ChatGPT (OpenAI) was used to assist with structuring, condensing, and refining the prose and to help synthesize the author's ideas. All conceptual, analytical, and interpretive content was produced and verified by the author.

The Neural Sensitivity Model: Sensitivity and Large-Scale Neural Network Dynamics in Creativity and Mental Illness

By Enes Cakir

Abstract:

Creativity and mental illness have long been linked, yet the neural mechanisms underlying this relationship remain poorly understood. Recent developments in network neuroscience have provided a tripartite network architecture of the brain — the Default Mode Network (DMN), Executive Control Network (ECN), and Salience Network (SN) — which provide a new approach to understanding this relationship. Specifically, viewing the brain through this architecture offers a way of interpreting creativity and psychopathology through network connectivity patterns. In parallel, empirical work on the construct of sensitivity has established that individuals differ in the depth and intensity with which they process internal and external stimuli. Notably, sensitive individuals are both more creative and vulnerable to mental illness. Recent research that analyzes the construct of sensitivity through network connectivity patterns within the tripartite framework of the brain provides support for a link between creativity and psychopathology in terms of sensitivity. Despite independent advances in these literatures, no existing model integrates network connectivity, sensitivity, creativity, and psychopathology into a unified developmental model. In this paper we propose the Neural Sensitivity Model which conceptualizes sensitivity as the foundational neural developmental substrate linking creativity and mental illness. This model seeks to clarify when and how heightened sensitivity gives rise to creative expression or psychopathology and how pathological states can be bridges into higher forms of creativity, ultimately reframing contemporary understandings of illness and creativity/health in terms of a connected developmental continuum rather than as opposed constructs. Philosophically, we present a definition of the human as a fundamentally creative organism where creativity itself is implication of the necessary process of oscillating between imbalance and balance.

Introduction

This paper proposes a model of understanding the link between creativity and psychopathology through the developmental neural trait of sensitivity. It builds the foundation for this model through the emerging science of network connectivity patterns in the brain which have transformed methods of approaching human cognition and behavior. Rather than viewing the brain in a static manner, network neuroscience enables a perspective that incorporates dynamisms, interrelations, and multi-modal complexities to generate a more holistic view of the mind. We view all of the relevant elements of this model in isolation before integrating them into a comprehensive holistic model which views the human as a constantly developing entity where, as it were, the parts cannot be separated from the whole.

The research we cover is largely recent and developing. Gaps exist and continue to emerge as our basic understandings and assumptions continue to be challenged by research. It appears to the authors that though these domains are not integrative in their approach, the research is leading towards such an integrative approach as a result of the discoveries which necessitate such a view to maintain coherence. Additionally, limitations persist in terms of research methodology. Though we relate the gaps and limitations we find in the literature, the model we present forms an infant appraisal of what we believe research *will* arrive at. As such, this model is, as the brain it seeks to describe, developmental and creative.

To relate a road map for this paper, first, we review the current literature regarding network connectivity patterns and relate them to the neural dynamics underlying both creativity and psychopathology. Next, we examine existing research on the creativity–psychopathology relationship. We then review the current literature on sensitivity as well as its relation to creativity and psychopathology. Following this review, we investigate the limited research on sensitivity in terms of network connectivity dynamics. From this investigation, we propose our neural sensitivity model and present our hypotheses for understanding creativity and mental illness. We then contextualize this framework in relation to contemporary understandings of mental illness and creativity and reframe how these present understandings may change given this theory. Accordingly, we outline avenues for empirical testing and then discuss the

therapeutic and conceptual implications of reframing mental illness through the lens of neural sensitivity. Finally, we present further consideration, the limitations and gaps of our study, and future directions of study.

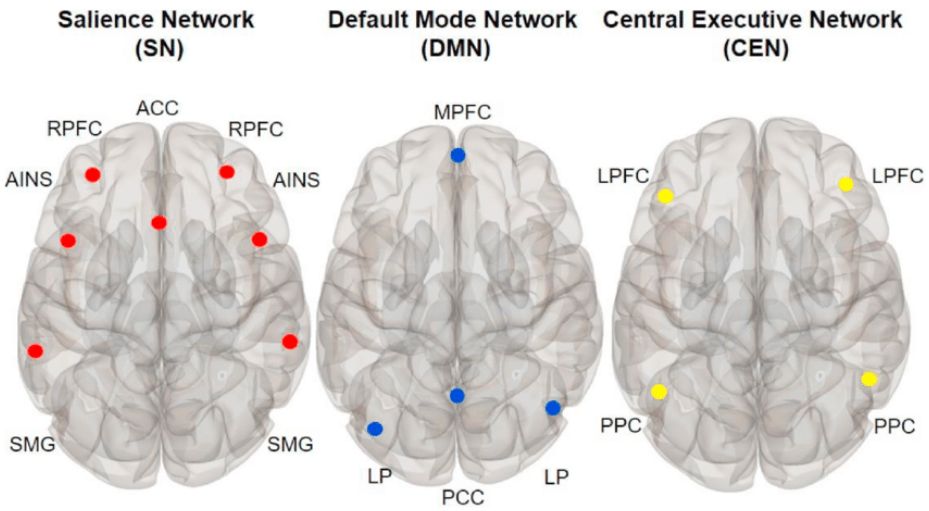
1. The Network Model of the Human Brain

Over the past fifteen years, developments in network neuroscience have reshaped our understanding of the operational structure of the human brain into a neural architecture rooted in large-scale inter-related functional structures rather than isolated or total regions(Fox & Raichle, 2007; Bullmore & Sporns, 2009; Bassett & Sporns, 2017). Menon's tripartite model (2011) was a significant movement in this process that proposed that the various elements of human cognition emerge from the dynamic interaction of three core networks: the Default Mode Network (DMN), Executive Control Network (ECN), and Salience Network (SN). This model has become foundational in contemporary accounts of creativity, emotion regulation, and psychopathology and is key to the model we will propose in this paper.

The DMN comprises regions such as the medial prefrontal cortex, posterior cingulate cortex, and angular gyrus(Raichle et al., 2001). It supports internally oriented processes such as mind-wandering, autobiographical memory, mental simulation, and spontaneous associative thought, and can be understood as the structure of the mind occupied with the inner world of the self (Raichle et al., 2001). The ECN — sometimes called the Frontoparietal network (FPN) or Central Executive Network (CEN) — is anchored in the dorsolateral prefrontal and posterior parietal cortices and is responsible for focused attention, working memory, planning, and the inhibition of irrelevant or intrusive information, functions which are central to adaptive goal-directed behavior (Shen et al., 2019). In contrast to the DMN, the ECN can be practically understood as the structure of the mind that is occupied with the external world. The SN, centered on the anterior insula and dorsal anterior cingulate cortex, detects salient internal and external events and coordinates the switching between DMN- and ECN-dominant states, and can be understood as the network which mediates and recursively relates these two respectively internal and external modes of experiencing and cognition (Seeley et al., 2007).

These networks and other interrelated regions of the brain are identified through patterns of correlated activity in blood-oxygen-level–dependent (BOLD) signals during rest and task performance (Fox & Raichle, 2007), (Bassett & Sporns, 2017). A region is deemed part of a network and/or network activity based on degree of BOLD correlated activity with other regions

during cognitive processes. It is important to recognize that the brain's functional architecture is highly dynamic; networks can strengthen, weaken, and shift their coupling patterns on timescales ranging from seconds to years.



These dynamics are not only measured by fMRI. Additional modalities such as magnetoencephalography (MEG) and electroencephalography (EEG) measure the millisecond-scale neural oscillations that underlie network reconfiguration (Uhlhaas & Singer, 2012). Diffusion-weighted imaging is also a supplementary measurement which maps the cortico-cortical axonal pathways that make up the neural structures beneath these interactions (Hagmann et al., 2008). Together, these multimodal approaches give us a more detailed picture of the way that large-scale brain networks reorganize across various circumstances.

Additionally, research on neuroplasticity demonstrates that these network configurations are constantly changing and dynamically reconfigure due to environmental input, stress exposure, and learning demands (Kolb & Gibb, 2011; Bassett et al., 2011). These findings reiterate the importance of environmental stimuli on the development of the brain, and further studies aim to expand on these findings. The tripartite model therefore delivers a network based framework for understanding human cognition in the context of the dynamically developing network correlations of various regions of the brain.

1.1 Network Dysregulation and Psychopathology

A key contribution of the tripartite framework is the comprehensive explanatory heuristic it provides for understanding psychopathology. In the context of a tripartite model, essentially all forms of psychopathology demonstrates themselves in the form of some network disruption or dysregulation(Menon, 2019).

For example, anxiety disorders reflect heightened DMN activity (in the mPFC and PCC) which supports the inner expressions of excessive self-referential worry; an overactive SN increases salience of perceived threats and broadens the scope of what is considered threatening; and a hypoactive ECN limits the ability to exert top-down control over anxious ideation — inhibitory power over distractions and stimulus is overwhelmed by the vigilant overactivity of the SN as well as the rumination of the DMN on these recurring salience triggers, resulting in a mode of both heightened awareness but reduced ability to focus on entities outside of the scope of the internally perceived threats (Sylvester et al., 2012; Makovac et al., 2016; Etkin & Wager, 2007; Bishop, 2009).

Similar network signatures of hyper and hypo activation of networks and altered connectivity patterns characterize depression, post-traumatic stress disorder, bipolar disorder, and schizophrenia (Menon, 2011). Viewing these disorders in the scope of a network framework of the mind can provide new means for engaging with, interpreting, and treating mental illness.

1.2 Network Neuroscience as a Neuroplastic Framework for Adaptation

Viewing these connectivity patterns through the lens of neuroplasticity brings to view a developing central insight of neuroscience: network configurations reflect adaptations to lived experience. Developments in neuroscience have demonstrated that the brain is far more flexible in both structure and function that previously believed (Kolb, B., & Gibb, R., 2014). Research has found that the brain changes both functionally and structurally as a result of stressful experiences and places the brain's plasticity as a key player in the adaptation to stressful experiences (McEwen, B. S., & Gianaros, P. J., 2011). Large-scale functional networks exhibit flexible reconfiguration over time, with connectivity patterns reshaping as individuals adapt to experience and context (Bassett & Sporns, 2017). Learning itself drives large-scale plasticity, producing measurable shifts in functional coupling and network organization (Dayan & Cohen,

2011). Altogether etwork neuroscience increasingly converges on the view of the brain as a perpetually recursive adapting system mediated by experiences with the external environment.

Viewing the brain as a constantly creative and recursively adapting system is integral to deepening our understanding of human behaviors. Not only is there an external environment which can impact us, but, indeed, and perhaps even more importantly, an inner environment which actively shapes our experiences, sense of self, and actions. As predictive-processing accounts emphasize, internally generated models of the world and of the body continuously shape perception, emotion, and the sense of self (Seth, 2013). Intrinsic brain activity—especially within the DMN—provides an internal context that shapes how external events are interpreted and integrated (Raichle, 2015). Pessoa's research into the dynamic connection between emotion and cognition argues that internal emotional and motivational states fundamentally bias cognition, attention, and action (2013). Research from Keller and colleagues supports the view that inner perceptions are vital to lived experience, arguing that much perception emerges from internally generated predictions that actively shape experience, not merely from external input (Keller & Mrsic-Flogel, 2018). These perspectives make clear that the brain is not merely shaped by the world—it is constantly shaping the world from within, generating an internal environment that is as developmentally and behaviorally consequential as external experience itself.

