

# Multiscale contextual emergence of neural dynamics, cognition, and action

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## Abstract

There is growing evidence that brain processes involve multiscale overlapping networks and that the mapping between such neural processes and cognitive functions is many-to-many. So, the answer to the question what spatiotemporal scales in the brain are most relevant for cognition, action, experience, etc., is that several inextricably interconnected and integrated scales are relevant. There is also growing evidence that brains and embodied agents (people) are part of “larger” distributed “bio-psycho-social networks.” One cannot fully appreciate what brains do and how they work in isolation from these larger multiscale, multi-level, and multi-faceted “4E” networks (embodied, embedded, extended, and enactive). Nor can one explain human experience, cognition, or action without such an understanding. Establishing these claims is the purpose of this paper. Section 2 will unpack the claim that the brain itself is best viewed as several multiscale, dynamical, multifunctional, coordinated, and fully integrated overlapping networks. Furthermore, such individual brain networks and conscious cognitive agents are embedded in “larger” “4E” dynamical networks. Section 3 argues that the best characterization of such 4E networks is not in terms of mechanistic reduction or modularity, but contextual emergence. Section 4 will draw key connections between contextual emergence and the related work of other philosophers and neuroscientists. Lastly, Section 4 will conclude that conscious cognitive agents are reasonably conceived of as highly non-decomposable “4E” contextually emergent multiscale dynamical systems. In short, people are not brains and brains are not computers.

## 1. Introduction

There is growing evidence that brain processes involve multiscale overlapping networks and that the mapping between such neural processes and cognitive functions is many-to-many. So, the answer to the question what spatiotemporal scales in the brain are most relevant for cognition, action, experience, etc., is that several inextricably interconnected and integrated scales are relevant. There is also growing evidence that brains and embodied agents (people) are part of “larger” distributed “bio-psycho-social networks.” One cannot fully appreciate what brains do and how they work in isolation from these larger multiscale, multi-level, and multi-faceted “4E” networks (embodied, embedded, extended, and enactive). Nor can one explain human experience, cognition, or action without such an understanding. Establishing these claims is the purpose of this paper.

## 2. The multiscale dynamical brain

There is growing evidence from across cognitive neuroscience about the way brains work (Pessoa, 2022; Parker, 2022; Thiebaut de Schotten & Forkel, 2022; Axer & Amunts, 2022; van den Heuvel et. al, 2019). The brain is best viewed not in old school computational or modular terms, but in terms of overlapping, spatiotemporally widely distributed, and functionally integrated multiscale dynamical networks. What makes such brain networks irreducibly multiscale will be spelled out in detail in the next section, but the basic point is that the determination relations between elements and processes at various scales are not strictly “bottom-up” or “top-down.” Such multiscale mutual determination relations are interdependent. This is sometimes characterized in terms of “circular causation” (Juarrero, 1999; Thompson, 2007; Witherington, 2011). Such processes involve mutually

inextricable, overlapping, and interconnected local-to-global and global-to-local causal and constraint-based relationships. As we will see below and in Section 3, such mutual interscale determination relations can involve many different types of causal relations, various types of dynamical or acausal global constraints, and other types of constraints as well.

In such networks the “function” and behavior of any given elements such as neurons, glia cells, neurotransmitters, hormones, neural circuits, brain regions, connectomes, etc., is determined dynamically by multiscale relationships with other elements throughout the brain and contextually given constraints such as cognitive tasks being undertaken, bodily activity, and environmental interactions. This implies that the activities at larger scales in the brain are in no way reducible to the behavior of elements at smaller scales, but also that the behavior of elements at smaller scales are in part mutually determined by activities at larger scales. Again, the determination relations in question include a variety of causal relationships, functional relationships, and various constraint-based relations, all of which are context sensitive and interdependent (Silberstein, 2021, 2022; Bishop et al., 2022).

For all elements of such brain networks including the overlapping networks themselves, the relationship between structure and function is many-to-many. Such constantly morphing overlapping dynamical networks work simultaneously and over time to subserve many different cognitive, conscious, and bodily activities all at once. Thus, such networks fail to be explicable in terms of localization, decomposition, and computational modularity (Silberstein, 2022; Pessoa, 2022; Bishop et al., 2022; Silberstein, 2021, 2016; Silberstein & Chemero, 2013). As Pessoa notes, the implication is that phenomenal states, perception, cognition, action, and emotion are structurally and functionally closely interrelated and highly distributed processes. I would go further and say that the often-assumed dichotomy between cognition and intentionality on the one side and phenomenal consciousness on the other, is a non-starter in most cases (Silberstein & Chemero 2012). As we will see shortly, all this real-world multiscale, multiple pathways, multi-realizability relating structure and function, strongly suggests such brain networks form a self-organizing goal directed unity at the top of which is a conscious volitional agent, all of which in turn constrains the behavior of multiscale processes in the

brain to those ends. Finally, neural processes are multiscale in the sense that many elements at various scales within the brain are involved in the brain’s cognitive functions, not just neurons and glia cells. All the preceding is what I mean by the claim that neural and cognitive processes are irreducibly and inherently multiscale.

Taken together sections 2.1-2.5 will show that neural and cognitive processes are irreducibly multiscale and are not explicable in principle via localization and decomposition in the brain.

### ***2.1 Multiscale interactions among components involved in neural processes***

The brain’s structure and functions are inherently multiscale. Molecular neuroscience, systems neuroscience, and computational neuroscience can and do use a variety of different tools to explore all kinds of activity at multiple interacting scales. These tools include molecular techniques and dynamical systems theory, as well as different neuroimaging modalities, and computer simulations involving network or graphical models (Pessoa 2022). All the tools together are used to create mechanistic (structural) and statistical (functional) models, as well as other formal models. One goal is to relate more deeply structural and functional analyses, as they both enlighten and constrain the exploration of each other.

Suppose we start with a single neuron and a single neurotransmitter system such as serotonin. One cannot properly model ionic currents without bringing in the background condition of the inherently larger-scale property of cell voltage. In turn, the biophysical properties of neurons affect the propagation of electrical currents (action potentials). Modeling this most basic process in the brain already requires interactions among the scales of molecular, subcellular, cellular, and neural circuits. Action potentials cause the release of neurotransmitters to their postsynaptic target. Neurotransmitters are varying chemicals that travel across neurons and bind to specific receptor sites, depending on the type of neurotransmitter.

Accordingly, the neurotransmitters in turn affect the behavior of the cells with which they bind. Action potentials and neurotransmitters regulate one another in all kinds of complex ways. Stimulation of excitatory

receptors by neurotransmitter binding causes depolarization of the postsynaptic plasma membrane, promoting generation of an action potential. Conversely, stimulation of inhibitory receptors causes hyperpolarization of the postsynaptic membrane, repressing generation of an action potential. While we often think of neural signaling via action potentials as being strictly digital (all-or-nothing) and being the central mode of information transmission, there is now evidence that things are much more complicated than that in both respects. For instance, subthreshold changes in presynaptic membrane potential before triggering the spike also determines spike-evoked release of neurotransmitter. The changes in presynaptic voltage that regulate spike-evoked release of neurotransmitter (through the modulation of biophysical state of voltage-gated potassium, calcium, and sodium channels in the presynaptic compartment) are themselves analog (Zbili et al., 2016).

