ENCEPHALOS 62, 69-76, 2025

Delusional Beliefs: Neurobiology and Recent Research Findings

Nikolaos Soultanis, Athanasios Tselebis, Dionisios Bratis, Argiro Pachi[1]

Summary

Delusional beliefs are a central feature of psychotic disorders and have been the focus of intensive investigation in the field of neuroscience. This article examines theoretical models of delusion formation, with emphasis on the aberrant salience hypothesis and the prediction error framework. Findings from neuroimaging and computational psychiatry are presented, supporting the link between neuronal activity and delusional ideation.

Keywords: delusional beliefs, psychosis, dopamine, neuroimaging, computational psychiatry

[1]Psychiatry clinic, ''Sotiria'' General and Thoracic Hospital of Athens, Athens, Greece

*__Corresponding Author:__
Mesogeion Avenue 152, 11527, Athens
Email: soultanisnikolaos@gmail.com

Introduction

This review aims to examine theoretical models that seek to elucidate the pathophysiology of delusional beliefs, focusing primarily on primary psychotic syndromes. According to the Aberrant Salience Theory, dysfunctions in the reward system and dopaminergic activity lead to the misinterpretation of irrelevant stimuli as significant, thereby promoting the development of delusional beliefs. Similarly, the Prediction Error Theory highlights the brain's inability to adapt beliefs based on sensory data, thereby creating conditions for distorted reality assessments and the formation of delusional constructs.

Neuroimaging research supports the association of delusions with activation in networks such as the salience network, which includes regions like the prefrontal cortex, anterior cingulate cortex, and striatum. Disruptions in dopaminergic and glutamatergic neurotransmission play a crucial role. Experimental evidence also supports the hypothesis of impaired prediction error processing within neural circuits of the frontal cortex, limbic system, and striatum.

The synthesis of these theories may offer a more comprehensive understanding of the neurobiology underlying delusional beliefs, which could be further advanced by research in the field of computational psychiatry.

Definition and Characteristics

Delusional beliefs are erroneous convictions maintained with absolute certainty and are impervious to counterarguments or evidence to the contrary. The DSM-5 identifies various types of delusions based on their content, such as grandiose, religious, persecutory, erotomanic, nihilistic, somatic, referential, and associative delusions. They are classified as either bizarre or non-bizarre depending on whether they stem from common life experiences, their plausibility, and their comprehensibility within the individual's cultural context. Bizarre delusions involve perceived loss of control over one's mind and body, such as thought broadcasting and experiences of passive influence (American Psychiatric Association, 2013).

The Problem of Contemporary Neurobiology

Contemporary literature indicates that no clear and universally accepted neurobiological mechanism has yet been identified for the formation, expression, or progression of delusional beliefs. The pathophysiology of this disturbance in thought content remains vague, as most literature focuses primarily on its phenomenology (Corlett et al., 2010). The aim of this review is to highlight a possible theoretical model of the neurobiology and pathophysiology of delusions, based on international literature. The data pertain primarily to individuals suffering from primary psychotic syndromes. The literature search was conducted via the platforms PubMed, Scopus, and Google Scholar

A Known Theory on the Formation of Delusional Beliefs: The Theory of Aberrant Salience

The Theory of Aberrant Salience posits that delusional beliefs observed in psychotic disorders arise from a dysfunction in the attribution of significance to various environmental stimuli.
It is widely accepted that dopamine plays a central role in the brain's reward and reinforcement systems. According to the motivational salience hypothesis, dopamine regulates the transformation of external stimuli into neuronal representations. Neutral information, through dopamine's action, becomes either attractive or aversive. The mesolimbic dopaminergic system appears to assign importance to external events or stimuli (attribution of salience). The individual then focuses attention on these and shapes goal-directed behavior to obtain reward or avoid punishment.

In psychosis, dysfunction in dopaminergic transmission is observed, where dopamine is secreted without an external trigger. This leads to the incorrect attribution of importance to seemingly insignificant stimuli, which may relate to external objects or internal representations. Consequently, the human brain attempts to cognitively interpret these new, pathologically significant stimuli through the formation of new delusional beliefs (Kapur, 2003).