Drawing from this perspective, we can view pathological patterns as possibly originating from functional responses to environmental pressures both within and without — such as chronic stress, inconsistent caregiving, trauma exposure, instability, negative thought patterns, rumination — which form short term coping mechanisms but long terms maladaptations in new and developing contexts (Ellis & Del Giudice, 2019; Sinha, 2008). These adaptive responses become deeply ingrained through plastic development due to environmental experience, gradually forming into network configurations that persist even after the original environmental demands have disappeared (McEwen & Gianaros, 2011). From this perspective, psychopathology reveals itself not as mere dysfunction, but the neural patterns developed from engagement with the world that persist after the experiences which formed them have dissipated. Hypervigilant network patterns, for example, can offer short-term survival advantages in threatening environments — such as a dysfunctional home, an abusive relationship, war — yet

persist long after the conditions that shaped them. Post-traumatic-stress disorder is a prime example of a lingering experience response, and this point is especially relevant in child development given the higher plasticity of the developing brain.

From this vantage point, developing research on the neuroplasticity of the brain in terms of neural networks and mediation with the environment within and without can move research and clinical practice towards reframing mental illnesses not simply as disorders, but as dynamically formed adaptations stemming from the brain's continuous attempt to regulate itself relative to its environment. The tripartite model provides a cogent foundation for conceptualizing psychopathology as developing patterns of network dynamics interacting with environmental conditions.

2. Creativity and Psychopathology Through the Network Neuroscience Framework

Creativity and psychopathology have long been studied across psychology, neuroscience, and the arts. Over the past two decades, advances in network neuroscience have transformed our understanding of both creativity and psychopathology by researching the two in the context of large-scale patterns of functional connectivity. Within this framework, creative cognition is not merely an amorphous trait but a network coordination signature — dynamically coordinated and balanced interaction between the DMN, ECN, and SN — which frees the concept of creativity from the grip of particular individuals and reframes it as potentiality in all humans. Likewise, psychopathology, rather than mere dysfunction, can be understood in the context of a network framework of the mind where pathological conditions are observed as particular imbalanced network signatures.

2.1 Neural Bases of Creativity

In terms of network neuroscience, creative thought emerges from a mixture of spontaneous internal processes and deliberate inhibitive control (Beaty et al., 2016; Beaty et al., 2018; Benedek & Fink, 2019). Functional neuroimaging studies show that creative thought is realized through dynamic coupling between the DMN and ECN (Beaty et al., 2016; Beaty et al., 2018). The DMN contributes spontaneous ideas, internal images, autobiographical associations, and conceptual remapping (Raichle et al., 2001; Andrews-Hanna, Smallwood, & Spreng, 2014),

while the ECN supports working memory, evaluation, planning, and the selection or inhibition of ideas (Niendam et al., 2012; Cole et al., 2013). Creativity therefore relies on the co-activation of these two networks — that is, the co-operation of internal and external cognitive processing.

Recent work has expanded the view that creativity is a dynamic oscillation and increased connectivity between the DMN and ECN. Chen et al. (2025) found that the frequency of transitions between DMN–ECN states at rest reliably predicted creative ability, with the determination of a goldilocks zone of switches for optimal creative production. These findings align with broader views of creativity as a recursive process of internal processing and inhibitive refinement that depends on alternating internal and external orientations. In other terms, too much internal processing without executive pruning leads to inner richness but without form, and, inversely, too much executive function without inner introspection enables high functioning but without novel idea generation. Rather than viewing these poles as opposing, network neuroscience increasingly supports the perspective that they are complementary.

Although the DMN–ECN relationship has been the focus of network creativity research, the role of the SN has received less empirical attention (Beaty et al., 2016; Menon, 2011; Zabelina & Andrews-Hanna, 2016). However, because the SN controls transitions between internal and external modes of condition, salience detection, and the prioritization of emotionally or cognitively relevant stimuli, it self-evidently provides the switching mechanism that enables creative cognition to oscillate between generative and evaluative states. Existing studies suggest that individuals with higher creative capacities show stronger SN involvement during insight and improvisation tasks (Zabelina & Andrews-Hanna, 2016), indicating that SN functioning may be essential for creativity's temporal dynamics.

Together, the literature suggests that creativity relies on a flexible, balanced, and cooperative tripartite network connectivity in which the SN orchestrates transitions between the DMN and ECN. The flexibility that leads to higher connectivity between networks however may be the same mechanism which leads to pathological states of imbalanced connectivity patterns.

2.2 Network Connectivity Patterns in Psychopathology

To reiterate from earlier points, network neuroscience has significantly reshaped and continues to reshape contemporary understandings of mental illness. Notably, nearly all major psychiatric conditions involve disruptions in the DMN–ECN–SN circuitry (Menon, 2011). Network neuroscience research therefore supports the view that, rather than explicit categorical definitions, psychopathologies reflect particular neural organization patterns which are contained with a dynamic, neuroplastic, and recursively adapting system. Network research into various disorders has revealed introductions into the underlying network dynamics at play.

Anxiety Disorders

Anxiety disorders are characterized by excessive self-worry and hyper-vigilance. In a network framework of the human brain, anxiety disorders show patterns of dysregulation in the DMN, ECN, and SN. Resting-state and task-based imaging studies reveal DMN hyperconnectivity in the medial prefrontal cortex (mPFC) and posterior cingulate cortex (PCC), a dynamic which leads to excessive rumination and consistent worry. These patterns are accompanied by hyperactivity of the SN, amplifying threat detection and increasing sensitivity to stimuli (Etkin & Wager, 2007; Seeley et al., 2007; Sylvester et al., 2012; Menon, 2011). Complementing these alterations is reduced ECN connectivity, which impairs top-down regulation, attentional control, and the inhibition of intrusive anxious thoughts (Sylvester et al., 2012; Makovac et al., 2016; Etkin & Wager, 2007; Bishop, 2009). All together, these network connectivity patterns demonstrate a neural architecture that is characterized by hyper-connectivity in the DMN, hyperactivity in the SN, and reduced connectivity in the ECN.

Depression

Major depressive disorder (MDD) is a disorder that is associated with excessive rumination and difficulty to focus on cognitive tasks. In terms of network neuroscience, the brains of individuals suffering from MDD show patterns of DMN hyperconnectivity (Sheline et al., 2009; Hamilton et al., 2015; Whitfield-Gabrieli & Ford, 2012). Functional imaging studies reveal weakened coupling between the DMN and ECN, a pattern which reduces the ability to focus on cognitive tasks, increases rumination, self-focus, vigilance, and causes emotional, visceral, and autonomic dysregulation (Sheline et al., 2010; Kaiser et al., 2015). Kaiser et al. (2015) found that major depressive disorder is marked by hypoconnectivity within frontoparietal executive networks,

alongside hyperconnectivity within the DMN and increased coupling between DMN regions and frontoparietal control systems, network findings which reflect the symptomatology that characterizes depression: decreased cognitive control and heightened internally oriented thought. Additionally, aberrant SN function—including altered anterior insula and dACC activity—disrupts the detection and prioritization of emotionally salient stimuli, contributing to symptoms such as anhedonia, diminished motivation, and blunted affect and the difficulty to effectively switch between networks (Sheline et al., 2009; Hamilton et al., 2015). Viewing depression disorders through the context of the tripartite model of the brain reflects a basic patter of hyperactivity of the DMN, hypo-connectivity of the ECN, and aberrant SN functioning.

Schizophrenia

Schizophrenia is a disorder that reflects a significantly altered tripartite network architecture. Converging neuroimaging research demonstrates that schizophrenia is characterized by disordered large-scale network organization, including dysregulated interactions among the DMN, ECN, and SN (Pettersson-Yeo et al., 2011; Menon, 2011). A central feature of schizophrenia is DMN hyperactivation during externally oriented tasks, denoting an increased intrusion of inner thoughts — such as hallucinations or delusional ideation — during states that require cognitive focus (Whitfield-Gabrieli et al., 2009). This inhibitive deficiency and hyper-connectivty of the DMN is naturally accompanied by pronounced ECN dysconnectivity, reflected in deficits in working memory, cognitive control, planning, and goal-directed behavior. Perhaps most notably, the SN is also disrupted, particularly in the anterior insula and dorsal anterior cingulate cortex, leading to impaired switching between internal and external modes as well as aberrant assignment of salience to neutral stimuli (Palaniyappan & Liddle, 2012). The dysregulation of the SN in individuals suffering from schizophrenia is key to understanding the experiential dimension of the disorder; aberrant saliency tags significance on various and many things, producing a neural signature that is highly internally oriented that attaches high significance on seemingly irrelevant entities, this salience tagging greatly altering the experience of reality. Together, these network dysfunctions contribute to schizophrenia's core symptoms: impaired reality monitoring, cognitive fragmentation, and disorganized behavior. However, current models remain limited by the nuance of the disorder and the correlational nature of large-scale network analyses, underscoring the need for more studies.

Bipolar Disorder

Bipolar disorder is a unique disorder that is characterized by a distinctive oscillatory pattern of state-dependent network dysregulation (Phillips & Swartz, 2014). Bipolar individuals experience both manic/hypomanic and depresssive/hypodepressive episodes, alternating between the two largely at casually ambiguous rhythms (Rowland et al., 2018). Extensive research has documented that bipolar disorder is connected to disrupted neural connectivity among various brain regions, notable changes in structural and functional brain networks, and abnormal reactivity in particular brain regions (Perry et al., 2019; Yoon et al., 2021).

A 2024 study found that bipolar disorder reflected reduced time spent in a globally integrated state and increased time in anticorrelated configurations driven by DMN dyscoupling with sensorimotor and salience-related networks (Zhang et al., 2024 —Reconfiguration of brain network dynamics in bipolar disorder: a hidden Markov model approach). The observed hypoactivation and structural disruption of frontal–parietal cognitive control regions further point to weakened ECN regulation and impaired SN-mediated switching, supporting tripartite network models in which BD arises from unstable DMN–ECN–SN dynamics underlying mood oscillation (Menon, 2011; Zhang et al., 2024).