Even at the molecular scale, neurons and neurotransmitters are not anywhere near the whole story. For example, regulatory RNA is generated at the synapses and in turn controls the production of various proteins at these sites. The latter proteins are gene-regulatory proteins that can travel to cell nuclei and affect gene expression therein. Thus, the local activity at different synapses can have a profound effect on the long-term genetic behavior of the neurons involved. Some of the regulatory RNA produced by neurons is packaged in membrane-bound vesicles called exosomes, which can carry information to distant cells, affecting their function and behavior. For instance, traveling from a postsynaptic neuron to presynaptic ones, exosomes can strengthen and enhance connections related to learning. Exosomes communicate with other neurons in brain and glia (or “neuroglia”) cells, helping to activate and inhibit both, as well as helping to synchronize gene expression in nearby neurons (Parrington, 2021).

Glial cells—once thought to be nothing but support structures and maintenance for neurons—do not generate action potentials. However, they do communicate chemically with many of the same surface receptors as neurons, as well as other glial cells (Parrington, 2021). Glia, such as astrocytes, also aid in the formation of synapses. Microglia both help defend against disease as well as help pruning neural connections and are involved in memory formation by activating NMDA receptors related to long-term potentiation via amino-acid D-

serine, thereby changing synaptic structure and gene expression, all of which in turn involves regulatory RNA networks.

It seems that everything in the brain involves interdependent multiscale interactions that dictate behavior and function. This fact helps explain the many-to-many relationship between structure and function in the brain. Oxytocin for example is not a dedicated “love hormone.” Its presence is neither necessary nor sufficient for pair-bonding, displays of affection, etc. As Carter notes, Oxytocin both regulates and is regulated by components of the immune system including glia. The effects of Oxytocin are hierarchical and context dependent. For example, Oxytocin is known to have paradoxical effects on people who are extremely stressed or traumatized (Carter, 2022). Nor is Oxytocin necessary for certain kinds of behavior. As Manoli puts it, “because of evolution, the parts of the brain and the circuitry that are responsible for pair-bond-formation don't rely only on oxytocin” (Manoli 2023, as cited in Hamilton, 2023). New work involving CRISPR mutagenesis where the effects of oxytocin were effectively knocked out in prairie vole pups and their parents, showed no disruption to their normal social behavior or pair-bonding interactions (Berendzen et al, 2023). Manoli says, “*the result makes sense because pair bonding is essential to a prairie vole's survival. And evolution tends to favor redundant systems for critical behaviors. There's not a single pathway. But rather, these complex behaviors have really complicated genetics and complicated neural mechanisms*” (Manoli, 2023, as cited in Hamilton, 2023). All of this goes for other neurotransmitters as well. Pessoa makes exactly the same point regarding the often-repeated false claim that dopamine is a “reward molecule.” Not only is dopamine not a reward molecule, but as Pessoa (2022) puts it, “...*there is no such thing as a ‘reward molecule’—the message is not in the molecule*”. All of this illustrates that what neurotransmitters, hormones, etc., do and what happens in their absence, is often a function of various other multiscale contextual states of affair.

One can give many more such examples. Take the strategy of sleep (with all its attending phenomenology in dreams or lack thereof in stage 4 sleep), for instance. While experts still debate the various possible functions of sleep, it is ubiquitous across species. There is evidence now that sleep even predates animals with brains and central nervous systems; witness Hydra (Kanaya et al.,

2020). Sleep-like states are conserved across most species but involve different mechanisms (Hayashi & Lui 2017). For instance, hypocretin has a crucial role in maintaining waking states in mammals. Invertebrates have no homologue while in fruit fly's neuropeptide pigment dispersing factor is critical in regulating circadian rhythms and wakefulness (Hayashi & Lui 2017). Different neural circuits, neurochemicals, hormones, and genes are engaged in sleep regulation in different animals. *There is simply no universal mechanism or molecular pathway that encodes sleepiness or wakefulness.* This is one reason why it seems absurd to seek a neural-identity theory in such cases, and why it seems wrong to put all the explanatory onus on the molecular mechanisms. Macroscopic evolutionary processes and environments are clearly a big part of the explanation here. Such advantageous and adaptive functions often remain the same across species and environments even when the molecular or structural details change. In short, "The fundamental forces at work in evolution are independent of which molecules interact with which and how" (Kershenbaum, 2020).

This is of course the kind of point that functionalism was founded on. Mechanism hunting in the brain while essential for cognitive neuroscience, tends to make us blind to functionalism's point about multiple-realizability and larger contexts and constraints such as evolutionary ones. Indeed, all such mechanisms are *highly historically contingent*. For example, there are evolutionarily much earlier Oxytocin-like molecules that worked similarly on both vertebrates and invertebrates, some of which evolved in a completely convergent manner with no shared evolutionary history or causal connection (Carter, 2022). As Carter notes, "many other biologically active molecules, including common neurotransmitters and variations on the CRH molecule existed prior to the Pre-Cambrian explosion. In that context the evolution of the specific molecule known as oxytocin is comparatively recent. Oxytocin's functions also are intertwined at many levels with the ancient immune system" (Carter, 2022). Sometimes what matters most is not the mechanism but the function it subsumes. This seems to be especially true in brains which defy any simplistic mapping between brain regions and cognitive functions. As Salehi et al. (2020) notes based on their meta-analysis of fMRI data, "*there is not a single functional parcellation atlas but rather that the flexible brain reconfigures these functional parcels depending upon what it is doing*". For example, Fedorenko & Blank

(2020) note in their paper: "*Broca's Area Is Not a Natural Kind*" that Broca's area, once thought to be where the speech articulation mechanism was localized, contributes causally to many cognitive functions, making it "structurally and functionally heterogeneous."

Continuing to move up and away from the molecular and cellular scales, highly coordinated and oscillating neural synchrony affects the action potentials of individual neurons, which in turn affects the overall charge of cells and their background activity. Just as with small-world and rich-club networks, many different insects and animals have similar endogenous synchronous global brain wave activity associated with sleep, attention, and other processes existing at many different scales. This is thought to be yet another method for coupling and communication in the brain. For instance, there is evidence that primates use beta waves to consciously switch between different pieces of information, while gamma waves are associated with, among other things, retrieval of different pieces of information. Beta waves are thought to act like signals that open access to working memory (Ahmadi et al., 2021).

On a more global scale, all this activity generates electrical fields in the brain which can affect spike timing dependence and synchronization of neural activity. Thus, the electrical fields are generated by neural activity, while these fields alter that activity in turn, such as ionic flux across cell borders (Godfrey-Smith, 2020; Parrington, 2021; Yoshimi & Vinson 2015; Cacha & Poznanski, 2014; Gurwistch, 1964).

## 2.2 Topological constraints in multiscale brain networks

In every area of cognitive neuroscience including learning, memory, decision making, action, empathy, mind reading, the contents of phenomenal states, etc., the central theoretical unit of study and analysis is the multiscale network (Shine et al., 2019; Bertolero & Bassett, 2020; Schirner et al., 2018; Jansson, 2020). Whether we are talking about "task neutral" networks such as the Default Mode Network (DMN) and the Salience Network (SN), or task positive networks such as Executive Control Network (ECN), Dorsal Attention Network (DAT), Frontoparietal Network (FPN), Amygdala Network (AN), Action-Perception Network (APN), Empathy-Network (EN), etc., the idea is that various functional multiscale brain networks are the key



unit of investigation (Huang et al., 2020; Scheinin, 2020).

