, we propose that the affective implications from limbic networks act as primitive priors of motive control to negotiate with the evidence of experience that is provided by the neocortical hierarchy generally, and that is mediated proximally by heteromodal cortex. In addition, the limbipetal feedback from heteromodal cortex may provide an experience-based conceptual representation to regulate the affective priming (in the moment) and adaptive binding (in memory consolidation) thereby cognitively regulating strong affective charge.

Because limbic motive priors appear to be charged in ways that allow affective priming of semantic space, motivated cognition might be described as an *adaptive Bayes process*. The prior expectancies from personal experience are inherently charged with adaptive significance. In addition to a motive basis in specific homeostatic requirements, such as hunger or lust, the clinical neuropsychological literature suggests that each motive prior is charged by a more general allostatic mood state: varying degrees of elation or anxiety that set the tone of cognition. Within a predictive coding framework, including negotiation of causes and states and the weighting of expectancy vs evidence, the allostatic biases of the person's mood states tune the process globally, in line with a generic affective assessment of the adaptive context for experience and behavior.

How are expectancies — either the elementary motive priors or the more articulated cognitive expectancies formed in heteromodal networks — charged with motive significance? Is there a neurophysiological mechanism — such as may be coded in the limbifugal infragranular to supragranular projections — for recruiting limbic drive in a way that could operate like affective charge in Bayesian affective inference? Important clues to the neural mechanisms of limbic motive charge, and thus the adaptive foundation for the multiple levels of predictive coding in the neocortical hierarchy, come from neurophysiological evidence that limbic circuits regulate not only mood states and memory consolidation, but the electrophysiological excitability of the neocortex. This evidence suggests that the regulation of neural excitability underlies the allostatic bias of cognition and memory consolidation in ways that could explain the neurophysiological nature of the negotiation of expectancy

with error-correction at each level of the corticolimbic hierarchy.

4. Neurophysiology of corticolimbic excitability: seizures, anxiety, and error-correction

The control of neural networks requires tuning the balance of excitation and inhibition. In the application of predictive coding theory to models of canonical cortical networks (Bastos et al., 2012), one suggestion has been that the neural projections conveying expectancy (in the outgoing or limbifugal direction) are primarily inhibitory, consistent with the notion that prior expectancies inhibit the error signals of the adjacent networks. Similarly, Bastos et al. (2012) cite evidence suggesting that error propagation from sensory input, in the incoming, limbipetal (incorrectly described as "feedforward") direction of cortico-cortical signaling, is mostly excitatory.

Classical findings on the limbic control of neocortical excitability seem to contradict these proposals, showing that — at least in certain modes of operation — limbic areas act to increase the excitability of the neocortical networks to which they are connected. Ignoring for the moment the limbic control of thalamic, striatal, nucleus basalis, and brainstem reticular formation influences, all of which are highly relevant to neocortical excitability (Tucker and Luu, 2012), the direct connectivity of limbic cortex to the neocortex is mediated primarily through the same cross-laminar connectivity described by the Structural Model and interpreted by predictive coding theory. If the limbic connections serve primarily to excite rather than inhibit the neocortical targets, then this influence should be understood within a predictive coding model, particularly one that includes affective charge.

On the other hand, other recent findings, specifically on the mechanisms of memory consolidation in sleep, also suggest the reverse may occur, that limbic activity can mediate inhibitory synchronization of cell assemblies in the process of memory consolidation. A brief review of these two forms of limbic influence in this and the next section may provide preliminary clues to the neurophysiological properties of limbic control of the neocortex.

A remarkable phenomenon in experimental neurophysiology is *kindling*, in which repeated electrical stimulation of a neocortical site results in increasing electrophysiological responses (*after-discharges*) not only at the stimulated site but also in limbic cortex, often including the hippocampus. A condition similar to epilepsy may be artificially induced by sufficiently strong repeated cortical stimulation, leading to a continuation of both limbic and neocortical discharges in the absence of external stimulation (Bertram, 2007; Fisher, 1989). In the most typical form of human epilepsy, spontaneous or idiopathic temporal lobe epilepsy (described as partial epilepsy in the current clinical jargon), limbic areas become the most typical sites of seizure discharges, and limbic (amygdala-hippocampal) resections typically result in cessation of seizures (Holmes et al., 2000). The experimental kindling phenomenon suggests that neocortical networks recruit the limbic networks, which must be through the linked corticolimbic hierarchy of the Structural Model. Furthermore, the limbic networks are not only sufficiently excitable, but immediately modifiable by activity-dependent plasticity, to then generate continuing and often chronic pathological discharges of epilepsy in the experimental animals. Similar mechanisms of limbic kindling have been implicated in the etiology of human epilepsy (Fisher, 1989). The synchronization between limbic and neocortical areas often appears to be mediated by the limbic theta rhythm, which is observed to recruit the pathological network dynamics of many seizure onsets (Kuo et al., 2018).