Dopaminergic hyperactivity in the striatum, which is present even before the onset of psychosis, attri butes heightened and excessive significance to perceptions and thoughts. This aligns with many patient reports during the early phase of psychotic episodes:

“I developed a heightened awareness... My senses became sharper. I was fascinated by the small, insignificant things around me.”
“My senses felt vivid.”
“I felt like it all had tremendous significance.”
“It was like I was putting together the pieces of a puzzle.”
“My capacity for aesthetic appreciation and heightened sensory receptivity had become intensified. I had felt that same intensity before, when I was 'normal,' but those episodes were shorter and also blended with other feelings.” (Mishara & Fusar-Poli, 2013)

Within this framework, delusional beliefs represent a top-down cognitive effort by the individual to explain the experiences of aberrant salience. Here, one can see the role that individual experiences and cultural background play—for example, persecutory delusions involving the police in an urban dweller versus beliefs of attack by a shaman in a tribal African setting (McKenna, 2017).

Antipsychotics reduce the salience of unusual experiences. While they inhibit dysfunctional dopaminergic transmission, they do not radically alter it. As long as the individual continues antipsychotic treatment, the delusional beliefs are not eliminated but become more peripheral in the person's mind. Thus, when medication is discontinued, the same ideas and perceptions that were once part of the symptoms regain significance and once again dominate thought and behavior. The delusions then re-emerge strongly (Kapur, 2003).

Neuroimaging Evidence Supporting the Theory of Aberrant Salience

Neuroimaging studies involving salience processing tasks have revealed that dopaminergic mesencephalic neurons, their projections to the striatum, and frontotemporal and frontoparietal cortical regions are involved in the allocation of significance to stimuli. The salience network appears to be located in the anterior insular cortex, the dorsal anterior cingulate cortex (dACC), the supramarginal gyrus of the parietal cortex, the anterior prefrontal cortex, the striatum, the thalamus, and the cerebellum. This network processes important external and internal information and is activated in multiple cognitive and emotional processes.

Recent studies have identified a dual-subsystem structure within the salience network, located in the dorsal and ventral anterior insula. In a resting-state fMRI study comparing healthy individuals to patients with schizophrenia, the latter showed reduced functional connectivity in the dorsal salience subsystem, specifically between the dorsal anterior insula (dAI) and frontoparietal regions (e.g., paracingulate segment of the midcingulate cortex, middle frontal gyrus, precentral gyrus, and supplementary motor area). These findings are associated with difficulties in attention and processing external environmental information. Conversely, increased connectivity was observed in the ventral subsystem, particularly between the ventral anterior insula (vAI) and the subgenual anterior cingulate cortex (sgACC). This heightened connectivity is linked to intense emotional responses and may be related to symptoms such as delusional beliefs (Leonidas Mantonakis et al., 2024).

A systematic review of fMRI studies on individuals at high risk of psychosis and those already diagnosed revealed associations between aberrant salience and the ventral

striatum and insula—regions involved in reward processing and salience attribution. Aberrant salience has also been linked to source monitoring errors, a cognitive phenomenon involving misattribution of the origin of information in old memories. Source monitoring is associated with the medial prefrontal cortex and the superior and middle temporal gyri. These two cognitive functions appear to involve overlapping neural circuits, including the anterior cingulate cortex and hippocampus, which are implicated in both salience attribution and source monitoring, potentially influencing the development of delusional beliefs (Kowalski et al., 2021).

The theory of predictive errors and Bayesian predictive mechanisms

Another theory found in the literature is the theory of the Bayesian brain. It is mostly encountered in the field of computational neuroscience and computational psychiatry. According to this theory, the human brain can be likened to a statistical machine with hierarchical functions that attempts to predict present and future events based on prior experiences. The brain generates probabilities for hypotheses that best explain sensory data, thereby interpreting both the external and internal world. These hypotheses are continuously updated based on mathematical rules of probability. These rules correspond to Bayesian algorithms. The aim is to optimize prediction of the environment and reduce surprise and uncertainty, which corresponds to the computational and thermodynamic efficiency of the brain. Uncertainty may relate to the ambiguity of beliefs derived from a person's experiences as well as the uncertainty of environmental stimuli that is actually received (Friston, 2010).