The explanations involving these networks for various cognitive abilities and various states of phenomenal consciousness, all have to do with multiscale contextually driven changes such as the presence or absence of anesthesia, psychedelics, deep sleep, stress, specific cognitive tasks, social interactions, etc. There is a reason that in [Feldman Barrett's \(2020\) \*Seven and a Half Lessons About the Brain\*](#), the second lesson is that your brain is a network.

As will be made clear going forward, the reader should not get the idea that there is a one-to-one relationship between certain networks and certain cognitive functions or phenomenological states. As [Thompson \(2020\)](#) notes regarding meditation for instance, *“it's empirically unwarranted to map the cognitive functions involved in meditation practices in general, and mindfulness meditation in particular, onto particular brain areas or networks”*. More generally, [Pessoa \(2022\)](#) wrote an entire book dedicated to showing that even widely distributed and multiscale brain networks don't have a one-to-one mapping with various cognitive functions, whether at a time or across time. As he puts it, *“subdividing the brain into discrete and separate networks still seems too constraining...An alternative is to consider networks as inherently overlapping”*. Such networks and their elements are constantly in flux and “softly assembled”, changing rapidly as cognitive tasks and situations require. Thus, even multiscale networks themselves are not modular, hence the expression “overlapping.”

Let us now say more about large-scale topological constraints in such multiscale brain networks (see [Sporns, 2011; Silberstein & Chemero, 2013; Silberstein, 2021, 2022; Pessoa, 2022](#) for more details). Network analyzes of the brain are based on the thought that brain function is not just relegated to individual regions and connections but emerges instead from the topology of the brain's entire global network, such as the connectome of the brain-as-a-whole. In such graphical models of neural activity, the basic units of explanation are not neurons, cell groups, or brain regions as strictly structural local mechanisms, but multiscale networks and their large-scale, often distributed, and nonlocal connections or interactions ([Silberstein & Chemero, 2013; Silberstein, 2021](#)). The study of this integrative brain function and

connectivity is mostly based in topological or architectural features of brain networks.

Thus, one essential feature of network analysis is to illuminate the topological structures of brain networks such as small-world networks and rich-club networks which seem to appear over and over in the brain and elsewhere. Such networks are ubiquitous across species, come online early in the development of individual organisms, and are instantiated by very diverse structural/molecular components and interactions ([Parrington, 2021](#)). Thus, such networks have a kind of topological “universality.” What accounts for the explanatory power and autonomy of such networks is the topology itself, regardless of which molecules induce changes in those networks ([Sporns, 2011; Silberstein & Chemero, 2013](#)). This again is an instance of real world multiple realizability at various scales ([Silberstein & Chemero, 2013; Silberstein, 2021](#)).

We can use tools from network neuroscience and graph theory to model both structural networks (the various elements and their inter-relations at multiple scales) and functional networks (the various types of ‘causally relevant’ statistical dependencies or correlations that exist between different distant regions of the brain, indicating that even if not structurally connected, they participate in the same cognitive functions in some important way). (See [Silberstein & Chemero, 2013; Bassett & Sporns, 2017; Silberstein, 2021; Pessoa, 2022](#) for more details). Again, what we infer from such analysis is that multiply realized functional networks are partially insensitive to, decoupled from, and have a one-to-many relationship with respect to lower-level neurochemical and structural “wiring” details.

More specifically, a graph in this case is a mathematical representation of some actual many-bodied biological system such as the brain. The nodes in such models can represent neurons, cell populations, brain regions, etc., and the edges represent connections between the nodes. The edges can represent structural features such as synaptic pathways and other wiring-diagram-type features, or they can represent more topological/functional features such as graphical distance, functionally significant statistical correlations between activities across the brain, and network types. What matters in such graphical explanations is the topology or pattern of connections. Different geometries or arrangements of nodes and edges can instantiate the same

topology (see Silberstein & Chemero, 2013; Silberstein, 2021, 2022).

When mapping the interactions (the edges) between the local neighborhood networks, we are interested in global topological features, i.e., the topological architecture of the brain-as-a-whole. While there are local networks within networks, it is the global connection between these that is often of greatest interest in systems neuroscience. Graph theory enables the description of many different kinds of network topologies, but one of great interest to systems neuroscience are small-world networks. This is because it was thought that various regions of the brain and the brain-as-a-whole instantiated such networks. The key topological properties of small-world networks are:

1. Sparseness: relatively few edges given the large number of vertices;
2. Clustering: edges of the graph tend to form knots, for example, if X and Y know Z, there is a higher-than-normal chance they know each other;
3. Small diameter: the length of the most direct route between the most distant vertices, for example, a complete graph, with  $n^2/2$  edges, has a diameter of 1, since you can get from any vertex to any other in a single step. Most nodes are not neighbors of one another yet can be reached through a short sequence of steps.

That is, (1) there is a much higher clustering coefficient relative to random networks with equal numbers of nodes and edges and (2) short topological path length. Small-world networks thus exhibit a high degree of topological modularity and nonlocal or long-range connectivity. There are many different types of small-world networks and other types of networks with unique topological properties that allow researchers to make predictions about the robustness, plasticity, functionality, health, etc., of brains that instantiate these networks (Sporns, 2011; Silberstein, 2014).

Regarding especially the large-scale topological structure of the brain, another related type of network of particular interest is called the “Rich-Club” network (Pedersen & Omidvarnia, 2016; van den Heuvel & Sporns, 2011). Such network architectures are called “rich-club” based on the analogy with wealthy, well-connected people in society. Such networks are even more interconnected than small-world networks. “Members” of this club constitute a few “rich” brain-regions or central “hubs” that distribute many of the

brain’s global neural communications. The “Rich-Club” topological brain architecture is instantiated when the hubs of a network tend to be more densely connected among themselves than nodes of a lower degree. As Pessoa (2022) puts it:

*“It turns out that the brain is more interconnected than would be necessary for it to be a small world. That is to say, there are more pathways interconnecting regions than the minimum needed to attain efficient communicability. So, while it is true that local connectivity predominates within the cortex, there are enough medium-and long-range connections—in fact, more than the ‘minimum’ required—for information to spread around remarkably well...In sum, the theoretical insights of network scientists about ‘small worlds’ demonstrated that signals can influence distal elements of a system even when physical connections are fairly sparse. But cerebral pathways vastly exceed what it takes to be a small world. Instead, what we find is a ‘tiny world’ (Pessoa, 2022).*

The dynamical interactions in such networks are recurrent, recursive, and reentrant. Therefore, the arrow of explanation or determination in such systems is both ‘top-down’ (graphical to structural) and ‘bottom-up’ (structural to graphical). Global topological features of complex systems are not explicable in principle via localization and decomposition. The many-to-one relationship between the structural and the graphical features demonstrates that specific structural features are neither necessary nor sufficient for determining global topological features. So again, topological features such as the properties of small-world networks exhibit a kind of “universality” with respect to lower-level structural details.

In the case of random networks for example, power laws and other scale-invariant relations can be found. These laws, which by definition transcend scale, help to predict and explain the behavior and future time evolution of the global state of the brain, irrespective of its structural implementation. Power laws are explanatory and unifying because they show why the macroscopic dynamics and topological features exist across heterogeneous structural implementations (Silberstein & Chemero, 2013).