The relevance of limbic regulation of neocortical excitability to the mechanisms of normal memory consolidation is suggested by two striking findings. First, kindling may be classically conditioned (Janowsky et al., 1980; Myslobodsky et al., 1983), raising the possibility that limbic regulation of neocortical excitability could be a causal mechanism in the normal learning process. Second, seizures impair memory, not just of recent experience, but with a progressive retrograde effect that points to the dynamic nature of long-term memory storage in the neocortex, and that implies that the memory network is directly accessed by the seizure discharge.

It is commonly observed that retrograde amnesia extends into the past proportionally to the severity of the seizure: the longer the seizure the more past is lost. However, the duration of amnesia following seizures is typically minutes and hours (Drane et al., 2006). Longer term memory loss also appears proportional to the degree of disruption induced repeatedly by seizures, as is observed for medically induced seizures. Seizures are induced by electroconvulsive therapy (ECT) to treat depression, apparently by disrupting some poorly understood mechanisms of mood regulation that become chronically dysfunctional, but that may be interrupted by ECT with immediate mood improvement (Sackeim et al., 1995). In studying the damage to long-term memory caused by repetitive ECT, Squire and associates developed an assessment of the patient's memory for television programs that could be dated to specific past years (Squire et al., 1979). The results showed that, whereas memories older than 3 years were relatively unaffected (suggesting a transition to a more permanent if less accurate storage), memories for television programs within the last 3 years were significantly impaired in patients receiving ECT. Apparently, memory consolidation must be maintained by some active neurophysiological process that can be disrupted if it is not ongoing in time over several years.

Seizures and epilepsy reflect pathological forms of neurophysiological excitability, and may not seem directly relevant to the limbic regulation of adaptive cognition in everyday living. However, an important observation is that patients affected by temporal lobe epilepsy show a high incidence of anxiety disorders (Vazquez and Devinsky, 2003). Consistent with Hughlings Jackson's initial clinical observations, the anxiety seems not to be related to worry over seizures, but rather an intrinsic effect of the neuropathology (Vazquez and Devinsky, 2003). The implication may be that the corticolimbic sensitization associated with the epileptic pathology somehow affects the ongoing cognitive appraisal of daily life.

Providing experimental support for this observation, experimental studies of partial kindling of the amygdala in animal studies by Adamec and associates have shown that excitation of limbic areas with electrical stimulation, even though it is maintained below the threshold of inducing seizures, leads to a sensitization of the animal's apparent anxiety as reflected in defensiveness and avoidance of novelty. The limbic (amygdala) sensitization of excitability at subseizure levels thus shows a specificity of motive control that, for the fight-flight priming that is typical of amygdalar influence, reflects an integral limbic regulation of the ongoing cognitive appraisal organized across the neocortex (Adamec, 1990, 2001; Adamec and Morgan, 1994).

These several lines of evidence suggest that — at least under certain conditions — the limbic regions of the hemisphere can be recruited to exert an excitatory rather than inhibitory influence on the functioning of the neocortex. Furthermore, this limbic sensitization of excitability imbues cognition with a specific adaptive, allostatic bias, in this case anxiety. The regulation of excitability within a physiological range seems to be necessary for maintaining effective, and ongoing, memory consolidation, and we have evidence that this same limbic-cortical recruitment may become dysregulated in kindling and seizures. Furthermore, at least for the role of the amygdala and its unique adaptive influence on the neocortex, an increase in cortical excitability leads to a specific affective charge toward the perception of threat mediated by the mood state of anxiety. As we consider the role of a limbic motive control shaping an adaptive Bayes inference process, there may be both neurophysiological and functional implications for projecting expectancies, in the limbifugal or outgoing direction, and for integrating error-correction, in the limbipetal or incoming direction.