To understand how this model works, one must imagine a person's beliefs about a stimulus and the actual stimulus as Gaussian normal distributions. The width of the expectation curve represents the uncertainty of the belief—a wider curve indicates less certainty about the reality. The discrepancy between the expectation and the actual sensory input is called the prediction error. Based on this error, the individual updates their beliefs and forms a new estimation about reality (Sterzer et al., 2018).

The comparison and storage of information concerning beliefs and reality may occur through oscillations of cortical neural networks. Neurons in these networks fire synchronously and oscillate at frequencies detectable via EEG (Corlett et al., 2010). When a significant mismatch exists between a belief about an event and the actual event—a prediction error—learning is triggered in order to update predictions and focus attention on the new inputs. These functions correspond to hierarchical neural levels ,where higher levels send top-down signals to lower levels to predict incoming sensory information, allowing the individual to act accordingly. Prediction signals may be transmitted top-down via NMDA-mediated glutamatergic transmission (Sterzer et al., 2018). In contrast, prediction errors are believed to be transmitted bottom-up via AMPA-mediated glutamatergic transmission. The properties of NMDA and AMPA receptors play a key role in the timing and coordination of signal transmission across cortical networks (Durstewitz, 2009).

Higher hierarchical levels, such as the prefrontal cortex, update their predictions to minimize future prediction errors. The extent of these updates depends on the precision of the prediction error transmitted. Precision here refers to the reliability or significance of the error. A prediction error with low perceived precision will have less influence on subsequent updates. In uncertain environments, prediction error signals are treated as less reliable, and thus have a diminished impact on belief revision.

The precision of a prediction error is influenced by the person’s prior experiences and the certainty of their beliefs. It can be encoded via the modulation of synaptic gain, i.e., the strength of the postsynaptic neuron’s membrane potential relative to the presynaptic membrane. Specifically, this involves changes to the postsynaptic gain of pyramidal neurons in the superficial cortical layers (Feldman & Friston, 2010), mediated partly by NMDA receptor activity. Additionally, precision may be modulated by dopamine and acetylcholine depending on the hierarchical level involved (Sterzer et al., 2018). Dopamine, serotonin, and muscarinic receptors influence synaptic gain. All except the muscarinic receptors engage in G-protein intracellular signaling, thereby altering neuronal excitability through changes to ion channels, including NMDA receptors (Adams et al., 2013). Synaptic dynamics are also influenced by neural network activity. Synchronization of fast oscillations—primarily in the gamma frequency range (40–100 Hz)—is regulated by inhibitory GABAergic signaling. In the cortex, a GABAergic interneuron known as a parvalbumin-positive basket cell (PVBC) connects to multiple pyramidal neurons, hyperpolarizing them. When this hyperpolarization subsides, the pyramidal neurons fire synchronously, creating a rhythmic oscillation in the network that recurs cyclically (Gonzalez-Burgos & Lewis, 2012).

A person’s perception is thus guided by integrating new sensory inputs with pre-existing empirical beliefs. Focused attention increases the precision and impact of prediction errors on higher neural levels. Bidirectional communication between hierarchical levels occurs in the cortex. Bottom-up prediction errors originate in superficial layers, while top-down predictions are sent from deeper cortical layers. Top-down prediction transmission is modulated by neuromodulatory mechanisms. Through the inferences drawn from this information exchange, behavior is adapted to reduce uncertainty in both the external and internal world (Corlett et al., 2010; Sterzer et al., 2018; UCL, 2024).

Delusions arise when mismatches and instability between predictions and sensory experiences persist, leading the brain to resolve the discrepancy by forming new, often false beliefs. Delusional ideas reflect a failure in both bottom-up and top-down communication within neural circuits, resulting in predictions that do not adequately adjust to actual sensory inputs. Top-down predictions become excessively rigid or distorted, causing the brain to interpret

new experiences based on inaccurate beliefs. This may explain why patients continue to hold delusional beliefs even in the face of contradictory evidence (Corlett et al., 2010; Sheffield et al., 2024).

Neural circuits involving the hippocampus, prefrontal cortex, and striatum are central to information processing. Studies indicate impaired prediction error processing in the midbrain, striatum, and cortical areas (Ermakova et al., 2018; Katthagen et al., 2020). Dopaminergic transmission dysfunction may be triggered by psychosocial stress, which activates microglia in the hippocampus and promotes dopamine release in the striatum.