We can model brain networks at various spatial and temporal scales often called “microscale, mesoscale, and

macroscale.” Such brain-wide networks often help harness, recruit, integrate, and unify all these scales and their components in the service of various cognitive functions and to subserve contents of conscious experience. As we discussed, the multiscale components involved in such networks often include complex interactions between the following: ionic flux, sub-cellular structures, proteins, genes, RNA, neurons and neural assemblies, glial cells, neurotransmitters and neuromodulators, hormones, large-scale neural synchrony and neural oscillations, electrical fields, etc. (Parrington, 2021). It is thus a mistake to focus only on networks involving neurons, their action potentials, and oscillations. Complex networks often involve brain-wide integration at every scale. The bottom line is that these *global purely graphical constraints* range over multiply interacting structural scales and are not themselves explained in terms of localization and decomposition.

### 2.3 Essential “design” features of multiscale brain networks

In addition to their graphical features, multiscale brain networks are also constrained by global design principles such as plasticity, reuse, redundancy, and degeneracy. Let us define these global features of multiscale topological networks— (i) *Neural plasticity*: Generally defined as changes in the structure, activity, or function of the brain on some scale relative to some change in context such as injury, stroke, or simply learning. An example would be synaptic plasticity, where experience, learning, and memory formation change the synaptic connections in the brain; cross-modal plasticity, where, for instance, the loss of one sensory modality inducing cortical reorganization that leads to enhanced sensory performance in remaining modalities such as the relocation and transfer of somatosensory and auditory functions to the former visual cortex; intramodal plasticity (plasticity within a modality) such as the expansion of cortical maps to neighboring regions of intact cortex that have been deprived of sensory input from within the same modality as supported by the expanding cortex; and supramodal plasticity (not unlike cross-modal plasticity but need not involve injury, sensory deprivation, or special training) such as occipital cortices not only serving as a basis for non-visual information processing, but also contributing something inherently visual to the non-visual input, as in “*non-visual input is being processed visually*” (Zerilli, 2021).

As Zerilli (2021) notes, “*plasticity is an intrinsic and persistent property of the nervous system*” at all scales in the brain, including not only the aforementioned cortical map reorganization, but neurotransmitters, neuromodulators, cellular changes caused by learning and memory consolidation, neuromorphology, neurogenesis, among others. Perhaps the most well-known example of plasticity is sensory substitution, such as converting visual images into soundscapes via a “*visual-auditory sensory substitution device*”. But there are many other examples. For instance, after a unilateral lesion to the pre-frontal cortex, areas in the opposite hemisphere dynamically change their activity to compensate (Lau, 2022). Neural plasticity is joined by a host of other types of plasticity in biology such as phenotypic plasticity, all of which helps explain the robustness and autonomy of complex biological systems in general.

(ii) *Neural reuse or recontextualization*: Each region of the brain ends up participating in many different functional coalitions over time/at a time. In other words, the same neural circuits can end up contributing to different tasks or functions, depending on context (Anderson, 2010, 2014; Zerilli, 2021).

(iii) *Neural redundancy*: Numerically distinct brain regions have the same structure and function. These regions become active due to changes in context such as injury or stroke.

(iv) *Neural degeneracy*: Different neural structures and mechanisms perform the same function, depending on specific contextual features that change over time. As Feldman Barrett (2020) puts it: “*Degeneracy in the brain means that your actions and experiences can be created in multiple ways. Each time you feel afraid, for example, your brain may construct that feeling with various sets of neurons.*”

These global “design” features of the brain in turn enable the robustness and autonomy of the brain-as-a-whole. Robustness (invariance in the face of environmental and *contextual* changes), and autonomy (adaptability and flexibility in the face of environmental and *contextual* changes) are the norm in complex biological systems (Silberstein, 2016, 2021), brains are no exception. As Bateson & Gluckman (2011) put it, “*The central elements underlying many forms of plasticity are epigenetic processes, and plasticity operating at different levels of organization often represents different descriptions of the same process. Underlying behavioral*

*plasticity is neural plasticity and underlying that is the molecular plasticity involving epigenetic mechanisms.”* There are many different forms of robustness and plasticity, such as developmental, phenotypic, a variety of neural, behavioral, immunological, etc., all of which can be found at work in the brain at various scales.

All these global constraints in the brain help explain the interdependent behavior and function of multiscale structural elements, all of which in turn helps explain the highly adaptive and environmentally responsive nature of brains. As we will discuss in Section 3, many of the aforementioned contextually given constraints are not causal relations either in the sense of production, mechanisms, or say Granger causality, but they are rather global constraints. As [Barack et al. \(2022\)](#) note, the term “causality” is used in many ways in neuroscience and different types of explanation often invoke different notions of causation. The mechanism hunting of the new mechanists mostly assumes the “causal dependence” model as characterized by [Woodward \(2003\)](#) in terms of intervention or manipulation. The focus here is on some “knob” we can tweak that makes a difference we are seeking.

#### **2.4 The dynamical brain at work**

It is important to stress the dynamical nature of multiscale brain networks. It is a misnomer to think of the brain as having static graphical properties and a fixed single scale connectome or “wiring diagram.” Take the following for instance:

*“Mastering the empirical, theoretical, and computational challenges for bridging the different spatial (and temporal) scales will open new perspectives for a deeper understanding of the connectome and its impact on brain function and disease. It is an intriguing concept to approach the connectome not only as a multiscale system in which each scale (e.g., neurons, microcircuits, and networks) has distinct features, but also as a system that has repetitive properties”* ([Axer & Amunts, 2022](#)).

The dynamics of brain networks as-a-whole and the behavior of their multiscale components can change rapidly as a function of new action-related needs, environmental constraints, changing cognitive tasks, etc. These networks must manage, modulate, and coordinate processes that are happening at very different spatial and temporal scales. There are many different heterogeneous

time scales in the brain ranging from milliseconds to seconds to minutes and beyond. This sort of multiscale integration is what multiscale networks do ([Bassett & Sporns, 2017](#); [Silberstein, 2021](#)).

Network analysis links structure and function, showing us very complex correlations and various ‘causal’ and other relationships that can exist between them. These highly context dependent relationships generally don’t look anything like the neo-mechanist’s localization and decomposition of cognitive function or their hierarchical, synchronic picture of how elements at different scales are related ([Bassett & Sporns, 2017](#); [Kaiser, 2020](#); [Silberstein, 2021](#)).

Graph theory and the big data tricks of network neuroscience, such as network simulations, time series analysis, various sorts of causal analysis such as Granger causality, etc., dimension reduction and universality class analysis, are perfect for illuminating these multiscale relationships and connections. That is, *“Networks can also bridge across data of very different types and from different domains of biology. One example is the joint investigation of gene co-expression patterns and patterns of brain connectivity. These studies raise important questions about the nature of the mechanisms that tie the topology of structural and functional brain networks to fundamental aspects of basic brain physiology”* ([Bassett & Sporns, 2017](#)).

As noted, multiscale brain networks have plasticity, reuse, redundancy and degeneracy built into them, thus enabling robustness and autonomy. All of this makes perfect sense evolutionarily speaking. As [Sporns \(2011\)](#) says, *“the same set of network elements can participate in multiple cognitive functions by rapid reconfigurations of network links or functional connections.”* [Anderson \(2014\)](#) describes such ever-changing coalitions of structural networks subserving overlapping functional networks as “TALoNs”.

#### **2.5 The “4E” nature of multiscale brain networks**

So far, we’ve been treating the brain in isolation from the body and environment, but this is a mistake. The brain and its networks discussed herein are unquestionably embedded in larger 4E ‘bio-psycho-social’ networks. While people use this expression in different ways, I mean the claim that biological, psychological, and social causal factors and constraints co-determine each other



just as processes at different scales and “levels” do within the brain. One cannot fully appreciate what brains do and how they work in isolation from these larger multiscale, multi-level, and multi-faceted “4E” networks (embodied, embedded, extended, and enactive).