During manic episodes, individuals exhibit reduced ECN engagement — impaired inhibitory control, planning, and risk evaluation — along with heightened SN-driven impulsivity that amplifies the emotional or motivational importance of stimuli. Moreover, manic states are further characterized by anomalies in DMN activity, including increased spontaneous ideation and internally generated thought, which may contribute to racing thoughts and grandiosity. In depressive phases, the disorder converges with unipolar depression with network connectivity patterns reflecting DMN hyperconnectivity, impaired DMN–ECN integration, and SN abnormalities that contribute to negative affective biases. A study comparing depression mood disorders with bipolar depression found that neuroimaging results suggest that bipolar individuals are more influenced by sensory and emotional processing in relation to the environment. This reactivity to the environment suggest that a key mechanism of the oscillatory nature of bipolar is the intensity of environmental experience on neural network patterns, further

suggesting a high latent neural flexibility in bipolar individual. Likewise, this high flexibility corresponds with the research on reduced ECN connectivity in bipolar individuals — lowered inhibitive functioning enables network switching to be more rapid, intense, and occur with less stimuli.

This research suggest that in terms of network connectivity, bipolar disorder reflects a greater flexibility of network change leading to instability and abnormal connectivity patterns across time. Further research of this disorder in terms of network connectivity patterns is necessary to draw more conclusions and support preliminary conceptual hypotheses.

Attention-Deficit/Hyperactivity Disorder (ADHD)

ADHD is a disorder marked by characteristic disruptions in the interplay between task-positive and task-negative networks. A robust finding is evidence of ECN hypoconnectivity, particularly in frontoparietal circuits supporting sustained attention, working memory, and inhibitory control (Cortese et al., 2012; Mattfeld et al., 2014). This reduced ECN engagement is accompanied by intrusions of DMN activity into task states, which reflect elevated DMN connectivity and failure to suppress medial prefrontal and posterior cingulate regions during cognitively demanding tasks —mechanisms linked to attentional lapses and mind-wandering (Castellanos et al., 2008; Sonuga-Barke & Castellanos, 2007). The SN also shows irregularities in ADHD, including reduced reliability in initiating network switching and impaired salience detection, which further undermines efficient transitions from rest to task engagement (Castellanos & Aoki, 2016; Sripada et al., 2014). Overall, ADHD appears as a disorder of insufficient top-down regulatory control combined with unstable coordination between internally and externally oriented processing modes, yet, with a rich mind primed for associations and ideation.

Summary

Our cursory review of the network neuroscience research on these common mental illnesses demonstrates the usefulness of a tripartite model for further developing our understanding of the mechanisms underlying psychopathologies. Notably, some similarities among these disorders

make themselves apparent in the context of a network approach. Significantly, the common impairment of ECN functionality and the over-activation of the DMN appear to be common themes among the most common mental illnesses.

2.4 Converging Theories on the Creativity–Psychopathology Link

Where creativity reflects a dynamic coordination of brain networks, mental illnesses reflect some dysregulated pattern of connectivity. Given our prior discussion of neuroplasticity and the environmental impact of stimuli on the development of the brain as well as our recognition of various brain connectivity patterns in mental illnesses and creativity, many questions are revealed as the developmental element of both creativity and psychopathology. If networks can develop connectivity patterns either negatively or positively, what cause both positive development and negative development?

Despite decades of research, the relationship between creativity and mental illness remains a topic of debate. Early clinical studies documented elevated rates of mood disorders among highly creative individuals which made movement towards an empirically established link between the two (Andreasen, 1987; Jamison, 1993). Additional evidence in psychological studies indicate shared personality and temperamental characteristics between creativity and mental illness, including openness, emotional intensity, and cognitive divergence (Nusbaum & Silvia, 2011; Kaufman & Paul, 2014). Recent developments in network neuroscience have created a landmark framework for analyzing the overlap of neural signatures across both mental illness and creativity (Beaty et al., 2016; Hamilton et al., 2015; Menon, 2011), however, there is no existing framework which attempts to explain this overlap in a cohesive and syncretic manner and current theories remain empirically partial and fragmented.

Though creativity research demonstrates that DMN-ECN cooperation is central in creative cognition, psychopathological research reveals the dysregulated dynamics at play in various disorders, and psychological personality studies demonstrate overlap in trait correlations (openness as a trait that indicates higher creativity and increases vulnerability to psychopathology), no integrative developmental model pulls these various developments

together to provide an explanatory framework for the interrelation of these processes. Even though network research reveals the neural signatures of these states, there are gaps in how and when similar neural and psychological processes diverge into creativity or psychopathology, and vice versa. Our model aims to fill this gap.

2.5 Summary

In this section, we observed the network neuroscience findings on the neural patterns that underly both creativity and psychopathology respectively. To reiterate, creativity is succinctly related as cooperative coupling between the DMN and ECN orchestrated by effective SN-mediated switching, whereas psychopathology reflects chronic disruptions in these same circuits leading to deeply developed dysregulated patterns—whether through DMN hyper-activity, reduced ECN connectivity, or aberrant salience switching. A basic theme of common mental illnesses is increased DMN activity and reduced ECN connectivity: a greater reactivity towards environmental stimuli and a reduced ability to executively inhibit and parse this reactivity. These converging lines of evidence support the foundational claim developed in the sections that follow: neural sensitivity—manifested as intensified internal perceptual, emotional, and cognitive processing—forms the core developmental neural substrate that links creative potential with psychological risk in the form of a neural architecture denoted by increased sensitivity towards stimuli and greater flexibility of network patterns. This synthesis establishes the scientific and theoretical basis for the Neural Sensitivity Model.

3. Neural Sensitivity

In order to fill the gap between creativity and psychopathology and explain how neural states can oscillate from creativity to psychopathology, perhaps even must oscillated between the two, we present the core foundational metric of our model: the neural substrate of both creativity and psychopathology that we term simply as neural sensitivity.

3.1. Defining Neural Sensitivity

Sensitivity refers to the degree to which an individual can register, process, and respond to internal and external stimuli (Aron & Aron, 1997; Aron et al., 2012; Dąbrowski, 1964).

Contemporary psychological and neuro-scientific research increasingly conceptualizes sensitivity as a multidimensional trait encompassing emotional reactivity, perceptual acuity, and cognitive depth of processing (Aron et al., 2012). Current research readily recognizes that individuals experience stimuli differently and some individuals are more vulnerable to adversity — a fact affirmed by the popularity of the diathesis-stress framework (Pluess & Belsky, 2013; Assary et al., 2020). Likewise, research concerning differential susceptibility theory regularly demonstrates that individuals vary in their developmental plasticity on the basis of environmental stimuli (Belsky & Pluess, 2009; Pluess, 2015). Though empirical research supports the notion of differing levels of sensitivity among individuals, the underlying neuro-biological components as well as environmental influence that form important parts of the mechanism remain an active area of study (Pluess & Belsky, 2013; Assary et al., 2020).

The term Sensory Processing Sensitivity (SPS) has been created to refer to a trait which manifests in individuals as lower perceptive boundaries — the inner milieu is more permeable to stimulus —, greater reactivity to subtle stimuli — stimuli produce greater reactions —, increased flexibility of engagement — stimuli produces a wider range of reactions —, and deeper processing of sensory and emotional information — stimulus necessitates greater engagement with information (Aron & Aron, 1997; Aron et al., 2012; Marhenke et al., 2023). These sensitivity-related traits are theorized to be found in roughly 20–30% of the population (Aron & Aron, 1997; Acevedo et al., 2014).

A precursor to SPS was Dąbrowski's earlier construct of overexcitabilities (OEs) (1964), a measurable trait system linking creativity and psychopathology. Dąbrowski's theory related five types of OEs in varying combinations among sensitive populations: emotional, imaginational, intellectual, psychomotor, and sensory OE. This system offered a developmental framework for understanding how increased sensitivity shapes inner life, increases vulnerability to mental illness, and enhances potential for creativity, in our estimation the most cogent, comprehensive, and solid precursor of this present theory described in psychological rather than neurological terms.

We seek to contain this basic mechanism of sensitivity under the term neural sensitivity which attempts to reconceptualize sensitivity in the context of neural networks, supported by developing research on network neuroscience in creativity and psychopathology. Like the concept of SPS and the earlier OEs, neural sensitivity represents a neurological disposition toward deeper, more intense processing of stimuli, characterized by enhanced experience, stronger affective responses, increased cognitive engagement, greater vulnerability to mental illness, higher flexibility, and greater creative potential. This disposition functions as a general amplifier of inner and external experience, intensifying both environmental benefits and environmental risks.

3.2. Genetic basis for sensitivity

Here we provide a short review of the literature surrounding the genetic components of sensitivity. Notably, twin and molecular genetics research provides converging evidence that sensitivity has a partially heritable basis. Twin studies estimate that approximately 40–50% of the variance in Sensory Processing Sensitivity (SPS) and related environmental susceptibility traits is attributable to genetic factors (Assary et al., 2020), supporting an interpretation of sensitivity as a biologically rooted temperament rather than a purely psychological construct.

Several nominated polymorphisms—such as the serotonin transporter-linked promoter region (5-HTTLPR) and the dopamine D4 receptor gene (DRD4 7-repeat allele)—have been associated with heightened emotional reactivity and stronger gene–environment interactions (Caspi et al., 2003; Canli & Lesch, 2007; Bakermans-Kranenburg & van IJzendoorn, 2006). However, findings in the candidate gene literature are mixed, and many associations have not been consistently replicated across large samples or different methodological designs (Border et al., 2019). For this reason, our model does not rely on any single genetic mechanism or polymorphism and views genetic influences on a spectrum of potential possibilities.

As with traits such as height or cognitive ability, genetic factors may establish a general range of potential developmental expression, but the realization of that potential depends on environmental conditions. Unlike height—whose developmental trajectory becomes limited relatively early (though bone can grow through trauma) — neural sensitivity is within the

neuroplastic architecture of the brain (Kolb & Gibb, 2011, Takesian & Hensch, 2013, Pluess, 2015). Given this, we treat genetic evidence as broadly supportive of a more general claim: sensitivity reflects a biologically grounded disposition toward deeper processing and greater environmental responsiveness/adaptation —one that operates through a complex reciprocal nexus of interrelations between environment and mind. In this sense, genetics provides a foundation for understanding sensitivity as a generic evolutionary element of the human brain that interacts with environmental conditions and neural architecture rather than determining outcomes in isolation. Put simply, sensitivity, like all things, exists in a matter of degrees. This means that sensitivity, while partially heritable, remains open to both positive and negative development across the lifespan in relation to the nexus of self-environment interactions.

3.3. Measuring Sensitivity

Sensitivity has historically been assessed through self-report instruments, the most prominent being the Highly Sensitive Person Scale (HSPS) developed by Aron and Aron (1997). Dąbrowski OE questionnaire to evaluate OEs forms a precursor to this scale (Dąbrowski, 1964; Dąbrowski & Piechowski, 1977). The HSPS measures four core dimensions of Sensory Processing Sensitivity (SPS): depth of processing, emotional reactivity, overarousability, and sensitivity to subtle environmental cues. In terms of the Big Five personality traits, highly sensitive person (HSP) traits are most strongly correlated with openness to experience and neuroticism (Smolewska et al., 2006).