Disruption of dopaminergic signaling has been noted in circuits such as the ventral tegmental area (VTA) and the striatum. Elevated dopaminergic transmission may play a role in encoding biologically salient events like prediction errors, thereby increasing the salience of irrelevant stimuli and fostering the formation of aberrant and persistent beliefs (Corlett et al., 2010; Katthagen et al., 2020). This dysfunction occurs in regions such as the hippocampus, anterior cingulate cortex, and striatum, where abnormal prediction error signals contribute to the formation and persistence of delusions. Impaired precision in representing prior beliefs relative to sensory data, and dysfunctional interactions between beliefs and sensory input in a dynamically changing environment, appear to underlie psychotic manifestations (Heinz et al., 2018).

The amygdala also plays a role in attributing emotional significance to prediction errors, influencing the intensity with which a person maintains a delusional belief.

Examples of types of delusional ideas

Ideas of Reference

In experimental studies, healthy adults administered ketamine exhibit delusional ideas of reference (POMAROL-CLOTET et al., 2006). Ketamine interferes with cortical neural signaling by blocking NMDA receptors, potentially disrupting top-down predictions of reality while simultaneously increasing bottom-up AMPA signaling and acetylcholine release at the synapse (Jackson et al., 2004).

Delusional ideas of reference may arise from attentional disturbances caused by pathological prediction errors (Pearce and Hall, 1980). Acetylcholine release from the basal nucleus of Meynert may mediate such surprise-triggered attentional shifts (Bao et al., 2001; Holland and Gallagher, 2006; Lee et al., 2005). Individuals may then focus attention on irrelevant external stimuli or events, assigning them personal meaning. These experiences call for interpretation, which may result in the formation of referential delusions (Corlett et al., 2010).

Ideas of Passive Influence

"My fingers pick up the pen, but I don't control them. What they do has nothing to do with me."

Such a statement may reflect impaired prediction of the sensory consequences of voluntary body movements (Blakemore, 2003; Blakemore et al., 2003; Frith, 2005). A deficit in motor prediction specificity in the cerebellum might be involved, along with parietal and frontal cortical structures (Frith, 2005; Schnell et al., 2008). A movement without a clear prediction of its outcome may then be attributed to an external agent.

Moreover, prediction errors caused by "noise" from dysregulated midbrain dopaminergic neurons projecting to the prefrontal cortex might disrupt the prediction process linking "intention to act" – "action" – "evaluation of outcome." Instrumental learning, involving the basal ganglia, does not necessarily require conscious evaluation of outcomes. Thus, while goal-directed behavior and outcome assessment may be impaired, habitual learning may remain intact.

Passive control experiences may be explained as behaviors governed by the habit-learning system when the goal-directed prediction system issues vague or distorted predictions (Corlett et al., 2010).

Neuroimaging and delusional ideas

Neuroimaging findings, primarily from fMRI and PET studies, point to the involvement of the dorsolateral prefrontal cortex (DLPFC), left orbitofrontal cortex, and hippocampus in the development and maintenance of delusional ideas. The right prefrontal cortex appears to be functionally linked to delusions regardless of content.

Activity in the hippocampus/parahippocampus and the left dorsomedial prefrontal cortex seems negatively correlated with delusional severity. Although results on cingulate cortex activity vary, most studies indicate reduced anterior and increased posterior activity.

The temporoparietal regions may also be involved in delusional pathology. Different delusional subtypes may activate specific brain areas. In Capgras syndrome, reduced activity in the posterior cingulate cortex has been reported, contrasting with other delusions. Enhanced connectivity in the left retrosplenial cortex—key to face-familiarity processing—may underlie misidentification delusions. Referential delusions appear to engage more brain regions than other types. Their severity is associated with the insula, ventral striatum, and medial prefrontal areas, including the ventromedial prefrontal cortex. The right hemisphere and dopaminergic signaling seem central to delusional jealousy. Persecutory delusions involve the limbic and paralimbic systems and the visual processing system. While few imaging studies have focused on persecutory delusions,

they often report dysfunction in the right frontal lobe, reduced dorsal anterior cingulate activity, and increased posterior cingulate activity. These circuits, along with limbic and visual areas, contribute to their emergence (Arjmand et al., 2020). In schizophrenia, delusional beliefs correlate with reduced gray matter volume in the dorsolateral prefrontal cortex, left caudate nucleus (self-awareness hub), hippocampus, insula, amygdala, thalamus, superior temporal gyrus, and medial frontal gyrus (Rootes-Murdy et al., 2022).