Historically, many different traditions of thought and many different thinkers feed into and are the foundation for the 4E movement (Chemero & Silberstein, 2008; Chemero, 2009; Newen et al., 2018). Furthermore, there are many different brands and varying strengths of 4E cognition. The basic idea is that cognition and action essentially involve not just brains, but bodies and peripheral nervous systems. Whether we are talking about people or octopuses, part of the explanation for an organism’s cognitive capacities is about the nature of their embodiment, sensory conductors, and subsequent movement and exploration of their environment (embodied). Embodied brains are crucially embedded in physical and social environments, generally implying some strong causal dependence or “scaffolding” on the environment of the cognitive system (embedded). “Extended” suggests that going beyond “scaffolding”, cognitive systems are constituted by environmental contexts to include tools, technology, and other aspects of culture (Extended).

The word “enactive” implies that the cognitive system is partly constituted by “active engagement in the agent’s environment” (Silberstein & Chemero, 2012; Newen et al., 2018). Such engagement is sometimes called perception-action cycles. The focus here is to see cognition as driven by and partly constituted by action. The notion of “environment” here means not just the organism’s physical or social environment as defined from a third-person perspective, but their “phenomenological niche” or Umwelt with its first-person affordances (Silberstein and Chemero 2012). Cognitive agents “enact a world” on this view, they don’t represent it passively (Varela et al., 1991). So, according to enactivism, cognition is “embodied sense-making”: an enactment or bringing forth of a lived world of meaning and relevance through embodied action.

Enactivism aside, more and more people are willing to accept something like the focus on cognition as action driven and something like the 4E take on brains and cognition. In the words of Feldman Barrett (2020), “*little brains wire themselves to their world. Little brains*

*typically require a social world to develop normally. For example, certain physical inputs, such as photons of light bombarding their retinas, must be provided or the brain won’t develop normal vision.*” Other examples of essential connections that she presents include the following: the role of caregivers in the social world for tuning and pruning the brain in a newborn’s neural development, guided attention, various stable features of the fixed environment, niches, training the senses, exposure to natural language, many different social inputs such as love and affection, education, and various socioeconomic conditions. Feldman Barrett (2020) notes, “*Our three examples of tuning and pruning demonstrate how the social world profoundly shapes the physical reality of the brain’s wiring.*”

Working up to social networks proper, Bassett and Sporns (2017) note that, “*Large-scale studies of brain-behavior relations and behavior-behavior dependencies, although still in their infancy, promise to provide a rich database for mapping the relations among brain processes and their contributions to perception, action and cognition.*” Such network analysis obviously includes correlates that fall under the category of embodied, embedded, enactive, and extended cognition such as action-perception cycles. Bassett & Sporns (2017) go on to say, “*network neuroscience asks how all these levels of inquiry help us to understand the interactions between social beings that give rise to ecologies, economies, and cultures. Rather than reducing systems to a list of parts defined at a particular scale, network neuroscience embraces the complexity of the interactions between the parts and acknowledges the dependence of phenomena across scales.*” As we will see in the next section, they are describing contextual emergence.

Feldman Barrett (2020) fifth lesson is that “*Your brain secretly works with other brains.*” For example, there is recent evidence for inter-brain wave neural phase synchronization even in people interacting only via online cooperative gaming, with no face-to-face interaction whatsoever. The data suggests that such synchrony involving alpha and gamma frequency bands is highly correlated with better performance in the game (Wikstrom et al., 2022). There is an entire discipline called *Social Neuroscience* devoted to exploring and explaining such cases (Silberstein, 2022) which we will discuss shortly.

The point is, the fact that we are a social species does not stop at early brain development or even at childhood. As [Feldman Barrett \(2020\)](#) puts it, “ultimately, your family, friends, neighbors, and even strangers contribute to your brain’s structure and function and help keep your brain humming along.” Our social interactions with others co-regulate and synchronize a number of biological and cognitive processes such as breathing, brain waves, motion and bodily movements generally, heart rate, circadian cycles, menstrual cycles, linguistic capacities, learning, etc. ([Spivey, 2020](#)).

In the widely used textbook *Introduction to Social Neuroscience* by [Stephanie & Cacioppo \(2020\)](#), they note that this relatively nascent field of social neuroscience is based on the following assumptions:

First, “human brains are not regarded as isolated computational devices, but like a device networked with other brains and people both physically and socially”.

Second, “evolutionarily speaking, there are conserved neural, hormonal, cellular, and molecular mechanisms involved in social behavior”.

Third, “social connectedness, social complexity and social/cultural learning are some of the driving forces behind the evolution of the human brain”.

Fourth, “brains and their evolution underly social processes, but the reverse is true as well. This can be seen on both evolutionary and developmental time scales”.

Fifth, “the social brain hypothesis. Larger and more complex brains enabled more social interaction and vice-versa. As culture developed many more complex problems were solved by social groups, all leading to positive feedback in the direction of ever increasing neural and cultural complexity”.

Sixth, “the focus is on connection and coordination, e.g., inherently social functions such as communications, social perception, recognition, imitation, empathy, competition, cooperation, etc. For example, in *Social Neuroscience* language is not viewed primarily as an information processing medium, but as a means of communication”.

Seventh, “multiple interacting scales and levels of organization from genes on up connect brains and social interactions, such that there are multiple multiscale avenues of mutual-determination and multiple multiscale interacting causal factors. There is “reciprocal determinism” from social-to-biological and vice-versa at

*multiple scales*. Such bio-psycho-social dynamical systems are highly complex, often non-linear and “interaction dominant” ([Spivey, 2020](#)). Examples include the growing evidence that social environment can modulate gene expression, the severe effects of social isolation and loneliness on neurological, cognitive, and genetic processes, and that the effects of pharmacological interventions such as stimulants or even placebos is partly a function of social hierarchy and other social factors ([Cacioppo & Cacioppo, 2020](#)).

Social neuroscience then is an entire discipline devoted to studying the way neural and social processes contextually constrain and enable one another to emerge, over both evolutionary and developmental time scales. The import of all this should be clear. Very highly regarded hardnosed, mechanistically minded neuroscientists at the top of their profession think that we can now regard it as well confirmed neuroscientific commonsense that contextual emergence is the right way to think about the relationship between brains and their physical and social environments. All this evidence of course dovetails with a growing body of evidence from epigenetics and epigenomics more generally ([Silberstein, 2021](#)).

I hope all of this is sufficient to persuade the reader that my opening remarks about the nature of how brains work were no exaggeration. Researchers often discuss multiscale feedback and feedforward loops in the brain, multiscale non-linear interactions, etc. But such organizational features only scratch the surface when it comes to just how functionally integrated a unity the brain really is. When it comes to determination relations in the brain, it seems they are often symmetric, multiscale, multi-directional, and multifaceted. In the next section we’ll see that such interactions are best described by contextual emergence.

### 3. Contextual emergence

I have argued that neural and cognitive processes are inherently multiscale systems and not amenable to mechanistic reduction such as localization and decomposition. Therefore, we need an ontic and explanatory framework that fits the facts. Contextual emergence is such a framework ([Bishop & Atmanspacher, 2006](#); [Atmanspacher & beim Graben, 2007](#)).