Thus, cognition is not only under homeostatic control, linked to specific biological need states. Rather, there are also generic allostatic mood states, such as elation and anxiety, that bias the cognitive process

to anticipate general adaptive directions of experience and behavior. In one case the mood state of elation acts an allostatic bias to anticipate beneficial and pleasurable opportunities. In another case the mood state of anxiety primes the corticolimbic hierarchy for cognitive appraisal of aversive threats (Tucker and Luu, 2012).

In contrast to the role of affective charge in the affective inference model, which involves monitoring the success of prediction (Hesp et al., 2021), observations on the mood state bias of cognition, such as the enhanced anxiety and threat priming with amygdala kindling (Cavanagh and Shackman, 2015), thus suggest that expectancies arising from limbic influences may engage integral allostatic mechanisms of motive control to bias expectancies directly, where weighting of evidence is then biased to be congruent with this feedforward motive control. Clinical observations of cognition in anxiety disorders show that the appraisal of threat easily overrides the precision of the objective evidence, even as the evidence is distorted to be congruent with the motive bias. In this exaggerated allostatic process, strong anxiety leads to a breakdown of rationality, including irrational avoidance and exaggerated error-correction (APA, 2013). One example is paranoia, in which anxiety constrains the interpretation of evidence to be consistent with the anxiety, regardless of the veridical threat. Another example is *checking* behavior, in which the person with obsessive-compulsive disorder cannot refrain from checking the lock on a door that is known to be locked. The abnormal neocortical excitability in anxiety not only primes threat but enhances the uncertainty of expectancy and simultaneously distorts the process of error-correction.

The neuroimaging evidence of amygdala engagement in human anxiety (Cavanagh and Shackman, 2015) suggests that the mechanism of amygdala kindling may be relevant to human motivation in many contexts. The allostatic bias of anxiety seems to reflect an increased excitability of corticolimbic communication linked to the specific limbic drive that the amygdala and its ventral limbic networks bring to cognition. Under the limbic drive of anxiety, the Bayesian process is biased to suppress current impulses (expectant behavioral priors) and to strongly weight error-correction (detecting threats). Although such mechanisms appear to be easily exaggerated in anxiety disorders, it may be that this same mechanism of limbic allostatic bias, in optimal cases as gleaned from accurate appraisal of the current context, are relevant to managing decisions under uncertainty in normal as well as disordered mental states (Slovic, 1987).

An example of limbic motive control of decisions was provided in an early study of personality differences in the electrophysiological activity associated with error-monitoring. Luu observed that university students higher in anxiety (Negative Affect) showed significantly increased amplitude of the Error-Related Negativity (ERN) over the medial frontal area in response to making errors on an attention task (Luu, 1998; Luu et al., 2000). In examining the EEG features generating the averaged ERN responses, it became clear that midline frontal (limbic) theta becomes phase-aligned as errors are detected (Luu et al., 2004). This analysis implies that effortful control of decisions engages a synchronization of activity in the anterior cingulate cortex by the limbic theta rhythm (Dehaene et al., 1994; Miller, 1991). Given the strong ventral limbic input to the anterior cingulate cortex (Price and Amaral, 1981; Price et al., 1996), the amygdala priming of neocortical excitability in anxiety may be observed to enhance the neurophysiological mechanisms of perceived uncertainty of expectancies, as well as the subjective sensitivity to errors, in many decision contexts (Cavanagh and Shackman, 2015; Slovic, 1987; Tucker and Luu, 2012).[4]

[4] Although we have emphasized anxiety in the adaptive priming of cognition, there are also suggestions that the mood disorders of depression and mania may reflect kindling of corticolimbic excitability (Harkness and Tucker, 2000; Post, 1986; Post et al., 1994). Trauma and strong emotional responses appear to kindle limbic sensitization: previous episodes of depression are the best prediction of future episodes (Lewinsohn et al., 1999).

