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Delusion Persistence in Schizophrenia: A Cross-Theoretical Integration of Cognitive, Psychodynamic, and Predictive Processing Models

Yichan Sun

RDF International School, Shenzhen, China
sunyichan2@gmail.com

Abstract. Schizophrenia affects about 23 million people around the world. As one of its core positive symptoms, delusions are characterized by the fact that they cannot be easily corrected in the face of counter-evidence, which has become a difficult point for clinical intervention. Different theoretical schools have put forward systematic explanations of the formation and maintenance of delusions from their own perspectives, but there is a lack of sufficient cross-comparison and integration between these theories. This paper adopts the methods of narrative literature review and cross-theoretical comparison to carry out a systematic analysis from the three theoretical perspectives of the cognitive model, the psychodynamic model and the predictive processing model. The cognitive model emphasizes the role of interpretive bias and attention bias, the psychodynamic model focuses on the function of externalization and defense mechanisms of internal conflict, and the predictive processing model explains delusions from the perspective of an imbalance in the precision weighting of prediction errors. The three theories are different in their levels of interpretation, but they are complementary at the level of the interaction between cognition and experience. The integrated framework proposed in this article suggests that future research should promote the understanding of delusions from a multi-level and multi-method perspective. Clinical intervention should integrate cognitive-behavioral techniques and relational treatment strategies, and optimize individualized intervention plans with the help of computational models.

Keywords: schizophrenia, delusion, cognitive model, predictive processing, psychodynamic

1. Introduction

The core symptoms of schizophrenia are usually seen in disturbances of perception, thought, and emotion. These symptoms include positive symptoms such as delusions and hallucinations, negative symptoms such as affective flattening and avolition, and cognitive problems involving attention and working memory [1, 2]. According to the World Health Organization, schizophrenia affects about 23 million people worldwide, and its lifetime prevalence is estimated to be between 0.3% and 0.66% [3].

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For a long time, different approaches in psychology and psychiatry have explained schizophrenia in rather separate ways. Biological psychiatry has mainly paid attention to genetic risk and neurotransmitter systems. Clinical psychology has focused more on biases in information processing and attribution. Psychodynamic theory, on the other hand, has tried to understand symptoms in relation to inner conflict and personal experience. In recent years, predictive processing and computational psychiatry have offered another way to understand psychotic symptoms, especially from the idea of the Bayesian brain [4]. However, existing research still has some limitations. Many studies discuss schizophrenia from only one theoretical perspective, and there is still not enough dialogue between different theories. Some comparative studies only place different theories side by side, but do not really explain how they may be connected at a deeper explanatory level.

In the positive symptom spectrum of schizophrenia, there is an essential difference between delusions and hallucinations: hallucinations involve abnormalities at the perception level, while delusions belong to disturbances at the content level of thinking, which are manifested as stubborn persistence in false beliefs, and are difficult to change even in the face of strong counter-evidence [5]. It is this characteristic of "still difficult to correct in the face of counter-evidence" that makes delusions one of the most theoretically and clinically significant symptoms in the study of schizophrenia. Why do patients still insist on their delusional beliefs even when they are provided with clear counter-evidence? The exploration of this problem is not only related to the understanding of schizophrenia, but also touches on the basic mechanism for the formation and maintenance of human beliefs.

This article aims to build a multi-level integrated framework through systematic cross-theoretical comparison and provide more comprehensive theoretical guidance for the understanding and intervention of delusions. Specifically, it answers the following three core questions:

- How do different theories explain the formation and maintenance of delusions?
- How do these theories explain the persistence of delusions in the face of counter-evidence?
- Can these theories form a complementary explanation framework?

Focusing on these three issues, this article first elaborates on the theoretical explanations of the cognitive model, psychodynamic model and predictive processing model on the formation and maintenance of delusions, and puts forward a multi-level integrated framework and discusses its theoretical contribution, clinical implications and future research direction.

2. Methodology

2.1. Literature search strategy

This article adopts the method of narrative literature review and cross-theoretical comparison. Narrative literature review is suitable for sorting out the main theoretical lines of complex problems, and is suitable for academic topics with complex research objects and diverse theoretical sources. This article uses English keywords such as schizophrenia, delusions, cognitive model, psychodynamic model, predictive processing, psychosis, etc., and a systematic search is carried out in four databases: PubMed, PsycINFO, Web of Science and Google Scholar.

The inclusion criteria include: (1) papers published in peer-reviewed journals; (2) papers directly related to the formation, maintenance or treatment of delusional symptoms in schizophrenia; (3) papers that clearly propose a systematic theoretical model or from which a theoretical model can be extracted. The exclusion criteria include: (1) purely empirical papers without theoretical contribution; (2) case reports; (3) research published in non-English languages, except for the original works of classical theory.

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2.2. Comparison dimensions and framework

In order to ensure the systematic nature of cross-theory comparison, this article conducts a comparative analysis from three dimensions: the formation mechanism dimension – how theories explain how delusions originally arise, and why individuals develop certain false beliefs; the maintenance mechanism dimension – how theories explain that delusions persist in the face of refutation, and why it is difficult for patients to change their beliefs through rational arguments; the intervention implication dimension – how each theory provides guidance for clinical practice and helps to understand the direction and strategy of treatment. In addition, this comparison also focuses on the core assumptions, the degree of empirical support and the main limitations of each theory, reveals the deep differences and potential complementarity between different theories through multi-perspective systematic comparison, and builds an integrated understanding framework on this basis.