Historically the relationship between biology and the ever-evolving concept of emergence is a complex one. Historically, the biological sciences have played a very important role in discussions of emergence. [Pigliucci \(2014\)](#) has argued that one can understand the history of biology as a struggle between reductionism and holism. The reductive-mechanistic side of biology is obvious but as [Pigliucci \(2014\)](#) notes, “*there is a long holistic tradition that focuses on the complexity of developmental systems, on the non-linearity of gene–environment interactions, and on multilevel selective processes to argue that the full story of biology is a bit more complicated than that.*” It is true that the successes of genetics and molecular biology over the past decades might appear to fuel the reductionist intuitions of many biologists. But this is far from the whole story, [Pigliucci \(2014\)](#) maintains: “*Holism has built on the development of entirely new disciplines and conceptual frameworks over the past few decades, including evo-devo [evolutionary development] and phenotypic plasticity.*” He further observes that “*a number of biologists are still actively looking for a way out of the reductionism–holism counterposition, often mentioning the word ‘emergence’ as a way to deal with the conundrum.*”

Given the many meanings and uses of the word “emergence” and the various baggage the concept has accrued over the years, it is important for the reader to understand why contextual emergence is different from the standard notions in the literature.

While there are by now many, many different definitions of emergence in the scientific and philosophical literature, they tend to reduce to either “weak emergence” or “strong emergence.” If ontological reductionism is true, then epistemological or weak versions of emergence are the only kinds possible. For instance, [Chalmers \(2006\)](#) defines weak emergence as follows:

*“To capture this, one might suggest that weak emergence is the phenomenon wherein [non-obvious] complex, interesting high-level function is produced as a result of combining simple low-level mechanisms in simple ways...This conclusion captures the feeling that weak emergence is a ‘something for nothing’ phenomenon. The game of Life and connectionist networks are clear cases: interesting high-level behavior emerges as the consequence of simple dynamic rules for low-level cell dynamics.”*

[Chalmers \(2006\)](#) talks about weak emergence<sup>1</sup> in terms of “*ease of understanding one level in terms of another. Emergent properties are usually properties that are more easily understood in their own right than in terms of properties at a lower-level.*” However, weak emergence does not seem to capture the multiscale and context sensitive processes discussed in the last section.

There are those who think that while everything emerges from and depends on fundamental physical processes, they doubt that weak emergence is sufficient to explain everything. These people thus tend to champion what is sometimes called strong or radical emergence, which historically, has many different definitions (see for example [Morgan, 1923](#); [Alexander, 1920](#); [Broad, 1925](#); [O’Connor, 1994](#); [Humphreys, 1997](#); [Silberstein, 1999](#); [Chalmers, 2006](#)). However, the basic idea is that if X is strongly emergent with respect to Y then Y does not determine X. Or, if Y does determine or cause X, it is a brute fact that has no further explanation. Thus, a strongly emergent property is one that, *in principle*, cannot be derived from, predicted from, or fully explained by some more fundamental (physical) theory because the emergence of such properties are brute facts.

Strong emergence is a strictly ontological conception of emergence that threatens a disunified picture of the world and would seem to be beyond scientific explanation. Thus, this sort of emergence, even if it is true of say phenomenal consciousness, is of little use here.

It seems to me that those neuroscientists who are coming around to the picture of the brain and cognition painted herein, are in search of some conception of emergence that is neither weak nor strong. Take the following two examples:

*“The concept behind integration comes from emergentism, which postulates that ‘the whole is something besides the properties of the isolated components.’ In neuroscience, there is a growing consensus that functions are an emerging property of the interaction between brain areas. Thus, function-specific brain activity involves the integrative effort of several brain regions”* ([Thiebaut de Schotten & Forkel, 2022](#)).

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<sup>1</sup> Bear in mind that “weak emergence” is defined differently by different people (e.g., [Bedau, 2008](#); [Huneman, 2008](#); [Wilson, 2015](#)). Some would argue that weak emergence need not be strictly epistemic, though it is often defined in terms of *in practice* failures of derivability, prediction, or computability.

*biological example of an emergent effect. We cannot explain these effects by describing individual cellular or system properties but must consider the relationships between local and global effects simultaneously”* (Parker, 2022).

I would say that the preceding passages are gesturing towards something like contextual emergence. As the preceding passages suggest and as we have seen, when it comes to brains the arrow of explanation and determination is not strictly bottom-up, not local, not decomposable, nor unidirectionally from smaller length and time scales to larger scales. What emerges in this case includes not just larger-scale phenomena such as cognitive processes, decision making, and action, but the behavior of the overlapping functional networks in the brain and the behavior of their multiscale elements. This kind of emergence is nothing like a simulation of a neural network or a finite automaton because everything emerges together in a mutually interdependent multiscale fashion.

This is what contextual emergence is all about. Contextual emergence suggests that determination relations between smaller and larger scales need not be anti-symmetric, transitive, or anti-reflexive. Yet, contextual emergence does not imply any kind of discontinuity or disunity in nature. Contextual emergence emphasizes the ontological and explanatory fundamentality of multiscale contextual constraints, often operating globally over interconnected, interdependent, and interacting entities and their relations at multiple scales. This is a unifying fact about the nature of reality.

This means new relations/interactions at multiple scales in differing contexts, naturally leads to the emergence of novel entities, properties, laws, etc. In terms of both ontology and scientific explanation, it is best to think of such a world in terms of multiscale contexts and constraints (Bishop et al., 2022). If contextual emergence has a slogan, it is that phenomena at lower levels and smaller scales provides at best a necessary condition for phenomena at higher levels and larger scales. The reverse is also true.

Contextual emergence can be summarized as follows:

1. Contextual emergence is a type of scientific explanation that emphasizes the equal fundamentality of what are often multiscale contextual co-constraints and

interdependent relations at multiple interacting scales. Such constraints are characterized by stability conditions treated as being external to the system in question.

2. Such constraints can include global or systemic constraints such as topological constraints, dimensional constraints, network or graphical constraints, order parameters, etc.

3. Such constraints can be causal-mechanical and dynamical, but they can also involve global non-causal or adynamical difference makers, such as conservation laws, free energy principles, least action principles, information theoretic principles, symmetry breaking, etc.

4. Such constraints can also include global “design” principles such as plasticity, robustness, and autonomy in complex biological systems.

5. Contextual constraints can even be behavioral, social, normative, etc.

6. Contextual constraints can be symmetric, such that X and Y can simultaneously act as contextual constraints for one another.

7. Contextual constraints represent both the screening off and opening up of new areas of modal space, i.e., degrees of freedom at multiple scales, and thereby new patterns emergence and become robust.

8. Contextual emergence provides a framework to understand two things: (A) how novel properties are produced, and (B) why those novel properties matter.

As suggested above, there are many different types of contextually given constraints. In Section 2 we saw many examples of such contextually given constraints. There will often be many such constraints operating on and across different scales and levels at the same time. The kinds of explanations involving contextual emergence often rely on stability conditions at multiple scales, such as dynamical and topological features, as well as the network of other multiscale relations involved in the context of the target system. In general, stability conditions are the contingent conditions characterizing contexts that guarantee the existence and stability of relevant systems and their states and observables over time. The key feature of stability conditions is that they are whatever ‘environmental’ or contextual features, however concrete or abstract, that we are treating as being outside the system, often at multiple scales, that together make up the full set of conditions for the emergent in question to come into being. That is, stability conditions enable emergence and robustness (Bishop et



al., 2022). As discussed in Section 2, we can also treat ‘stability conditions’ as being a part of the multiscale extended system, as with the case of bio-psycho-social networks. The boundaries of such systems are fluid, context sensitive, and depend on the explanatory task at hand (Silberstein & Chemero, 2012).