4.1. Neurophysiology of limbic inhibitory synchronization: deep sleep, anxiety relief, and memory consolidation

Intuitively, it seems as if the brain would decrease its excitability with increasing time awake, as we become sleepy. However, considerable evidence suggests that neurophysiological activation of the cortex increases with time awake (Meisel et al., 2013), such that sleep may be required to reverse the extended neural activation of daytime alertness. A theoretical explanation has been suggested by the *synaptic homeostasis hypothesis*, which proposes that sleep results in a normalization of synaptic weights that have become progressively strengthened through waking neural activity (Cirelli and Tononi, 2008; Tononi and Cirelli, 2006). Just as limbic drive may exaggerate the excitability of the cortex in states of anxiety, the limbic regulation of sleep may normally down-regulate cortical excitability, apparently through the same limbic-neocortical projections.

One clue to how this might occur comes from observations that a region of cortex that has been activated by daytime functioning (such as a region of parietal cortex particularly engaged by a spatial cognition task) shows strong Slow Oscillations (SOs) during deep sleep (non-REM stage 3 or N3) the following night (Huber et al., 2004). Apparently, some homeostatic mechanism is engaged by daytime functional activation of the neocortex that leads to compensatory or recovery activity mediated by the N3 SOs.

The functional importance of deep sleep SOs to cognition has been emphasized by findings showing that experimental enhancement of SOs with transcranial electrical stimulation results in improved daytime memory performance (Marshall et al., 2020, 2006). SOs have been associated with a suppression of local neural firing, in what is described as a *down state*, before activity resumes at the end of the SO (Nir et al., 2011). Recent examination of the small residual activity during the down state has suggested that this residual activity reflects a robust cell assembly, a group of neurons involved in replay of activity during previous spatial learning (Todorova and Zugaro, 2019). This observation may suggest that the suppression of regional activity by the SO achieves not just a generic synaptic stabilization, but a kind of figure-ground selection. In this process, the GABAergic inhibition of the ground allows the figure, a robust functional neuronal ensemble that survives the inhibition, to emerge (Todorova and Zugaro, 2019). This enhancement of key neuronal ensembles may reflect a fundamental mechanism of memory consolidation, which can be seen as a necessary complement of predictive coding that occurs in each night's sleep.

In localizing human SOs of deep sleep with dense array EEG, Morgan and associates have recently found that, whereas some SOs may emanate from neocortex, the majority originate from focal discharges of limbic cortex, specifically in anterior medial temporal and caudal orbital frontal areas (Morgan et al., 2021). These limbic sites generating the majority of SOs in normal adults are localized in the paleocortical or ventral limbic division, often described as the *extended amygdala* (Heimer and Alheid, 1991). This localization of SOs may be significant in light of recent findings that, although they reflect the local electrophysiological activity of cortex, SOs are stimulated by projections of the claustrum (Narikiyo et al., 2020). Recent embryological studies have shown that the claustrum develops in close alignment with several paleocortical areas of the ventral limbic division including piriform cortex, amygdala, and insula (Binks et al., 2019). Not only does genetic ablation of the claustrum projections block SOs, but optogenetic activation of the claustrum results in postsynaptic activation of GABAergic inhibitory interneurons that create a down-state in the cortex, followed by an up-state transition typical of normal SOs (Narikiyo et al., 2020). As the claustrum inhibitory suppression generates the SOs in ventral limbic areas, these areas apparently propagate the down states to create the local down states that have been observed in widespread areas of the human neocortex in intracranial recordings (Nir et al., 2011). This propagation must occur through the pattern of connectivity described by the Structural Model, such that the regulatory effect of ventral limbic

inhibitory suppression during deep (N3) sleep appears to be integral in some way to the neocortical mechanisms of consolidating the outgoing expectancies and incoming error-correction described by predictive coding theory.

4.2. Compensatory limbic inhibition in epileptic sleep

The apparent homeostatic enhancement of deep sleep SOs observed for focal cortical cognitive and behavioral activation has also been observed for the abnormal corticolimbic excitability of epilepsy. The importance of sleep mechanisms to the control of seizures has long been emphasized in neurophysiological studies (Amzica and Steriade, 1998; Steriade, 2003). Dense array EEG recordings of a human patient showed that seizure oscillations, at the onset of a clinical seizure, were co-localized with this person's SOs (Amzica et al., 2009; Tucker et al., 2009). More recent dense array EEG studies have further suggested that localization of an epileptic patient's SOs may be useful in identifying the typical seizure onset zone (Moffet et al., 2020).