Multiple versions of the HSPS have demonstrated strong psychometric reliability across diverse populations, suggesting that SPS is a stable and measurable temperament trait (Smolewska et al., 2006). However, because self-report measures depend on introspective accuracy and show partial conceptual overlap with adjacent constructs such as neuroticism and introversion, researchers increasingly complement these tools with behavioral, physiological, and neurobiological assessments (Smolewska, McCabe, & Woody, 2006; Evans & Rothbart, 2007; Aron et al., 2012). Further research on SPS measurement methods can help support the basic claim of SPS — that particular individuals are more sensitive to environmental stimuli than others.

Behavioral research consistently demonstrates that individuals high in SPS display enhanced attentional bias toward subtle or emotionally salient stimuli (Jagiellowicz et al., 2011; Jagiellowicz et al., 2020; Aron et al., 2012). Tasks assessing perceptual discrimination/inhibition and attention alteration reveal that highly sensitive individuals detect lower-intensity changes in their environment and exhibit increased orienting responses to ambiguous or nuanced cues (Jagiellowicz et al., 2020). High-SPS individuals also typically show elevated autonomic reactivity, including stronger startle responses, greater heart-rate variability during emotional tasks, and intensified sympathetic activation in response to overstimulating environments (Aron et al., 2012). These findings suggest that sensitivity involves a broader biological preparedness to respond to and adapt to environmental information, not merely a subjective perception of being "over-reactive."

Though SPS has been extensively studied in behavioral and psychological settings, expansion into identifying the neural dynamics at play is recent and a limited realm of study. This study however, enabled by developments in network neuroscience, is key to bridging gaps of understanding between sensitivity, psychopathology, creativity, and the nature of the human brain.

3.4. Neural Network Correlates of Sensitivity

Neuroimaging methodologies have begun to further deepen our understanding of SPS and develop pathways into observing SPS in terms of network connectivity patterns. Jagiellowicz et al. (2011) showed that SPS is associated with increased activation in higher-order visual and attentional cortices during fine perceptual discrimination tasks, suggesting deeper perceptual encoding. Acevedo et al. (2014) found that SPS predicts stronger activation in regions such as the cingulate cortex, insula, inferior frontal gyrus, middle temporal gyrus, and premotor areas when individuals view emotional expressions of romantic partners and strangers. These regions form part of the brain's salience and socioemotional processing systems, suggesting that SPS involves heightened awareness, empathic resonance, and integrative socioemotional processing.

A study by Acevedo et al. (2021) found that higher Sensory Processing Sensitivity (SPS) is linked to distinct patterns of resting-state brain connectivity, supporting the trait's characteristic

"depth of processing." After an empathy task, individuals high in SPS displayed stronger connectivity within the ventral and dorsal attention networks and the limbic network, suggesting greater ongoing engagement of attentional control and emotional processing systems. They also showed increased connectivity between the hippocampus and precuneus, a pathway involved in episodic memory, while exhibiting weaker connectivity between the amygdala and periaqueductal gray and between the hippocampus and insula—regions tied to anxiety and habitual processing.

A 2025 study found that high SPS corresponds to weaker SN–FPN(ECN) connectivity, which in turn predicts emotional reactivity(Liu et al., 2025). This supports the view that sensitivity functions as the psychological expression of a more fundamental large-scale network architecture involving heightened salience detection and reduced regulatory integration.

Overall, the emerging evidence shows that SPS can be analyzed in terms large-scale brain networks communication. The research we have discussed indicates that high-SPS individuals tend to show stronger engagement of salience and socioemotional systems, deeper integration within attention and memory networks with weaker coupling between the SN and ECN. From this vantage point, the sensitive mind is one that takes in more, processes more, and regulates less inhibitively—an architecture consistent with a hyperconnected DMN and hypoconnected ECN. This basic neural architecture reflects the basic pattern of many of the psychopathology neural signatures we discussed in prior sections, only disorders states reflect intensification of these basic patterns. At the same time, the current studies remain limited by small samples, cross-sectional designs, and heterogeneous methods, making it difficult to draw firm conclusions about connectivity dynamics or causality. Much more work is needed to examine SPS through the lens of whole-network organization, however, based on the existing evidence, we predict that SPS will ultimately be characterized as a developmental profile of heightened DMN connectivity, reduced ECN regulatory coupling, and greater SN sensitivity—an architecture that supports depth of processing but also increases vulnerability to emotional overwhelm and requires an executive approach towards higher development. This executive approach can be mediated in many ways — environment, therapy, relationships — however, a key internally latent method is creativity.

3.5. Sensitivity and Creativity

Multiple lines of evidence suggest that sensitivity enhances creative cognition. Perceptually, sensitive individuals exhibit deeper and more fine-grained processing of sensory information, providing a richer set of raw material from which creative associations can emerge (Jagiellowicz et al., 2011). Personality research shows a strong correlation between SPS and Openness to Experience, the Big Five trait most consistently linked to creativity (Smolewska et al., 2006). Feist's (2010) work further identifies cognitive and affective sensitivity as recurrent traits among creative individuals.

In line with this, a recent review argues that the creativity literature has largely overlooked temperament—particularly sensitive temperament—as a foundational contributor to creative cognition (Bridges & Schendan, 2019). Synthesizing emerging work, the review identifies orienting sensitivity as the core temperamental component most strongly tied to creativity, a component that is characterized by heightened automatic attention, lower inhibition, and greater neural responsivity. According to this framework, sensitive, open individuals become more creative not through a single trait but through the interaction of plasticity genes, neurosensitivity mechanisms, and attentional networks that allow richer perceptual input and more flexible cognitive processing—precisely the capacities implicated in creative thought (Bridges & Schendan, 2019).

This connection was strengthened by a recent large-sample study showing that sensitivity is positively associated with everyday creative activity and social-emotional attunement. Laros-van Gorkom et al. (2025) found that higher SPS predicts more frequent creative ideas, greater engagement in daily creative behavior, and stronger affective and cognitive empathy. These associations persisted even after controlling for Openness, suggesting that creativity and empathy may not be incidental byproducts of personality but core expressions of the sensitive temperament itself. The finding that high-SPS individuals report less emotional disconnection aligns with the idea that their deeper processing of sensory and social cues fosters richer

interpersonal understanding—though this same depth may also increase vulnerability to emotional overwhelm.

The complexity of this dual nature becomes especially clear under adverse conditions. A study of artistically inclined adults during the COVID-19 restrictions illustrates the double-edged consequences of SPS later in life (Chou, 2023). Higher SPS, low peer support for artistic interests, and greater depression were all linked to reduced resilience, with SPS subcomponents diverging sharply between high- and low-resilience groups. Notably, the protective influence of creative self-concept (CSC) on depression varied depending on SPS level—even after controlling for neuroticism—suggesting that sensitivity can amplify the benefits of creativity when external support is present but may magnify vulnerability when it is absent. In this way, SPS emerges as both a resource and a risk factor, depending on environmental context. — Add Difffertnaul susceptiblity section.

Our discussion of SPS in creativity naturally leads to the network-level mechanisms underlying these patterns. Creativity depends on dynamic interplay between the DMN (idea generation, associative thought) and the ECN (evaluation, refinement), with the SN coordinating transitions between them (Beaty et al., 2015; 2016). Sensitive individuals exhibit connectivity patterns that appear to facilitate precisely this dynamic: heightened DMN engagement supporting generativity and internal richness; reactive SN responsivity increasing salience detection and orienting; and reduced ECN rigidity enabling broader associative range and more fluid cognitive transitions.

Yet these same network dynamics that support creativity also foreshadow potential vulnerabilities. As we turn to the next section on sensitivity and psychopathology, we will examine how heightened DMN activity, SN hyper-reactivity, and reduced ECN constraint—while advantageous for rich internal experience—can become liabilities under stress, contributing to rumination, emotional overwhelm, and dysregulation into pathology.

3.6. Sensitivity and Psychopathology

Sensitivity is a well-established risk factor for mood disorders. Individuals high in SPS report elevated stress, anxiety, and depression symptoms (Liss et al., 2005; Benham, 2006).

Importantly, these vulnerabilities do not arise solely from trait emotionality but from interaction effects with early-life environment (Belsky & Pluess, 2009; Ellis et al., 2011). Highly sensitive children show disproportionately negative outcomes under adverse caregiving conditions and disproportionately positive outcomes under supportive environments (Brindle et al., 2015; Aron et al., 2012). This dynamic is referred to as differential susceptibility theory and indicates that sensitivity amplifies environmental input: both nurturing and harmful conditions exert greater force on development.

Neurocognitively, SPS aligns closely with risk mechanisms seen in anxiety and depression, including increased DMN activity, heightened salience responsivity, and reduced regulatory control (Liss et al., 2005; Benham, 2006; Aron et al., 2012; Sylvester et al., 2012; Liu et al., 2025; Hamilton et al., 2015). DMN hyperactivity facilitates rumination and self-focused loops, SN hyperreactivity intensifies threat detection and autonomic arousal, and reduced ECN connectivity impairs top-down regulation, attentional control, and cognitive inhibition, mechanisms that are all implicated in multiple forms of psychopathology (Sylvester et al., 2012; Whitfield-Gabrieli & Ford, 2012). These convergences suggest that heightened sensitivity, when coupled with insufficient regulatory resources or chronic stress, increases the probability that neural dynamics will transition into dysregulated patterns associated with psychiatric disorders.

Notably, Liu et al.'s 2025 research found that individuals with SPS demonstrate an association with lower SN-FPN (ECN) connectivity, suggesting that reduced SN–FPN connectivity may mark heightened emotional reactivity in high-SPS individuals. This finding supplements the research linking sensitivity to vulnerability to psychopathology given our understanding of common theme of increased DMN connectivity in common mental illnesses and mood related disorders.

The research on creativity and sensitivity reveals the beginnings of a dual approach to creativity and psychopathology in terms of sensitivity, suggesting the usefulness of creativity therapy for individuals suffering from disorders characterized by reduced ECN connectivity and increased DMN connectivity. Creative functioning appears to form a natural mechanism enforcing balance of these network patterns, a balance that individuals with excessive DMN dominance may

internally orient towards as a means of self-regulation. These complementary insights begin to introduce us to the model we propose in this paper to link creativity, psychopathology, and sensitivity.

3.6. Lack of an Integrative Model

Despite substantial evidence linking sensitivity to both creativity and psychopathology, no integrative neural model currently explains when sensitivity leads to creativity, when it leads to psychopathology, or how environmental and regulatory factors determine this divergence. Aron's SPS framework characterizes sensitivity as a biological trait but does not incorporate large-scale network dynamics or creative cognition. Carson's shared vulnerability model (2011) highlights overlapping traits between creativity and mental illness but does not address network connectivity signatures or developmental pathways. Dąbrowski's model is the most powerful and cogent explanatory model given present network neuroscience research into these adjacent domains, but given researchers have not treated it with sufficient interest, his model presently remains in its latent psychological form without neurological integrations. Finally, neuroscientific models of creativity emphasize DMN–ECN–SN coupling but seldom incorporate sensitivity as a moderating variable and are limited in their discoveries. Likewise, psychiatric network models rarely consider sensitivity as a central causal factor.