Finally, let’s compare contextual emergence with typical intuitions about emergence. In surveying the literature on emergence, four typical intuitions or “marks” of emergence are often discussed (e.g., Kim, 1999):

*Arise*: Emergents at a higher-level arise out of properties and relations characterizing the entities and properties at a lower-level.

*Unpredictable*: Emergents are unpredictable, even given exhaustive information concerning the lower-level.

*Inexplicable*: Emergents are inexplicable in terms of lower-level properties.

*Novel*: Emergents have novel features not found at the lower-levels.

Regarding *Arise*, first, contextual emergence calls into question the fundamentalist and hierarchical assumptions built into this intuition. That is, contextual emergence calls into question the existence of some fully autonomous or fully independent microscopic causally/dynamically closed basic physical process sufficient to determine all other phenomena at larger scales. With contextual emergence, emergent only arise from a “lower-level” or smaller scale provided relevant stability conditions, often found at larger scales, are present. While some necessary conditions for the emergents may exist at the “lower-level” or smaller scale; nonetheless, for contextual emergence, the sufficient conditions are represented by all the relevant stability conditions at various scales. Second, emergents can also come into being at smaller scales or levels of organization as the result of how constraints at larger scales or levels of organization are implemented. This is modal accessibility in physical possibility space at work.

Emergents are often *Unpredictable* given exhaustive information at the “lower level” or smaller scale alone. However, given contextual emergence, the emergent is often predictable given the “lower level” information plus the relevant contextual features at other “levels” or

intuition. Thus, neither being unpredictable nor inexplicable need be hallmarks of emergence.

Contextually emergent explanation is not going to be reductive in either the intertheoretic sense of derivation nor in any synchronic notion of reduction involving the properties of parts determining the properties of wholes (what analytic metaphysics calls realization), and not in any causal-mechanical sense of reduction as with localization and decomposition in biological systems. But there will be predictability in many cases, and multiscale explanation, nonetheless.

The term *Novel* is certainly loaded. We have already said that an emergent can be predicted and explained, so given contextual emergence, novel means unexpected and irreducibly different in kind from features and concepts connected to the “lower level” or smaller scales. It can however mean more than this. For example, Kim (1998,1999), among others, has argued that if new “causal powers” emerge at a “higher-level” not reducible to or realized by “lower-level” “causal powers,” then we face a mystery as to where such “powers” come from (e.g., Kim, 1998, 1999). Given contextual emergence, so called “causal powers” are just extrinsic dispositions that typically require interdependent multiscale conditions. Kim’s worries about microphysical causal closure and exclusion have no purchase given contextual emergence. This is the real source of ontological novelty.

Finally, contextual emergence explains why novel emergents arise. Contextual emergence is multiscale in that “higher-level” or target domain information is required to enrich and constrain the laws and properties of the “lower level” or underlying domain to produce the set of contingent necessary and sufficient conditions for explanation of the emergent. Thus, contextual emergence focuses on making explicit the essential features absent in the fundamental level or underlying domain. Scientific explanations don’t float free in their own “level” or domain alone. Instead, scientific explanations implicitly rely on contextual features not contained in or implied by the lower level or smaller scale. Nevertheless, the absence of explanatory reduction does not imply explanatory or ontological disunity—pluralism yes, disunity no. Contextual emergence does not suggest the hierarchical structure implied by foundationalism, it also does not suggest a world of reified and explanatorily closed levels of

organization. Nor does it suggest the “gappy” world of C. D. Broad and his “transordinal” laws (Broad, 1925).C

Contextual emergence implies a contingent multiscale web of inextricably interconnected and interdependent extrinsic dispositions most of which are in constant flux. Some laws, constraints, principles, and so forth, are more general and subsume more phenomena than others, but such constraints and laws, while not violated by emergents, need not determine all the other phenomena at every scale. The kinds of multiscale relationships in brain networks described in Section 2, certainly seem like a case of contextual emergence par excellence (Silberstein, 2022; Bishop et al., 2022).

#### 4. Conclusion

It is important to begin by noting that something like contextual emergence seems to be at work in some other recent accounts of complex biological systems such as brains. I think this is important because whether this is a minority view that needs bolstering or the beginning of an inevitable trend, all interested parties need to know where to look for support and insight. This is not to say that there is complete unanimity and agreement here, far from it. So, after annoyingly quoting some of these people at length to make my point, I will then summarize some of the lessons I think we are learning from all this, or at least what I wish to conclude from all this.

Let us begin with philosophers of neuroscience Winning & Bechtel (2018). They have recently been arguing that mechanistic explanation is best conceived in terms of constraints: “We provide a new account on which the causal powers of mechanisms are grounded by time-dependent, variable constraints” (Winning & Bechtel, 2018). They also note: “The framework of constraints can be applied iteratively—a macroscale object can be further constrained by incorporating it into a yet larger-scale object” (Winning & Bechtel, 2018).

All of this sounds a great deal like contextual emergence, but especially the following characterizations: “Thus, on our view, when constraints enable objects to have novel, emergent behaviors, this is tantamount to the emergence of causal powers.” Winning & Bechtel (2018) points out, by means of possessing such emergent powers, mechanisms and components causally produce the effects they do.” And finally: “By restricting some degrees of freedom of its components and thereby enabling the whole mechanism to do things that would otherwise not be possible, constraints determine the causal powers of a machine or mechanism. Of particular importance are

*those constraints that are flexible and time-dependent These enable machines to operate in different ways on different occasions.”* Winning & Bechtel (2018) argue that mechanisms conceived as constraints grounds the causal powers of mechanisms. The idea seems to be that mechanisms just are sets upon sets of constraints. If I understand them correctly, then I agree.

Philosopher of cognitive science Weiskopf (2016) possibly goes further noting that the multiscale and multiply-realizable nature of neural processes calls mechanistic explanation itself into question: “One upshot of this form of organization is that the neural regions that participate in this assembly may have no identifiable cognitive function outside of their role in the ensemble. While classical localization assumed that distinct cognitive systems would have disjoint physical realization bases, massive redeployment and network theory seem to demonstrate that different systems may have entangled realizers: shared physical structures spread out over a large region of cortex. This suggests that not only will there not be distinct mechanisms corresponding to many of the systems depicted in otherwise well-supported cognitive models but given that the relevant anatomical structures are multifunctional in a highly context-sensitive way, perhaps there will be nothing much like mechanisms at all—at least as those have been conceived of in the dominant writings of contemporary mechanistic philosophers of science. And while it might be that these networks should count as mechanisms on a sufficiently liberal conception of what that involves, widespread entanglement still violates Poldrack’s constraint that distinct cognitive structures should be realized in distinct neural structures” (Weiskopf, 2016).

Some neuroscientists are also suggesting similar ideas about the contextually emergent organization of the brain and the nature of multiscale constraints. Thompson & Varela (2001) prefer the expression “reciprocal causality.” Such “causal” or dynamical relations are bi-directional, simultaneous, synchronous, and global, in defiance of standard accounts of causal relationships. Here I would substitute ‘causal’ talk for the various kinds of constraints as characterized by contextual emergence. In Pessoa (2022) he characterizes brain functions as “emergent properties.” He emphasizes the “interactional complexity of the brain” and notes the distributed, mutual, and reciprocal nature of multiscale interactions in the brain. He characterizes the brain as a decentralized heterarchy. He specifically focuses on the: (1) the massive combinatorial anatomical connectivity in the brain; (2) the highly distributed functional coordination; and (3) overlapping networks/circuits as functional units.