These results imply that the abnormal neocortical excitability associated with epilepsy is similar to functional activation from a cognitive task in creating some form of local neocortical homeostatic requirement for inhibitory control that is then recruited in deep sleep to generate targeted SOs. In the active phase, the excitability engaged by corticolimbic recruiting in kindling and seizures may share similar mechanisms as the excitability propagated between limbic and multiple neocortical levels in the normal process of motivated cognition. In the sleep and recovery phase, the neurophysiological inhibition reflected by SO discharges at the site of the seizure focus may be a healthy compensatory response to pathological corticolimbic activation in epilepsy, reflecting the mechanism of inhibitory suppression of ancillary network noise to support the enhanced signal of essential cell assemblies in memory consolidation (Todorova and Zugaro, 2019).

4.3. Corticolimbic excitability and the sleep deficits in anxiety disorders

Given the common increase in anxiety in patients with epilepsy, it seems reasonable to question whether the apparent inhibitory synchronization of neocortical targets by SOs in deep sleep is relevant to normal limbic regulation of the specific threat-related corticolimbic excitability in anxiety. It is commonly observed, of course, that being anxious means losing sleep. However, the reverse causality, where sleep restriction leads to increased anxiety, is not intuitive, but is now well documented (Simon et al., 2020). Furthermore, it is deep sleep and SOs that are particularly impaired in anxious persons, including generalized anxiety, panic disorder, and post-traumatic stress disorder (Horváth et al., 2016; Simon et al., 2020). The finding that SOs in normal individuals are generated primarily by peri-amygdalar, ventral limbic cortex (Hathaway et al., 2021; Morgan et al., 2021) may be relevant to the importance of inhibitory consolidation of adaptive corticolimbic excitability in normal neuropsychological self-regulation, where anxiety can be traced to motive control of the neocortical hierarchy by the amygdala and its paleocortical networks.

As noted in the introduction, the focus of the present paper is on the networks of the cortex and limbic system, and this has led us to ignore the multiple subcortical systems that are essential to regulating the neuraxis, including direct influences on the excitability and integral adaptive biases of the corticolimbic hierarchy. Within predictive coding theory, tuning the balance between expectancy and error-correcting has proposed to be mediated by subcortical dopamine systems that weight the precision of the evidence as processed in cortical networks (Friston et al., 2012). The importance of dopaminergic regulation to anxiety, and thus the example of adaptive control of the neocortex emphasized in the present paper, has been integral to several lines of evidence and theoretical approaches (de la Mora et al., 2010; Derryberry and Tucker, 2006; Gray, 1982; Kokkinidis and Anisman, 1980; Tucker and Derryberry, 1992; Tucker and Williamson, 1984).

5. Limbic resonance in the motive control of Bayesian cognition

Thus both general neuropsychological observations and the specific neurophysiological mechanisms of excitability priming and inhibitory synchronization may provide insight into the way that limbic areas provide adaptive control of the neocortex. The regulation of cortical excitability may be directly relevant to the cognitive operations of the neocortex whose organization is explained by the Structural Model and whose information dynamics are addressed by the theoretical models of Bayesian active inference. The limbic control of the neocortex appears to be organized through the same cross-laminar projections that are found to convey expectancies across multiple levels of the neocortical hierarchy. Yet these limbic influences are obviously charged with motive implications, causing this first, deepest level of expectancy in the human cerebral hemisphere to act not as propositions about the state of the world, but as organismic needs and urges implying both the specific homeostatic requirements and the general allostatic mood states that then affectively charge the expectancy hierarchy with anticipations of success or threat. Because each level of expectancy must reflect some continuity with its predecessor in the hierarchy, it seems that the motive control (and affective charge) must be maintained through limbifugal feedforward propagation throughout the hemisphere, refined as it is by ongoing negotiation with increasingly current levels of evidence propagated in the reverse, incoming or limbipetal feedback direction of information processing.

The adaptive Bayes process model may provide a preliminary way to interpret the classical neuropsychological evidence that limbic and neocortical areas exist in a kind of dynamic balance. Limbic areas appear to resonate to significant cognitive representations — perceptual appraisals and action affordances — in ways that excite and maintain the neural activity (active cell assemblies) of these representations. Neocortical association areas then not only integrate this motive bias with mood-congruent evidence, but inherently reciprocate control in the normal brain by recruiting, and organizing in more complex network structures, both the specific motivational impulses and the general emotional reactivity of limbic areas.