This theoretical fragmentation has prevented the development of a unified account that situates sensitivity within emerging neuroscience of networks and within the dual pathways of creativity and psychopathology.

3.7. Neural Sensitivity as the Basis for Divergent Development

The network profile associated with SPS—DMN hyperactivity, SN hypersensitivity, and reduced ECN cohesion—creates an architecture that can support either creative flexibility or vulnerability to dysregulation, depending on environmental context, regulatory development, and stress exposure. Increased network flexibility enables imaginative thought, rapid associative access, and the integration of disparate ideas—core components of creativity. However, the same flexibility increases the system's susceptibility to instability: threat-labeled stimuli can dominate

attention, DMN activity can shift into maladaptive rumination, and insufficient ECN control can allow emotional responses to escalate.

Early-life environment plays a pivotal role in determining which developmental pathway predominates. Supportive, enriching environments appear to calibrate SN activity, strengthen ECN regulation, and convert heightened sensitivity into cognitive depth and creative potential. Conversely, chronic stress, trauma, or inconsistent caregiving may chronically upregulate SN threat detection, destabilize switching mechanisms, and produce the network signatures characteristic of anxiety and mood disorders, and similar intensities of inner experience may form developmental pathways into bipolar disorder and schizophrenia. Thus, sensitivity functions as a double-edged developmental substrate: its neural architecture magnifies both creative possibility and vulnerability to psychopathology. The same trait that amplifies cognitive richness and emotional depth can, under adverse conditions, amplify dysregulated network dynamics. Notably, however, given the neuroplastic nature of the brain, it is clear that alterations in the environment later in life can continue to impact the ever constant development of the individual. Neural sensitivity therefore serves as a crucial explanatory dimension linking creativity, psychopathology, and large-scale network function.

4. The Neural Sensitivity Model

4.1. Overview of the Model

Building on the relationship we have outlined between sensitivity, creativity, and psychopathology established in the prior sections, we propose a Neural Sensitivity Model that conceptualizes the mind as a fundamentally creative, adaptive, flexible, and environmentally responsive system with sensitivity as a dynamic developmental trait which determines degree of intensity of development. Highly sensitive individuals exhibit deeper processing of sensory, emotional, and cognitive stimuli due to a characteristic network connectivity pattern defined by increased DMN engagement, heightened SN responsivity, and reduced ECN connectivity (Liu et al., 2025; Acevedo et al., 2014). This triadic configuration results in greater internal elaboration, stronger affective resonance, and reduced top-down regulation—features that we have discussed which increase both the potential for creative cognition and the vulnerability to psychopathology. The reduced ECN connectivity may appear counterintuitive to creative processing, but the basic

underlying idea is that without the release of strong inhibitive elements of cognition, the generation of novel associative content is increasingly difficult. This corroborates with the observed higher prevalence of depression disorders and other forms of mental illness in creative individuals, supporting the idea that the seeming precondition for creative generation comes with an increased risk of pathology. It appears therefore from this perspective that creativity serves as a natural therapeutic agent for the sensitive DMN dominant individual — the forced inhibition to put into form the inner ideation increases ECN connectivity (a coupling mechanism necessary in creativity) as well as SN functionality as well as global connectivity and neural balance.

Notably, we hypothesize here that imbalance is a precursor to higher balance. The flexibility of the sensitive mind enables the unwiring of former pathways and the rewiring at different levels, or, the decoupling of former connectivity patterns and the coupling of new ones, a mechanism we hypothesize is necessary for creative development. Likewise, as is the case in nearly all functions, the learning of a different pattern involves the unlearning of a former pattern. Marines are first trained to walk and sit properly, singers decouple former vocal mechanisms to make way for new ones, and athletes regularly unlearn biomechanically inefficient movements to replace them with more effective ones. This adaptive mechanism of flexibility, foundationally a neural substrate, makes clear the developmental nature of the mind and its coming to terms with its environment.

Following this, we hypothesize that the DMN dominant network connectivity profile of sensitive individuals primes the neural architecture for the capacity of greater flexible reconfiguration both positively and negatively. Although research does not confirm the variability characteristic of individuals with SPS, it does confirm that sensitive individuals are more likely to be creative and that highly creative individuals demonstrate greater dynamic network switching and flexibility (Beaty et al., 2015, 2016; Liu et al., 2020; Zabelina & Andrews-Hanna, 2016). This cross section makes this point a valuable one for study. We hypothesize that the key to higher development is not the rejection of mental illness in a categorical sense, but the recognition of illness as a part of health. In the same way the immune system is injected micro-signatures of an illness so that the body can defend itself against the proper illness, the mind requires its own forms of "vaccination" against the existential terror of the world, especially in the case of individuals

more deeply impacted by these realities. This vaccination process takes the form of fostering emotion regulation processes, cognitive behavioral training, meditative training, as well as, and perhaps most importantly, a supportive community and family.

We theorize that sensitive individuals will demonstrate more intense network variability, flexibility, and connectivity patterns. Likewise, as sensitive individuals are more vulnerable to the environment and show higher cases of mental illness, we proposes that the pathological state is not in opposition to the creative one, but rather, indicative of the potential for creativity. The basic neural substrate that predisposes individuals to psychopathology is neural sensitivity, but this also demonstrates the capacity for greater network flexibility and potentiality, albeit with higher risk. This view has been held by others — Dąbrowski, Jamison, Otto Rank, Carl Jung — but has not been demonstrated in terms of network connectivity.

Our theory proposes to collapse the opposition between creativity and mental illness and view them in the same category as adaptive mechanisms. Sensitive individuals, as a result of a deeper impact by environmental stimuli, adapt more intensely than others, but, conversely, possess the capacity, to the degree of that intensity, for creative flexibility and higher network integration. As neurology research has supported the view that inner processing is central to the experience of reality, greater sensitivity indicates a more intense experience of reality. This intensity is not, by itself, positive nor negative, but rather, is a latent developmental potentiate.

Our theory also proposes a developmental view of these two false poles. That creativity can be viewed as a network balance of connectivity patterns and mental illness as a network imbalance of connectivity patterns indicates not that these are mutually exclusive, but, rather that they are interrelated. In order to achieve a different network connectivity pattern, the former pattern must be disrupted. This basic flexibility of the mind is the basis for network plasticity and reveals that the sensitive mind adapts more rapidly, that is, changes network connectivity patterns more readily. This basic flexibility indicates how the sensitive mind can be both more creative — which involves openness and dynamic flexibility between the networks — and more vulnerable to mental illness — greater flexibility and intensity of experience primes a mind for more pathological developments on the basis of environmental richness or poverty. Wealth here is not

material, though material things play a role, but a psychological wealth which manifests itself as support.

Furthermore, this developmental paradigm regarding network connectivity patterns indicates that disorder — imbalanced connectivity patterns — are important and sometimes even necessary (perhaps always) in the achievement of different connectivity patterns. Significant is the degree of disruption. Slight disruption can occur without much felt difficulty, but high degrees of disruption can be felt as traumatic and greatly alter neural architectures. The degree to which a disruption can impact the mind relates to the overall sensitivity/flexibility of the mind. Environmental support plays an enormous role in enabling effective achievement of differing connectivity patterns, but it is conceivable that certain individuals, as a result of some nexus of characteristics, are able to overcome negative environmental stimuli in the achievement of their own form of network balance. In addition, it is also clear that a sensitive individual can develop a network architecture into a position of high resiliency and flexibility, perhaps the integrated end state of neurological development — a mind that is capable of actively and efficiently adapting to its environment and self with a much lowered risk, or even erased, risk of the development of pathology.

Following from these two basic hypothesis, we find that there is a particularization that is necessary in the approach to mental illness and the individual as a whole. Rather than viewing treatment as a sui generic panacea for the mind, we conceptualize treatment as highly personalized and particular. Every individual's connectivity patterns and developmental trajectory are unique, and though higher balance is a perpetual aim, the means to achieving this balance will vary significantly on the basis of the individual. Treatment will not involve a medication regime as a panacea (though medication can be useful) but a total corrective approach that views the mind in totality. In terms of an analogy, we can use the example of an individual suffering from poor posture. In order to fix their particular postural makeup, programs personalized to them must be employed, and as the postural structure is corrected as a whole, other symptoms of poor posture — such as poor sleep, congested airways, digestive problems, and muscle fatigue — will remedy themselves. Treating these issues in isolation will only obfuscate the issue at hand and further worsen the individual's total condition. Patching a leak

only creates a new location to patch it — the entire structure must be viewed in reference to its parts.

Here, we broaden our scope of individuals as well. Though it has been demonstrated that particular neural factors predispose an individual to greater sensitivity, to us, it is clear that neural sensitivity, like all things, can be developed. Though it is true that certain individuals are born more sensitive than others, it is also clear that individuals can develop as a result of environmental richness, an environment richness that is external in the first case, but, eventually, as a result of development, becomes external in the second case. That is to say, the environment which impacts us in infancy and in the developmental phases of consciousness is external, as an individual develops conscious self-awareness, their experience of reality, as it has been shown, becomes increasingly determined by the inner perceptions of external stimuli.

Although the model is grounded in traits associated with high SPS, we hypothesize that all humans possess latent sensitivity, because sensitivity in its purest form is simply the capacity for experience to impact the mind. Neuroplasticity research consistently demonstrates that learning, trauma, and reflective practices reshape network dynamics (Kolb & Gibb, 2011), supporting the idea that sensitivity is a universal developmental substrate that varies in degree through both environmental and genetic causes. In this sense, sensitivity is the precondition for both mental illness and creativity: without the capacity to be impacted by experiences, neither meaning nor maladaptation could emerge. This indicates to us the basic malleable nature of our experience of the world, reinvigorating us to philosophical questions in the context of budding research in neuroscience as well as fostering the collective awareness of the importance of the curation of environments.

To return to our second hypothesis that the DMN dominant network connectivity profile of sensitive individuals primes the neural architecture for the capacity of greater flexible reconfiguration both positively and negatively, the context of our final hypothesis sheds greater light and generates support for this claim. Due to the reality that our inner perceptions determine our experience of the external world, an individual largely preoccupied with their inner world — a sensitive individual who's sensitivity has preconditioned this preoccupation — would

conceivably more intensely experience reality due to their increased internal processing and reduced external functioning. We find that the external world, as SPS theory and differential susceptibility theory demonstrate, are almost a maelstrom. With incredibly intense stimuli and a rapidly changing, terrifying, and tension filled world, individuals who are already predisposed to more intense experience are thrown into the deep end of the storm, especially without support. Thus, it is no coincidence that they drown. But, of course, as is a core theme of Western theology, there is always new life after death. The capacity for rapid alterations of brain connectivity patterns in mystical experiences, traumatic experiences, and psychedelic experiences indicate that not only can the brain change slowly but also rapidly. It is not within the scope of this paper to postulate evolutionary mechanisms underlying this neural dynamic of rapid configuration, but the authors have their insights.