Neuroimaging and predictive errors in psychosis

fMRI studies of individuals at high risk for psychosis, early psychosis patients, and chronic psychotic patients have investigated prediction error processing. To assess this, participants complete psychological tasks during scanning.

Disruptions in reward prediction error processing have been noted in dopaminergic midbrain areas, the striatum, limbic system, and cortical regions such as the dorsolateral prefrontal cortex (Morris et al., 2012; Murray et al., 2008; Schlagenhauf et al., 2014). In early psychosis, dysfunctional prediction error evaluation in the DLPFC has been found, whereas high-risk individuals with only mild symptoms maintain relatively normal cortical functioning (Ermakova et al., 2018). Regarding prediction error precision, one fMRI study indicates that early psychosis patients show impaired learning based on prediction error accuracy in the superior frontal cortex (Haarsma et al., 2020).

A synthetic perspective (A two factor theory)

According to several studies, phasic dopamine release corresponds to the strength and significance of discrepancies between expected and unexpected outcomes of a reward. The prediction error drives learning, reward, and punishment, by conveying to the brain whether the result of an action is positive or negative. These prediction errors carry either a positive or negative valence and are called reward prediction errors (Diederen & Fletcher, 2020). Phasic dopamine release corresponds to the attribution of salience to sensory stimuli and internal representations (Heinz et al., 2018; Maia & Frank, 2017). In psychotic syndromes, the formation of delusional beliefs is facilitated by dysregulated firing of dopaminergic neurons, which attribute meaning to irrelevant stimuli (Heinz, 2002; Kapur, 2003)—this is the previously mentioned "aberrant salience" phenomenon.

In schizophrenia, the disruption of the balance between the processing of prior beliefs and new sensory information can lead to prediction error signaling disturbances. In the Bayesian brain, this can be interpreted as imprecise signaling of prior beliefs (i.e., greater instability increases uncertainty), combined with inaccurate incoming signals from lower levels of sensory cortical areas. This results in pathological prediction error signaling, with diminished precision, distinct from the dopamine-dependent subcortical signaling of reward prediction errors.

The disturbed encoding of reward prediction errors might be explained by a hyperdopaminergic state in the brain's subcortical centers, under the framework of aberrant salience. However, psychotic experiences like delusional ideas are also linked to prediction errors unrelated to reward or punishment signals. This might explain why delusional beliefs may persist even after antipsychotic treatment—albeit with diminished subjective salience.

Therefore, the brain’s predictive function is not solely influenced by dopaminergic reward circuits but also by general sensory signals, which may be distorted or misaligned due to disruptions in sensory processing regions. If the sensory input received by the brain is altered or misfitted to a prior belief, it may assign excessive importance to stimuli regardless of their actual reward value (Sterzer et al., 2018).

These mechanisms could explain impaired filtering of relevant vs. irrelevant information, leading to false conclusions and erroneous belief updating. Such faulty reasoning may stem from pathological encoding of the precision of prior beliefs across various levels of cortical neuronal hierarchies—conditions conducive to the formation of delusional ideas. The key to a combined theoretical approach may lie in further studying the hippocampo-fronto-striatal circuitry using computational psychiatry methods (Heinz et al., 2018).

Possible relationship between neurodevelopmental hypothesis and predictive errors

There are multiple potential connections between neurodevelopmental disruptions and the emergence of schizophrenia, in line with the well-known neurodevelopmental hypothesis. HOX genes—DNA elements that regulate neural patterning—have been identified. Mutations in these genes may disrupt the development of neural networks in cortical and subcortical regions such as the prefrontal cortex, striatum, amygdala, and cerebellum (Corlett et al., 2010). Maladaptive anatomical and functional connectivity within these networks may help explain impaired processing of prediction errors, ultimately contributing to the formation of delusional beliefs.

Neuroinflammation and prediction errors

Neuroinflammation may reduce the functional connectivity of neural networks in cortical areas like the ventromedial prefrontal cortex, as well as in regions such as the striatum and the limbic system (Yin et al., 2019).

Connectivity impairments may correspond to disturbances in prediction error processing, supporting delusion formation, much like in the neurodevelopmental hypothesis.