Here is how [Pessoa \(2023\)](#) characterizes the centrality of contextuality at work in the brain:

*“To motivate the challenges of mapping structure and function, we discuss neural circuits illustrating the high anatomical and functional interactional complexity typical in the brain. The ideas of network overlap and dynamic organization are related. If brain areas can belong to multiple networks, what determines the strength of a region’s affiliation to a specific network? Here, context plays a pivotal role: region A will participate strongly in network N1 during a certain context C1 but will be more strongly linked with network N2 during context C2. Parts of the brain (say, populations of neurons within areas) affiliate dynamically with other elements in a highly context dependent manner driven by the current endogenous and exogenous demands and opportunities present to the animal. Critically, network properties are novel (with respect to that of individual regions), and key functions are distributed across regions or neuronal populations”* ([Pessoa, 2023](#)).

In what follows, neuroscientist [Parker \(2022\)](#), still happy to align with mechanistic reduction in some sense, affirms the multiscale, multiply-realizable, extended, and contextually driven nature of overlapping brain networks and beyond:

*“These variable relational effects show that multiple neurophysiological states ( $N1 \vee N2 \dots Nn$ ) can realize a single behavior or cognitive process ( $P1 \leftrightarrow \_N1 \vee N2 \dots Nn$ ). This could be considered a neurobiological example of multiple realisability...But the evidence above suggests that nervous system outputs are linked to multiple, not single neurophysiological states, even when the cellular properties and the output are both measured in comparable detail* ([Aizawa & Gillett, 2009](#)). *Relational aspects are not confined to interactions within the nervous system but also reflect interactions of the nervous system with the body (e.g., proprioceptive, neural-immune, and gut-brain interactions) acting in the environment (see* [Dreyfus, 2012](#)). *This has been called embodied cognition* ([Shapiro & Spaulding, 2021](#)) *and has been inspired by ecological psychology and neuroethological analyses* ([Chiel & Beer, 1997](#)). *These are referred to as levels, but while this may*

*provide a simplifying concept for organizing data and analyses it also gives the erroneous impression of effects working up or down through separate stages when all effects are occurring simultaneously* ([Noble, 2012](#))” ([Parker, 2022](#)).

My hope is that such philosophers and neuroscientists can rally around contextual emergence as a reasonable scientific ‘paradigm’ for 21<sup>st</sup> century cognitive neuroscience.

I think what we are learning is that human beings are not just brains, and brains are not just computers, certainly not in any Turing, cognitivist, or modularist sense of the computational theory of mind (CTM). As many people have noted there has long been a schism in cognitive science between the neo-mechanists who focus on biological mechanisms, computationalists who focus on functional mechanisms, and dynamists who focus on brains and cognition as dynamical systems ([Chemero & Silberstein, 2008](#)). While I am certain that there are some suitably liberal notions of computation that apply to brains and while I don’t doubt the importance of mechanism hunting for cognitive neuroscience, I think the dynamical networks model is starting to look pretty good.

Humans as conscious cognitive agents then are, among other things, hubs in spatiotemporally extended 4E multiscale bio-psycho-social networks. Given all of this, it is probably best to view mammalian brains and other nervous systems as plasticity-robustness-adaptability-engines that partly modulate said multiscale network interactions, i.e., brains are themselves multiscale hubs of a sort in such multiscale networks. This conception of cognitive agents certainly doesn’t ‘roll off the tongue’ like the computer metaphor of the brain, but I believe it is truer to reality.

I also believe this changing conception of brains and people is already bearing fruit in psychology and psychiatry. For example, there is now a discipline of “multiscale network neuroscience” devoted to explaining and better treating various mental disorders from this perspective. The idea is to view certain mental disorders such as schizophrenia as network disruptions or anomalies on one or another scale of the brain ([Silberstein, 2014](#)). As [Bassett et al.](#)

(2018) put it, “Major neuropsychiatric disorders such as psychosis are increasingly acknowledged to be disorders of brain connectivity. Yet tools to map, model, predict, and change connectivity are difficult to develop, largely because of the complex, dynamic, and multivariate nature of interactions between brain regions”

Here is how [van den Heuvel et. al \(2019\)](#) describe this discipline and their own work:

*“We discuss the upcoming field of multiscale neuroscience, with a specific focus on multiscale associations in brain connectivity in the context of mental disorders. We begin with an overview of empirical findings suggesting multiscale relationships among the genetic, molecular, cellular, and macroscale organization of healthy brain connectivity and behavior. With these interactions in mind, we then discuss multiscale findings in, among others, schizophrenia, and autism, as examples of mental disorders in which studies have highlighted multiple cross-scale alterations to brain structure and function. We discuss proposed mechanisms through which micro-and macroscale properties of brain-function and disfunction maybe related”* ([van den Heuvel et al., 2019](#)).

This approach to certain mental disorders focuses on the importance of looking at multiscale relationships of brain organization. The discipline combines information on various alterations in brain connectivity at the genetic, cellular, circuitry, and macroscale connectome levels. The goal is to improve understanding about the possibly multiscale causes of certain mental disorders, “*placing disease-related differences in brain connectivity into a continuum of effects across multiple scales of brain organization*” ([van den Heuvel et al., 2019](#)).

While no doubt some mental disorders only involve disruptions to multiscale brain networks, some of us have gone further to suggest that other mental disorders might be best viewed and treated as disruptions to multiscale bio-psycho-social networks, in which the brain and the person are embedded ([Silberstein, 2014](#); [Fuchs, 2012](#)).

Take the case of addiction for example. It is common these days to view it as a brain “disease” ([Levy, 2013](#)). We have already seen that the dopamine reward model has problems. With regard to addiction, it is important to recognize that dopamine itself does not equal pleasure and acknowledge the evidence that having a dopamine-based neural reward system is neither necessary nor sufficient for addiction ([Hart, 2021](#); [Levy, 2013](#)).

In contrast, contextual emergence focuses on brain, body, and material/social environment in explaining and treating addiction. The mechanisms that neuroscientists have identified provide at best some of the necessary conditions for addiction. It is not surprising that various features of our brains would be involved in addictive behaviors. But these neural mechanisms do not possess the necessary and sufficient conditions for addiction. The physiology of the body (embodiment), the environment and social contexts in which people are participating (extendedness), their activities in their environments (enactivity), and the many ways environments shape people (embeddedness), need to be brought into the explanation of addiction.

The key is in discovering what stability conditions at various scales define the nested contexts enabling people to become entrained in addictive behavior. For instance, the strongest correlates and biggest predictors of addiction are socioeconomic, such as poverty, family dysfunction, isolation and loneliness, lack of education, emotional abuse, trauma, and deprivation ([Hart, 2021](#)). The brain disease model alone does not stand a chance of making much progress on understanding and treating addiction, so has negative actual-world consequences for dealing with something like the opioid crisis in the United States. The point is that brains do not become addicts, people do.

All of this suggests that a major step forward in the science of such multiscale networks would be better formal tools and computer simulations that allow us to see key diachronic and synchronic relations at multiple scales between the various elements of such



networks. Hopefully, *The Journal of Multiscale Neuroscience* will help bring this about.

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