To return to our first hypothesis that decoupling — imbalance — of former patterns is necessary for new coupling — balance — patterns, we generate further insight and support on this point. The capacity to be deeply impacted by society is by all means an inevitability and necessity. That one experiences tragedy is not out of place, but rather, completely in place in life. It has been said that life is a long sequence of farewells, but, likewise, life is a long sequence of hellos. Once again, we wish to reiterate that these two poles — imbalance and balance, illness and health, pathology and creativity, death and life — are not oppositional but co-creative and integrated together. Reframing our understanding of the brain in the context of these neurological findings is a necessity.

To return to this inter relation of decoupling and coupling, it is clear that the world is rapidly changing and that, in fact, perhaps the only constant is change. Given this perpetual change, it is simple to see how the brain adapts to meet the needs of its environment, a view that we have documented research for numerous times in this paper. In this sense, we can view mental illness, on a collective and individual level, as merely creative adaption to life that becomes maladaptive. Rollo May wrote that culture was created by individuals who could not accept the world the way that it was, and thus, who endeavored to make it acceptable to themselves. We find that pathology is one making themselves “acceptable” to the world, and health, creativity, is one making the world acceptable to themselves. Of course, according to our view, the world, rather

than an external construct, though it is, is in reality an inner experience. An individual can alter their environment, as their environment is within. Of course, inner change begets external change and vice versa. This interrelation is necessary to understand for our model. We as humans have with ease and without hesitation created the world around us, but we have failed to create ourselves in relation to our own creations. Ideally, treatment ought to seek only one thing — to enable an individual to create themselves.

Therefore, we frame the human organism as a fundamentally creative and adaptive entity whose neural architecture continually reorganizes itself through interactions with the environment, and which can eventually become part of its own reorganization process. The extent to which sensitivity leads to creative versus pathological outcomes is mediated largely by environmental quality, developmental context, regulatory support, and the genetic nexus. This model redirects the focus of psychopathology away from deficit-based frameworks and toward a developmental, plastic, and creative understanding of the human mind.

4.2. Core Hypotheses

Hypothesis 1 — Sensitivity is a Neural Developmental Substrate
Sensitivity is the fundamental developmental trait that determines how strongly the environment impacts the mind. It is rooted in a particular connectivity profile: elevated DMN engagement, heightened SN responsivity, and reduced ECN regulation. This triadic configuration underlies both the creative potential and psychopathological vulnerability of highly sensitive individuals.

Hypothesis 2 — The Same Neural Profile Underlies Both Creativity and Psychopathology
Creativity and mental illness arise from the same neural profile and rather than opposed states, are in fact two sides of the same coin. The DMN–SN–ECN profile that supports deep internal processing, emotional resonance, and associative richness also reduces executive connectivity and increases vulnesrabitliy to dysregulation due to intensity of experience and the higher flexibility of the neural structure. Creativity and psychopathology are both outcomes of neural sensitivity and are differentiated by developmental trajectory and environmental support.

Hypothesis 3 — Imbalance Is a Necessary Precursor to Higher Balance

Creative development requires the decoupling of existing network patterns in order to couple new ones. Periods of network imbalance—manifesting phenomenologically as distress, crisis, or symptoms—are not failures of the system but developmental disruptions and adaptational symptoms that are part of the process of reorganization. Pathology is not the opposite of health but a potential stage of psychological and neural restructuring.

Hypothesis 4 — Sensitive Individuals Possess Greater Neural Flexibility

The DMN-dominant architecture of sensitive individuals predisposes them to greater dynamic variability, rapid switching, and flexible reconfiguration across large-scale networks. This flexibility increases creative potential while simultaneously amplifying vulnerability to environmental deprivation, trauma, and instability. In essence, the same flexibility that enables creative integration under integrative conditions also enables maladaptive reorganization under adverse conditions.

Hypothesis 5 — Creativity Functions as a Natural Developmental Neural Corrective

Because creative processes require coordinated DMN-ECN coupling and effective SN mediation, they can serve as effective natural regulatory mechanisms for sensitive individuals. Creative engagement increases ECN connectivity, motivates global network integration, and fortifies inner self structures necessary for resiliency. Thus creativity is not simply an outcome of sensitivity but a compensatory and developmental regulator for it.

Hypothesis 6 — Sensitivity Is a Universal Developmental Trait

Although certain individuals are genetically predisposed to higher sensitivity, all humans possess latent sensitivity. This is because sensitivity is simply the capacity for experience to reshape neural connectivity patterns. Through enriched or impoverished environments, trauma, as well as various meditative, reflective, contemplative, and creative practices, individuals can become more or less sensitive over time. Sensitivity is both innate and developmental, the central mechanism behind neuroplastic change.

Hypothesis 7 — Environmental Context Greatly Influences Developmental Direction

The same neural substrate can lead toward creativity or pathology depending on environmental richness, relational support, developmental context, and internal regulatory structures. Since sensitivity amplifies environmental effects, environment is the key influence on development.

Hypothesis 8 — Pathology and Creativity Are Developmental Stages, Not Categories
Mental illness reflects maladaptive adaptations that emerge when flexible neural architectures reorganize under threat, excessive stress, deprivation, or excessive intensity. Creativity reflects the same adaptive process of reorganization but successfully toward global network integration. Because network plasticity requires disruption, periods of dysregulation may be necessary for eventual creative synthesis, a view corroborated philosophically and artistically in various first hand accounts. Pathology and creativity therefore exist on a developmental continuum rather than as oppositional entities.

Hypothesis 9 — Psychological Treatment Must Be Personalized and Holistic
Treatment cannot rely on generic symptom-based medication regimes. Effective intervention must target the particular-universal connectivity pattern. The same way that posture correction addresses whole-body alignment rather than isolated symptoms, a therapeutic model must integrate biological, cognitive, emotional, and relational domains to guide the individual toward network balance and flexible integration.

Hypothesis 10 — The Brain Is a Self-Creative, Environment-Shaping System
Human beings are fundamentally creative organisms whose neural networks continually reorganize themselves in response to both inner and outer stimuli, inner stimuli eventually being subject to conscious change. As self-awareness develops, the individual can increasingly become an active participant in shaping their own neural architecture. Mental illness, creativity, and development are expressions of the same underlying process: the mind continually creating and recreating itself in relation to the world it encounters—and eventually, the world it internalizes.

Overview of the Neural Sensitivity Model
The Neural Sensitivity Model proposes that sensitivity is the foundational developmental substrate that determines how intensely the environment shapes the mind, rooted in a

characteristic network profile of elevated DMN activity, heightened SN responsivity, and reduced ECN regulation. This tripartite architecture predisposes individuals to greater creative potential and vulnerability to psychopathology. Rather than treating creativity and mental illness as categorical opposites, the model reframes them as spiralistic outcomes of the same sensitive neural system, differentiated by developmental trajectory and environmental support. Central to the model is the idea that periods of network imbalance—manifesting as mental illness— are necessary phases of neural decoupling that enable later reorganization and integration, making pathology a potential precursor to higher-order flexibility as well as decoupling process which couples at a stage of disintegration.

Because sensitive minds exhibit greater network variability and plasticity, they adapt more intensely to environmental conditions. Creativity functions as a natural regulatory mechanism for the neural sensitivity architecture since it requires coordinated DMN–ECN coupling and effective SN mediation. This coordinated coupling naturally increases global integration which improves resiliency to decoupling disruptions developing into disintegrated states. Sensitivity is understood as both innate and developmental, a universal capacity that all people possess and the mechanism through which experience with the environment reshapes network connectivity patterns. Following from this view, clinical treatment must be individualized, targeting whole-network dynamics rather than isolated symptoms. Ultimately, the Neural Sensitivity Model reframes the human being as a self-organizing, self-creative organize whose identity and mental health are not givens but achievements that emerge through continual cycles of neural disruption, reconfiguration, and integration.

4.3. Reframing Categories

The Neural Sensitivity Model reframes multiple categories of understanding. Firstly, it reframes the human mind not as a machine prone to malfunction, but as a dynamic, creative system continually reshaping itself in response to experience with the environment. Sensitivity reveals that the brain is not only a passive processor but can become an active creator of its internal environment, a process of creation which extends to the external world in terms of identity, relationships, vocation, and communal engagement. Everyday examples—from musicians acquiring skill through deliberate environmental structuring to athletes shaping physical and

cognitive capacities through training—illustrate how both external and internal stimuli sculpt internal architecture. Indeed, unlearning is a way of learning, as any athlete or musician can tell you — in order to learn new patterns of engaging with the body and the world, old patterns must be destroyed first. Neuroscience reinforces this view: experience consistently drives structural and functional reorganization across the lifespan (Draganski et al., 2004; Bassett et al., 2011). There is no end to growth.

The Neural Sensitivity Model significantly challenges the traditional DSM model for interpreting mental illness in several ways. Whereas the DSM treats disorders as discrete, symptom-based categories defined by dysfunction, the Neural Sensitivity Model reframes these definitions as developmental expressions of underlying network dynamics. Instead of locating pathology in fixed structures, it situates them within oscillatory processes of decoupling and recoupling across the DMN, ECN, and SN. That is, it locates illness within the scope of health. In this view, mental illness is not a stable entity but a perpetual achievement of a state of heightened neural integration. This continual integrative function occurs as a result of the mind's unending project of reorganization in relation to environmental stimuli. This stands in stark contrast to the DSM's categorical approach, which restricts the concept of individual variability and the possibility that certain maladaptive states are precursors to higher-order integration. The Neural Sensitivity Model therefore shifts the interpretive frame from disease classification to developmental trajectory, emphasizing how sensitivity, environment, and network plasticity converge to shape psychological outcomes and redefine ideas of health and illness.

Within the Neural Sensitivity Model, pathology is reinterpreted as a developmental phase of network reorganization. States classified as "disorders" in the DSM—marked by dysregulation, instability, or heightened affective intensity—are understood here as manifestations of temporary decoupling and maladaptive plasticity within large-scale networks. These states emerge when a highly sensitive neural architecture attempts to recalibrate itself in response to overwhelming environmental conditions or internal demands. Rather than signaling the failure of the system, pathology reflects the system's effort to adapt, reorganize, and ultimately stabilize at a higher level of integration in relation to the individual and the environment they inhabit. This reframing dissolves the false boundary between illness and health through a continuum of development and

positioning pathology as a possible precursor to creative reintegration and increased cognitive-emotional capacity.

Conversely, creativity in the Neural Sensitivity Model is conceptualized as the optimal functional expression of a flexible, adaptive, and sensitive mind. Creative cognition arises when the DMN, ECN, and SN achieve a dynamic balance: the DMN generating rich associative content, the ECN shaping these associations into coherent form, and the SN orchestrating fluid transitions between internal and external modes of processing. At this level of integration, the individual demonstrates a high generation of associative thought, resilient emotional regulation, executive conceptual clarity, and adaptive responsiveness to the environment. Creativity here is not a special talent or an outlier state reserved to genius — it is the continual conscious cultivation of higher global network coordination possible for everyone. Because sensitivity amplifies the impact of experience and increases the capacity for neural restructuring, the sensitive individual possesses a unique potential for reaching a higher integrated creative state that comes with the increased vulnerability for pathology. Thus, creativity is reframed not as an exception but as the teleology of the human mind: the state toward which its oscillations, disruptions, and reorganizations are consciously directed.