Clinical application of prediction error theory - a CBT example

Cognitive Behavioral Therapy for psychosis (CBTp) is considered the choice of psychotherapy for delusional beliefs. Core interventions include psychoeducation about psychological experience (e.g., fear and anxiety), emotional analysis of delusion-related stressors, and behavioral techniques to cope. Therapists help patients explore alternative interpretations of their experiences—not to confront the delusion directly but to support alternative beliefs. In prediction error theory, delusions are viewed as high-level beliefs formed through pathological prediction errors and meaningless stimuli. CBTp techniques increase confidence in sensory experiences and reduce uncertainty tied to entrenched delusional priors, thus reinforcing non-delusional beliefs (Sheffield et al., 2024).

Conclusion

A broader understanding of the neurobiological mechanisms underlying delusional beliefs remains an area of ongoing theoretical and empirical investigation. Advances in brain imaging and functional mapping technologies may yield clearer insights. Psychotic neurobiology may be further elucidated through computational psychiatry—a novel and promising approach, currently underrepresented in Greek academic literature. Developing this field and applying its findings in clinical settings would be valuable in Greece as well.

References

Adams, R. A., Stephan, K. E., Brown, H. R., Frith, C. D., & Friston, K. J. (2013). The Computational Anatomy of Psychosis. Frontiers in Psychiatry, 4(47).
https://doi.org/10.3389/fpsyt.2013.00047

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Pearson.

Arjmand, S., Kohlmeier, K. A., Behzadi, M., Ilaghi, M., Mazhari, S., & Shabani, M.
(2020). Looking into a Deluded Brain through a Neuroimaging Lens. The Neuroscientist, 27(1), 73–87. https://doi.org/10.1177/1073858420936172

Bao, S., Chan, V. T., & Merzenich, M. M. (2001). Cortical remodelling induced by activity of ventral tegmental dopamine neurons. Nature, 412(6842), 79–83.
https://doi.org/10.1038/35083586

Blakemore, S.-J., Oakley, D. A., & Frith, C. D. (2003). Delusions of alien control in the normal brain. Neuropsychologia, 41(8), 1058–1067. https://doi.org/10.1016/s0028-3932(02)00313-5

Blakemore, S.-J. (2003). Deluding the motor system. Consciousness and Cognition, 12(4), 647–655. https://doi.org/10.1016/j.concog.2003.07.001

Corlett, P. R., Taylor, J. R., Wang, X.-J. ., Fletcher, P. C., & Krystal, J. H. (2010). Toward a neurobiology of delusions. Progress in Neurobiology, 92(3), 345–369.
https://doi.org/10.1016/j.pneurobio.2010.06.007

Chawla, D., Lumer, E. D., & Friston, K. J. (1999). The Relationship Between Synchronization Among Neuronal Populations and Their Mean Activity Levels. Neural Computation, 11(6), 1389–1411. https://doi.org/10.1162/089976699300016287

Diederen, K. M. J., & Fletcher, P. C. (2020). Dopamine, Prediction Error and Beyond. The Neuroscientist, 27(1), 107385842090759.
https://doi.org/10.1177/1073858420907591

Durstewitz, D. (2009). Implications of synaptic biophysics for recurrent network dynamics and active memory. Neural Networks, 22(8), 1189–1200. https://doi.org/10.1016/j.neunet.2009.07.016

Ermakova, A. O., Knolle, F., Justicia, A., Bullmore, E. T., Jones, P. B., Robbins, T. W., Fletcher, P. C., & Murray, G. K. (2018). Abnormal reward prediction-error signalling in antipsychotic naive individuals with first-episode psychosis or clinical risk for
psychosis. Neuropsychopharmacology, 43(8), 1691–1699.
https://doi.org/10.1038/s41386-018-0056-2

Feldman, H., & Friston, K. J. (2010). Attention, Uncertainty, and Free-Energy. Frontiers in Human Neuroscience, 4. https://doi.org/10.3389/fnhum.2010.00215

Frith, C. (2005). The neural basis of hallucinations and delusions. Comptes Rendus Biologies, 328(2), 169–175. https://doi.org/10.1016/j.crvi.2004.10.012