Neurophysiological studies provide congruent mechanistic evidence for this functional neuropsychological interpretation, indicating that limbic connections regulate the excitability of neocortical networks through the organized cross-laminar network structure described by the Structural Model. The theoretical implication is then that the regulation of corticolimbic excitability operates through the identical network structure that mediates cognitive organization, and is in fact identical with the motive control of the cognitive process. The limbic recruitment of cortical excitability may be the literal mechanism for the spreading activation of semantic association in the process of organizing meaning. The normal regulation of network excitability is experienced in everyday cognition as the subtle excitements of everyday meaning. But the same mechanisms may become exaggerated to the point that the neurophysiological regulation of neocortical excitability is more obvious, as in the pathological dysregulation of epilepsy, or in the kindling and sensitization of neocortical activity in the defensive cognitive appraisal that can distort reality-testing in states of strong anxiety.

In the neurophysiological mechanisms of downscaling cortical excitability in sleep we find a complementary form of dynamic balance. The limbic resonance engaged to signal the significance of cognitive activity appears to be balanced by a kind of compensatory inhibitory control in deep sleep. This inhibitory control is applied specifically by the ventral limbic discharges in slow oscillations of deep sleep, which are, in turn, stimulated by projections from the claustrum. The resulting limbic discharges appear to propagate their down states through the linked hierarchy of the neocortex, thereby effecting some essential mechanism of memory consolidation, such as pruning the adaptively significant cell assemblies, at the same time as they suppress global neocortical excitability to achieve the synaptic downscaling of sleep.

When SOs are inadequate to modulate neocortical excitability, the cumulative result may be the pathological maintenance of anxiety, or — in the presence of pathological neuronal excitability — the corticolimbic sensitization of epilepsy.

6. Conclusion

The important advances in understanding the network architecture of the cerebral hemispheres reflected in the Structural Model can be used to frame a theoretical analysis of the roots of predictive coding in the motive control from limbic regions, through what we have described as the adaptive Bayes process model. In interpreting the parallels of neuropsychological with neurophysiological aspects of limbic control, we follow a neurodevelopmental analysis that recognizes the identity of the cognitive process with the activity-dependent plasticity of cortical synaptic organization (Tucker and Luu, 2012). Cognition is the adaptive Bayes process, achieved through the continuing cortical network differentiation throughout ontogenesis (Wright and Bourke, 2020). As a result, the neurophysiological regulation of excitability and the adaptive control of cognition must be two ways of describing the same thing (Tucker and Luu, 1998). The continuing plasticity of limbic networks into adulthood has been emphasized as an important basis for the organization of human learning (Barbas, 1995a). In the context of the extended human juvenile period, this limbic plasticity may explain the vulnerability of the human brain to emotional disorders in the challenging transition to maturity (Barbas, 1995a, 2015).

A theoretical model describing the affectively-charged relations between limbic cortex and more differentiated neocortex may allow a predictive coding framework to be generalized as a preliminary Bayesian theory of the organism. The interaction across the neocortical hierarchy is not just information exchange in an abstract sense, but appears to reflect a neurophysiological sensitization of active cognition during the day and an integral process of inhibitory synchronization and apparent stabilization of learned representations during sleep. The outgoing, limbifugal, expectancies may be charged not only with specific homeostatic concerns, but also with more general allostatic biases anticipating success or threat as they achieve the limbic sensitization of heteromodal association areas of neocortex to motivate cognitive appraisal of adaptive concerns. Given the relatively undifferentiated architecture of the limbic cortex, and the elementary influence from hypothalamic and other subcortical regulation of this cortex, we must assume the limbic priors are less complex than the representations in heteromodal cortex, and have properties more reflective of implicit control (values, significance) than explicit representation (concepts, propositions). At the same time, the ventral limbic areas appear particularly important to the inhibitory synchronization of cortical down states, apparently balancing excitability in ways that are important to stabilization of neocortical excitement in order to form lasting memories. Further insight into these mechanisms may clarify not only the nature of affectively-charged Bayesian priors in the unconscious inferences of everyday cognition, but also the adaptive binding of personally significant representations in the ongoing memory consolidation of each night's sleep.

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