The Neural Sensitivity Model also provides a framework for understanding the rising prevalence of mental illness in modern Western populations. Modern environments change at a pace that far exceeds the evolutionary and developmental rhythms the brain has evolutionarily dealt with. Sensitive individuals — already predisposed to deeper processing and heightened responsiveness — are particularly vulnerable to this acceleration. Rapid shifts in social norms, digital hyperstimulation, economic instability, and the erosion of communal structures produce a constant barrage of salient stimuli and disintegrative environmental conditions not only without corresponding external structures for regulation or integration, but the greater evaporation of these structures. Under these conditions it is only natural that the neural system is repeatedly forced into maladaptive reconfigurations, where decoupling occurs without sufficient environmental support to guide recoupling toward balanced integration and in which collective generations suffer from existential dilemmas of meaning and self-creation. The result is an increase in anxiety, depression, attentional disorders, and identity fragmentation—not because

individuals are “weaker,” but because the environment demands levels of neural flexibility and stability that it simultaneously undermines. The only recourse it provides is sedation — the reduction of sensitivity so as to reduce the possibility of pathology but also creativity, thus opposing the general project of human. In this view, widespread psychopathology is not an epidemic of defective minds but a predictable consequence of disintegrating developmental structures, where the world changes faster than individuals can reorganize themselves in relation to it. We have as a species learned all too readily and without hesitation how to transform the world around us, only, we are failing to transform ourselves in relation to the world we have created.

Lastly, the Neural Sensitivity Model reframes the idea of “human” as an unfolding, perpetually self-recursive creative project. In contrast to static or purely biological conceptions of personhood, this model understands the human organism as one whose identity, cognition, and emotional architecture are continually reconstituted through cycles of neural decoupling and recoupling which are mediated by the self-other relation of the environment. Because sensitivity amplifies the degree to which experiences reshape the brain, human development becomes a process of ongoing self-creation in response to an ever-changing and increasingly complex environment. The individual is not merely shaped by external forces but, through reflective awareness and adaptive restructuring, becomes an active participant in shaping their own neural and psychological form, either consciously or subconsciously. In this sense, to be human is to inhabit a dynamic trajectory in which pathology, creativity, and health are not endpoints but phases within a recursive spiral of becoming. The mind is both the product and the producer, development and developer, creation and creator, of its own evolution, engaged in the continuous project of creating itself.

4.4 Testing hypotheses

Given the developmental nature of our hypotheses and the inherent difficulty of capturing variability in large-scale networks, it is necessary to design studies that can both isolate specific connectivity patterns and track their change over time. Because the developmental substrate we propose unfolds across long windows and is subject to substantial individual variability, we

outline two core testing regimes that can lay the empirical foundation for further, more refined investigations.

Our first, foundational claim is that neural sensitivity is characterized by a specific network architecture—enhanced DMN activity, increased SN–DMN coupling, reduced SN–ECN connectivity, and reduced ECN engagement. To test this, we propose a study examining the connectivity patterns of the tripartite network across individuals who vary along the SPS dimension. Participants would complete tasks that project emotional, cognitive, existential, and affective stimuli while undergoing fMRI, allowing us to assess network flexibility and responsivity as a function of sensitivity to stimuli. In addition, we propose measuring the same individuals during non-creative, externally focused tasks (e.g., executive cognitive control tasks) to observe the intrusion of DMN ideation and reduced ECN recruitment, as well as during creative tasks (e.g., idea generation and evaluation) to characterize network variability and coordination under conditions of creative engagement. Comparing network dynamics across low, medium, and highSPS participants in these different task contexts would allow us to test whether the proposed sensitivity architecture reliably distinguishes sensitive individuals and simultaneously predicts both elevated vulnerability and creative potential. Limitations of this regime include the difficulty of isolating causal variables in cross-sectional designs, the influence of momentary mood and reactivity on network measures, and the challenge of decoupling sensitivity from overlapping traits such as neuroticism or introversion.

Our second testing regime seeks to support the developmental claim that mental illness can serve as a positive, reorganizational function in sensitive individuals through the corrective mechanism of creativity. Here, our aim is to demonstrate that structured creative practices confer particular benefits for sensitive individuals, both in terms of symptom improvement and network integration. Because sensitive individuals are more likely to experience mental illness and to engage in creative processes, we propose a longitudinal or intervention-based design in which participants high and low in SPS—with varying diagnostic profiles ranging from no diagnosis to mood or anxiety disorders—undergo creativity-oriented therapy or training. Creative interventions might include guided artistic production, expressive writing, or other treatment modalities that require oscillation between free ideation and structured refinement. Across time,

we would monitor changes in network connectivity, with particular focus on DMN–ECN coupling, indices of neural flexibility and adaptability, and markers of global integration and resilience. Our key prediction is that creative training will increase ECN connectivity and regulatory capacity while preserving or even enriching DMN generativity, especially in high-SPS individuals, thereby supporting the notion of creativity as a developmental neural corrective for sensitive minds.

If supported, this second regime would provide convergent evidence for several of our core hypotheses: that mental illness can be understood as a developmental stage oriented toward higher neural integration; that creativity functions as a corrective mechanism; that psychopathology and creativity are dual expressions of a shared sensitivity-based architecture; that sensitive individuals possess more flexible and adaptive neural systems; and that pathology and creativity represent stages along a developmental continuum rather than fixed categories. However, the limitations of this methodology are considerable. Differentiating individuals on the basis of sensitivity will require multiple, convergent measurement tools to form adequately distinct testing groups. The long-term nature of the design also makes it difficult to control for external environmental influences—such as life events, social support, and cultural context—which are especially consequential for sensitive individuals. More controlled, future work could partially address these constraints by examining smaller groups in curated environments, systematically varying exposure to creativity-oriented interventions and support structures while tracking network connectivity patterns over time.

These two testing regimes establish the empirical groundwork for evaluating the Neural Sensitivity Model and set the stage for the next section, where we outline how these principles can inform novel therapeutic approaches and new network-oriented treatment plans.

4.5 Treatment methods

Recognizing sensitivity as a core driver of neural development offers a powerful framework for rethinking treatment. Because sensitive individuals process experience more deeply, they are also more responsive to positive therapeutic interventions (Belsky & Pluess, 2009). In our model,

treatment is not conceived as symptom management or categorical diagnosis treatment, but rather as the process of developing integrative network reorganization. This process seeks to guide individuals from imbalance toward higher structural integration across the DMN, ECN, and SN.

Although numerous existing interventions already align with this developmental framework, we emphasize that medication should be used only as a temporary support in cases of severe dysregulation or rigidified network patterns. Given the brain's lifelong neuroplasticity, even highly rigidified connectivity profiles can be altered through learning, relational experience, and creative engagement. Adjacent research on psychedelics and mystical experiences further demonstrates the possibility of rapid, large-scale reconfiguration. Since pharmacological treatments target downstream symptoms rather than upstream causes we find that they are ineffective long term means of treating the individual, though they may treat particular symptoms. We approach medication as a causal treatment that fails to recognize the cause. If an individual suffering from poor posture develops dehabilitating digestive issues, these digestive issues can be treated in an isolated manner which, although treating the symptom, do not address the root cause. Medication for the mind is the same if not even more so divorced from the cause due to the fact that the brain is essentially neuroplastic, remains neuroplastic, and can become more neuroplastic. Medication ought rarely be used long term, and the aim of treatment ought always be a holistic and total one that views the individual as both a whole and a collection of parts — a particular universal — with the aim of the restoration and achievement of higher global integration. The clearest empirically measurable indicator of imbalance and progress in this view to measure the efficacy of treatment is large-scale network connectivity with an optimal state not mere global integration — as the brain is never static — but adaptive and flexible variability which continually oscillates towards higher integrations

There are numerous treatment methodologies that reflect this perspective.
Creative Practice–Centered Therapies.
Guided expressive writing, visual art, music, improvisation, and related techniques that require oscillation between spontaneous ideation (DMN) and structured refinement (ECN) directly strengthen the DMN–ECN coupling that underlies adaptive creative integration. These practices

also engage the SN in mediating transitions between internal and external modes of processing. Creative engagement therefore functions as a natural corrective mechanism, especially for sensitive individuals whose baseline profiles favor generativity but often lack executive regulatory structure.

Emotion Regulation and Mindfulness-Based Interventions.
Mindfulness-based stress reduction, acceptance and commitment therapy, and contemplative practices cultivate attentional stability, improve SN responsivity, and decrease maladaptive DMN rumination (Tang, Hölzel, & Posner, 2015; Brewer et al., 2011; Moore, Gruber, Derose, & Malinowski, 2012; Lutz, Slagter, Dunne, & Davidson, 2008). Through repeated practice, these methods help sensitive individuals manage the intensity of their internal experience and promote smoother network transitions.

Cognitive Behavioral and Metacognitive Therapies.
CBT, MCT, and related interventions strengthen ECN recruitment and top-down control, providing structural integration for individuals whose regulatory systems may be under-engaged (DeRubeis et al., 2008; Goldin et al., 2014; Siegle, Thompson, Carter, Steinhauer, & Thase, 2007; Normann, van Emmerik, & Morina, 2014). These therapies help the mind reinterpret stimuli, reduce reactivity, and develop more flexible and resilient structures of self and meaning.

Somatic and Embodied Therapies.
Somatic experiencing, yoga, breathwork, posture correction, and other embodied interventions regulate autonomic arousal and refine the SN's detection of bodily salience cues (Payne, Levine, & Crane-Godreau, 2015; Streeter et al., 2012; Farb et al., 2013; Critchley & Garfinkel, 2017). Because sensitivity amplifies interoceptive and affective signals, somatic methods support greater balance between internal and external attention and reduce physiological overwhelm.

Relational and Community-Based Therapies.
Group therapy, family systems approaches, and relational psychodynamic treatments provide consistent external structural support necessary for network integration (Yalom & Leszcz, 2020; Pinsof, 2015; Schore, 2012; Lemma, Target, & Fonagy, 2011). Sensitive individuals'

developmental trajectories are disproportionately shaped by their relational and environmental contexts; supportive relationships enhance regulatory capacity, emotional integration, and network stability.

Narrative and Existential Therapies

Meaning-making frameworks help sensitive individuals integrate intense internal experiences into coherent autobiographical narratives. These methods capitalize on DMN generativity while reducing fragmentation, strengthening identity, and promoting long-term psychological continuity (Pennebaker & Seagal, 1999; McAdams, 2001; Park, 2010; Adler et al., 2016).