Friston, K. (2010). The free-energy principle: a unified brain theory? *Nature Reviews Neuroscience*, *11*(2), 127–138. https://doi.org/10.1038/nrn2787

Gonzalez-Burgos, G., & Lewis, D. A. (2012). NMDA Receptor Hypofunction, Parvalbumin-Positive Neurons, and Cortical Gamma Oscillations in Schizophrenia. Schizophrenia Bulletin, 38(5), 950–957.
https://doi.org/10.1093/schbul/sbs010

Haarsma, J., Fletcher, P. C., Griffin, J. D., Taverne, H. J., Ziauddeen, H., Spencer, T. J., Miller, C., Katthagen, T., Goodyer, I., Diederen, K. M. J., & Murray, G. K. (2020). Precision weighting of cortical unsigned prediction error signals benefits learning, is mediated by dopamine, and is impaired in psychosis. Molecular Psychiatry, 26(9), 5320–5333. https://doi.org/10.1038/s41380-020-0803-8

Heinz, A. (2002). Dopaminergic dysfunction in alcoholism and schizophrenia – psychopathological and behavioral correlates. European Psychiatry, 17(1), 9–16. https://doi.org/10.1016/s0924-9338(02)00628-4

Heinz, A., Murray, G. K., Schlagenhauf, F., Sterzer, P., Grace, A. A., & Waltz, J. A. (2018). Towards a Unifying Cognitive, Neurophysiological, and Computational Neuroscience Account of Schizophrenia. Schizophrenia Bulletin, 45(5), 1092–1100.
https://doi.org/10.1093/schbul/sby154

Holland, P. C. (2006). Different Roles for Amygdala Central Nucleus and Substantia Innominata in the Surprise-Induced Enhancement of Learning. Journal of Neuroscience, 26(14), 3791–3797. https://doi.org/10.1523/jneurosci.0390-06.2006

Jackson, M. E., Homayoun, H., & Moghaddam, B. (2004). NMDA receptor hypofunction produces concomitant firing rate potentiation and burst activity reduction in the prefrontal cortex. Proceedings of the National Academy of Sciences, 101(22), 8467–8472.
https://doi.org/10.1073/pnas.0308455101

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

Katthagen, T., Kaminski, J., Heinz, A., Buchert, R., & Schlagenhauf, F. (2020). Striatal Dopamine and Reward Prediction Error Signaling in Unmedicated Schizophrenia Patients. Schizophrenia Bulletin, 46(6), 1535–1546.
https://doi.org/10.1093/schbul/sbaa055

Lee, H. J. (2005). Role of Amygdalo-Nigral Circuitry in Conditioning of a Visual Stimulus Paired with Food. Journal of Neuroscience, 25(15), 3881–3888.
https://doi.org/10.1523/jneurosci.0416-05.2005

Maia, T. V., & Frank, M. J. (2017). An Integrative Perspective on the Role of Dopamine in Schizophrenia. Biological Psychiatry, 81(1), 52–66.
https://doi.org/10.1016/j.biopsych.2016.05.021

McKenna, P. (2017). The Salience Theory of Delusions. *Cambridge University Press EBooks*. https://doi.org/10.1017/9781139871785.009

Mishara, A. L., & Fusar-Poli, P. (2013). The Phenomenology and Neurobiology of Delusion Formation During Psychosis Onset: Jaspers, Truman Symptoms, and Aberrant Salience. *Schizophrenia Bulletin*, *39*(2), 278–286. https://doi.org/10.1093/schbul/sbs155

Murray, G. K., Corlett, P. R., Clark, L., Pessiglione, M., Blackwell, A. D., Honey, G., Jones, P. B., Bullmore, E. T., Robbins, T. W., & Fletcher, P. C. (2008). How dopamine dysregulation leads to psychotic symptoms? Abnormal mesolimbic and mesostriatal prediction error signalling in psychosis. Molecular Psychiatry, 13(3), 239–239.
https://doi.org/10.1038/sj.mp.4002157

POMAROL-CLOTET, E., HONEY, G. D., MURRAY, G. K., CORLETT, P. R., ABSALOM, A. R., LEE, M., MCKENNA, P. J., BULLMORE, E. T., & FLETCHER, P. C. (2006). Psychological effects of ketamine in healthy volunteers. The British Journal of Psychiatry : The Journal of Mental Science, 189. https://doi.org/10.1192/bjp.bp.105.015263

Rootes-Murdy, K., Goldsmith, D. R., & Turner, J. A. (2022). Clinical and Structural Differences in Delusions Across Diagnoses: A Systematic Review. Frontiers in Integrative Neuroscience, 15. https://doi.org/10.3389/fnint.2021.726321

Kapur, S. (2003). Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. The American Journal of Psychiatry, 160(1), 13–23. https://doi.org/10.1176/appi.ajp.160.1.13

Leonidas Mantonakis, Efstratios Karavasilis, Stefanos Dimitrakopoulos, Poulou, L. S., Georgios Velonakis, Nikolaos Kelekis, & Nikolaos Smyrnis. (2024). Differential Effects in the Subsystems of the Salience Network in Schizophrenia. The Open Neuroimaging Journal, 17(1). https://doi.org/10.2174/0118744400303855240416071944

Li, H., Hu, J., Chen, A., Wang, C., Chen, L., Tian, F., Zhou, J., Zhao, Y., Chen, J., Tong, Y., Loh, K. P., Xu, Y., Zhang, Y., Hasan, T., & Yu, B. (2022). Single-Transistor Neuron with Excitatory–Inhibitory Spatiotemporal Dynamics Applied for Neuronal
Oscillations. Advanced Materials, 34(51). https://doi.org/10.1002/adma.202207371

McLean, B. F., Mattiske, J. K., & Balzan, R. P. (2018). Towards a reliable repeated-measures beads task for assessing the jumping to conclusions

bias. Psychiatry Research, 265, 200–207. https://doi.org/10.1016/j.psychres.2018.04.043

Morris, R. W., Vercammen, A., Lenroot, R., Moore, L., Langton, J. M., Short, B., Kulkarni, J., Curtis, J., O'Donnell, M., Weickert, C. S., & Weickert, T. W. (2012). Significant differences in fMRI related activity between healthy adults and people with schizophrenia during reward-related prediction-errors. Molecular Psychiatry, 17(3), 235–235. https://doi.org/10.1038/mp.2012.1

Kowalski, J., Aleksandrowicz, A., Dąbkowska, M., & Gawęda, Ł. (2021). Neural Correlates of Aberrant Salience and Source Monitoring in Schizophrenia and At-Risk Mental States—A Systematic Review of fMRI Studies. Journal of Clinical Medicine,
10(18), 4126. https://doi.org/10.3390/jcm10184126

Sheffield, J. M., Brinen, A. P., Feola, B., Heckers, S., & Corlett, P. R. (2024). Understanding Cognitive Behavioral Therapy for Psychosis (CBTp) Through the Predictive Coding Framework. Biological Psychiatry Global Open Science, 100333–100333. https://doi.org/10.1016/j.bpsgos.2024.100333

Sterzer, P., Adams, R. A., Fletcher, P., Frith, C., Lawrie, S. M., Muckli, L., Petrovic, P., Uhlhaas, P., Voss, M., & Corlett, P. R. (2018). The Predictive Coding Account of Psychosis. Biological Psychiatry, 84(9), 634–643.
https://doi.org/10.1016/j.biopsych.2018.05.015

Schlagenhauf, F., Huys, Q. J. M., Deserno, L., Rapp, M. A., Beck, A., Heinze, H.-J., Dolan, R., & Heinz, A. (2014). Striatal dysfunction during reversal learning in unmedicated schizophrenia patients. NeuroImage, 89, 171–180. https://doi.org/10.1016/j.neuroimage.2013.11.034

Schnell, K., Heekeren, K., Daumann, J., Schnell, T., Schnitker, R., Möller-Hartmann, W., & Gouzoulis-Mayfrank, E. (2008). Correlation of passivity symptoms and dysfunctional visuomotor action monitoring in psychosis. Brain, 131(10), 2783–2797.
https://doi.org/10.1093/brain/awn184

Yin, L., Xu, X., Chen, G., Mehta, N. D., Haroon, E., Miller, A. H., Luo, Y., Li, Z., & Felger, J. C. (2019). Inflammation and decreased functional connectivity in a widely-distributed network in depression: Centralized effects in the ventral medial prefrontal cortex. Brain, Behavior, and Immunity, 80, 657–666. https://doi.org/10.1016/j.bbi.2019.05.011