Psychedelic-Assisted Therapies

When administered safely and accompanied by integration work, psychedelics can facilitate rapid decoupling and recoupling of large-scale networks but had the added cost of greater integrative work needed to provide a conceptual grounding for accelerated neural development (Carhart-Harris et al., 2012; Carhart-Harris & Friston, 2019; Watts & Luoma, 2020; Roseman, Nutt, & Carhart-Harris, 2018). These interventions should be treated as catalysts rather than cures and must be embedded within structured therapeutic environments.

Holistic Lifestyle Interventions.

Sleep regulation, nutrition, exercise, creative routines, and environmental curation form a basic and necessary developmental foundation for neural integration (Walker, 2017; Gómez-Pinilla, 2008; Hillman, Erickson, & Kramer, 2008; Leder, 2005; Kaplan, 1995).

Precision TMS (PTMS).

Emerging PTMS approaches target stimulation not by diagnostic category but by individualized connectivity patterns (Cole et al., 2020). Our model strongly supports such techniques by reframing psychopathology as network-level imbalance rather than localized molecular deficit. With further development, PTMS may become a promising adjunct for individuals whose networks are significantly dysregulated, offering a top-down means of restoring connectivity patterns that other treatments aim to reorganize more gradually.

Summary

These treatment methodologies demonstrate how a sensitivity-centered, network-developmental framework reorients clinical practice away from symptom suppression and toward the cultivation of adaptive neural integration through a framework of network connectivity patterns. Rather than treating pathology as a fixed category, this model positions therapeutic work as a guided process of recalibrating large-scale networks through creative engagement, embodied regulation, relational support, and environmental enrichment, preparing the individual to effectively encounter the world and themselves, and, more importantly, consciously embody these practices in their own life. Medication and neuromodulatory tools retain a role but really only as temporary stabilizers in extreme cases. This holistic perspective accompanies the need for treatments that address the individual as an integrated particular-universal system.

4.6 Conclusion

The Neural Sensitivity Model offers a unified view of the mind as a dynamically rewiring, environmentally responsive, and fundamentally creative system. Sensitivity emerges as the foundational developmental substrate that amplifies the impact of experience, potentiating both heightened vulnerability to psychopathology and an exceptional capacity for creative reorganization. By situating creativity and mental illness within a shared tripartite network architecture — elevated DMN engagement, heightened SN responsivity, and reduced yet plastic ECN regulation — the model reframes pathology and mental health. Periods of imbalance become necessary phases of decoupling that, under supportive conditions, can be transformed into higher-order integration and flexibility.

Within this framework, creativity functions as the optimal expression of a sensitive, well-integrated network configuration and as a primary corrective mechanism by which dysregulated systems can be reorganized. Treatment, accordingly, is reconceived not as symptom suppression but as the guided lifestyle cultivation of adaptive network configurations through creative practice, internal conscious regulation, relational support, and enriched environments. The ultimate task of development is to enable individuals to participate consciously in their own reconfiguration, becoming active co-creators of their neural, psychological, and relational

worlds. Thus, the ultimate aim of therapy and human development reveals itself: that one becomes their own creator.

With this foundation established, we now turn to the limitations of the model, the empirical and conceptual gaps that remain, and the considerations necessary for refining and testing this framework in future research.

5. Considerations, Limitations, Gaps, and Future Directions

5.1. Conceptual Considerations and Theoretical Constraints

Despite its integrative optimism, the Neural Sensitivity Model remains a theoretical framework that combines constructs from personality psychology, network neuroscience, and clinical theory in ways that are still evolving. The central construct of "sensitivity" overlaps with existing dimensions such as neuroticism, introversion, openness, and negative affectivity, adding to the debate surrounding the term. Our emphasis on a DMN-dominant, SN-reactive, and ECN-reduced profile is grounded in converging but not definitive evidence which can risk oversimplifying a complex set of neural and psychological phenomena. Moreover, our developmental interpretation—that pathology, creativity, and health are phases of one process—competes with other models that conceptualize some disorders as primarily degenerative, rather than reorganizational. These conceptual tensions, and many others, will need to be further investigated and clarified.

The model also carries a philosophical stance that reframes the human as a self-creative, recursive project. While this view is consistent with existential, phenomenological, and constructivist traditions, it may not align with more strictly biological or reductionist approaches. There is a risk of inadvertently romanticizing suffering by framing mental illness as a potential gateway to higher "development." Our intention is not to glorify pathology but to recognize that in liue of neuroplasticity research, states of profound distress may reflect the mind's attempt to reorganize under intense pressure, thus indicating a basic adaptive strength that has ossified into maladaption. Affirming developmental possibility without minimizing suffering is a central conceptual and ethical challenge.

5.2. Empirical and Methodological Limitations

Empirically, the model faces several substantial constraints. Firstly current network neuroscience methods such as resting-state and dynamic connectivity analyses are limited by measurement complexities natural to the measurements of variability and flexibility (Hutchison et al., 2013). Metrics of flexibility or variability can be interpreted as adaptive plasticity, instability, or simple measurement error, depending on analytic choices, which underscores the need for long term study (Zalesky et al., 2014). Second, the kinds of longitudinal, multi-wave, and intervention-based studies needed to test developmental trajectories across long periods of time are expensive, hard to plan, and difficult to maintain (Ferrer & McArdle, 2010), though, there has been useful development in this realm with encouraging multi-modal longitudinal approaches to mental illness (Westhoff et al., 2024). Many of the key processes we highlight (e.g., crises, creative breakthroughs, mystical experiences) are hard to capture prospectively and ethically not to mention in a measurement setting. Likewise, the vastness of the possible confounding variables make any form of developmental measurement across a long of period of time complex when attempting to capture a dynamism such as network connectivity patterns where life events play such a pivotal role and cannot so easily be controlled for.

A further methodological limitation is the difficulty of operationalizing creativity and psychopathology in ways that accurately reflect their depth and complexity (Kaufman & Beghetto, 2009). Standard creativity tasks capture only narrow aspects of generativity and rarely reflect sustained artistic or intellectual work, while diagnostic categories imperfectly capture lived experience and developmental nuance (Kendler 2016). Integrating behavioral tasks, real-world outputs, and clinical observation will be necessary but complex. Finally, disentangling sensitivity from overlapping traits and isolating its unique contribution to network dynamics requires large samples, multi-modal methodologies, and sophisticated statistical modeling, all of which pose substantial practical demands.

5.3. Gaps and Priorities for Future Research

Several key gaps define the next steps for empirical work. First, foundational studies must refine and validate the proposed sensitivity-related network architecture, distinguishing it from

overlapping constructs and demonstrating its relationship to both creative potential and psychopathology. Second, longitudinal work is needed to map developmental trajectories in sensitive individuals, testing whether periods of network imbalance can, under certain environmental conditions, predict later integration and creative flourishing rather than chronic disorder. Third, intervention studies centered on creativity, emotion regulation, and relational support must examine whether these modalities systematically alter network dynamics in the directions proposed by the model.

Future research has many new major frontiers to explore. Given that the individual is altered by the environment and that the environment alters the individual, we find that this research integrates forms a possible bridge into various other domains of science. Notably, we can begin to connect individual development to collective change, applying the developmental dynamic of our brain — which shapes and is shaped by our environment — to other interdisciplinary domains of study, motivating a transdisciplinary perspective.

5.4. Integrative Outlook

Despite these limitations, the Neural Sensitivity Model offers a promising framework for rethinking the relationship between creativity and psychopathology. By connecting sensitivity, network dynamics, environmental context, and therapeutic practice within a single developmental framework, it opens a space for research that creates the human as a project of continual reciprocal development both individually and collectively. The challenge for future work is to translate this conceptual coherence into rigorous empirical paradigms and ethically grounded clinical applications, so that all people can be supported in the difficult but necessary work of becoming the creators of their own lives.

6. Conclusion

The present work has sought to reframe the link between creativity and mental illness through the framework of network neuroscience and the emerging research into sensitivity. Across our review we found that the same neural dynamics that denote creativity also increase vulnerability to psychopathology. The Neural Sensitivity Model relates creativity and psychopathology as

parallel expressions of a neural architecture denoted by increased DMN engagement, heightened SN responsivity, and reduced but flexible ECN regulation.

By grounding sensitivity in large-scale network organization, we have argued that creativity and psychopathology are not categorical opposites but correlated developmental trajectories emerging from the basic plasticity of the brain. Periods of imbalanced network dynamics are stages of decoupling that allow for new patterns of connectivity to develop. Within supportive conditions, these disruptions can reorganize into higher network integration and creative functioning while under impoverished conditions or chronic stress, the same mechanisms may solidify into maladaptive patterns as means of adapting. Sensitivity which leads to more intense inner experience and network flexibility becomes the explanatory link between vulnerability and possibility.

This reframing carries profound implications in various domains. Clinically, it shifts our focus away from symptom suppression and towards holistic positive development. This development appears as the cultivation of environments, practices, and relational structures that support individuals toward integrative network balance. Creativity becomes both a corrective mechanism of the mind and its telos. Therapeutically, this models presents a multi-modal approach that recognizes the individual as a dynamically reorganizing system whose patterns cannot be reduced to diagnostic categories or isolated symptoms but must be viewed in relation to the whole. A sensitivity-centered model requires personalized treatment, environmental support, and therapeutic methodologies such as creative, cognitive, somatic, relational, and contemplative therapy which all foster global network integration

At a philosophical level the Neural Sensitivity Model presents a redefinition of the concept of human. If sensitivity is the foundation of both pathology and creativity, and if neural architecture remains malleable across the lifespan, then the human organism is best understood as a self-recursive, self-creative being that continually reshapes both its internal and external world in response to both inner and outer conditions. Pathology, creativity, crisis, and growth become phases within a single developmental arc rather than distinct static categories. The mind is sculpted both by the world and itself, and more importantly, contains the potential to become the

sculptor of both the world and itself. Sensitivity to this inner and external dynamic relationship is the basic mechanism by which human beings generate meaning, adapt, suffer, transform, and create.

The Neural Sensitivity Model does not claim finality and remains introductory for the purposes of future research, empirical refinement, and conceptual reframing. It, like the mind, is recursively plastic. Its hypotheses require rigorous testing across cultures, developmental stages, and environmental contexts, and its therapeutic implications demand collaboration between neuroscientists, clinicians, artists, philosophers, and those with lived experience. However, even at this early stage, the model provides a compelling framework for bridging together previously disparate domains: creativity, psychopathology, sensitivity, neuroplasticity, and development.

Ultimately, this paper argues that to understand creativity and mental illness, we must understand sensitivity; to understand sensitivity, we must understand the network architecture of the brain; and to understand the network architecture of the brain, we must understand the human being as a creature in continuous recursive and relational creation. In recognizing this we can refine our scientific understanding of mind and development and also return agency to individuals whose depth of experience has long been pathologized rather than understood. The sensitive mind is not a broken mind. It is an unfinished one, always in motion, always reorganizing, always capable of becoming something more than it was before. The task of science and therapy alike is not to constrain or sedate this processual trait, but to support its development. That is, to create the conditions in which an individual can become the conscious creation and creator of itself and its environment.

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