3. Cognitive model: interpretive biases and delusions

The core assumption of the cognitive model is that the emergence of delusions does not appear out of thin air, but is closely related to the individual's interpretation of anomalous experiences [6]. When individuals experience perceptual changes, thinking disturbances or emotional abnormalities, they will use the existing cognitive framework to explain these experiences. Due to the underlying deficits in attention, working memory and executive function, patients with schizophrenia tend to adopt quick but rough interpretation strategies in the face of ambiguous stimuli. The cognitive model proposed by Beck and Rector further points out that neurocognitive deficits form a vulnerability basis. When encountering stressful events, these pressures give rise to negative core beliefs , which leads to maladaptive behavior, and these behaviors in turn trigger more negative experiences, eventually forming a vicious circle [7, 8].

In this process, multiple cognitive biases work together to promote the formation and maintenance of delusions. External attribution bias allows individuals to attribute negative events to external causes. Jumping to conclusions allows individuals to make judgments quickly when the information is insufficient, and selective attention makes individuals biased towards threatening information [1]. In terms of maintenance mechanism, the interpretation of the cognitive model focuses on confirmation bias and hypothesis protection: once an explanatory belief is formed, the patient tends to pay excessive attention to the evidence that supports their belief while ignoring the evidence that contradicts it. In addition, patients adopt avoidance or protective behaviors to avoid perceived threats, which prevent patients from obtaining empirical evidence that may refute their beliefs, thus allowing delusions to persist.

The advantage of the cognitive model is that it has a strong empirical support basis and clear clinical operability. Based on the understanding of this model, cognitive behavioral therapy (CBT) has become the main psychological intervention for delusions. Its core strategies include helping patients test evidence, slowing down the judgment process, reducing dependence on threatening interpretations, and gaining new experiences that may modify beliefs through behavioral experiments. In addition, cognitive remediation training has been proven to moderately improve cognitive function and daily functioning [9]. However, the model does not explain the individual differences of delusional content (such as persecutory type, grandiose type, and jealous type), which cannot explain why individuals with similar cognitive bias form different types of delusions [9].

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4. Psychodynamic model: delusions as externalization of inner conflicts

In the classical tradition of psychodynamics, delusions can be understood as a way for individuals to maintain self-meaning and defend against anxiety under psychological distress. Freud used libido theory to understand the essence of schizophrenia as a regressive psychological mechanism - that is, the psychological development of the individual retreats to the earlier stage of narcissism [10]. In schizophrenia, due to setbacks in the external world or internal conflicts, the individual's libido withdraws from external objects and reinvests in the self, resulting in a loss of interest in the outside world and impaired reality testing. In his analysis of Schreber's case, Freud further proposed that paranoia comes from his unconsciously repressed homosexual impulse, which is externalized through the defense mechanism of projection and transformed into the belief that "others are persecuting me" [11].

Following Freud, Melanie Klein's theory of object relations provides a more refined framework for understanding delusions. Klein's concept of the paranoid-schizoid position points out that infants use primitive defense mechanisms such as splitting and projective identification to deal with contradictory emotions towards the object. For patients with schizophrenia, their psychological function is fixed in or retreats to the paranoid-schizoid position, and patients transform their inner pain into external threat experiences through projection, forming persecutory delusions. Although this defense mechanism avoids the anxiety of internal conflict, the price is serious impairment of the ability to test reality [12, 13].

The advantage of the psychodynamic model is that it focuses on the individual meaning and psychological function of delusions, and understands the symptoms in the individual's life history and relationship background, which is conducive to establishing a therapeutic alliance and understanding the patient's inner experience. However, the core concepts of the model (such as unconscious conflicts and defense mechanisms) are difficult to measure and verify through objective methods, and lack testable hypotheses; the existing empirical support mainly comes from case studies and small clinical samples, and lacks verification by large-scale randomized controlled trials; the terminology system of the model is relatively abstract, and its guidance at the level of clinical practice is not as clear as that of the cognitive model. In terms of intervention, the model emphasizes that therapeutic relationships are a medium of change, providing a stable environment for empathy and understanding the psychological meaning behind delusions, and ultimately helping patients integrate split emotional experiences.

5. Predictive processing model: precision dysregulation and delusions

Predictive processing theory is an emerging computational psychiatry framework that understands the brain as an active prediction machine. Based on the predictive coding model of Rao and Ballard , the theory suggests that the cognitive process of the brain follows two basic patterns: top-down prediction signals (transmitting expectations of upcoming sensory input from higher-level brain areas to lower-level brain areas) and bottom-up prediction error signals (lower-level brain areas report the difference between the prediction and the actual input to higher-level brain areas) [14, 15]. The key to the framework lies in the precision weighting mechanism: the brain not only calculates the size of the prediction error, but also evaluates the reliability of the error. High-precision prediction error means high signal credibility, which should cause a large belief update [16].

The core explanation of the predictive processing model for delusions in schizophrenia lies in the imbalance of precision weighting, which is manifested in excessive precision for prediction errors at the sensory level, while the precision of higher-level prior beliefs is relatively insufficient [4, 17]. In

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normal cognitive processing, top-down prior beliefs can effectively suppress bottom-up noise signals. However, in patients with schizophrenia, the brain tends to distrust its own predictions and relies more on noisy sensory input, resulting in a large amount of random noise being mistakenly treated as important signals [18, 19]. The formation of delusions can be understood as: in the face of a large number of inexplicable abnormal prediction errors, the cognitive system tries to find a unified causal explanation for these chaotic signals, and finally captures a wrong but seemingly reasonable explanation and solidifies it into a stable belief.

The biggest advantage of the predictive processing model is that it provides a unified computational framework that can establish formal links between neurobiology, cognition and behavior, which traditional cognitive models and psychodynamic models do not have. However, the model also faces challenges: it is still highly theoretical at present, and the direct empirical evidence of many core assumptions is insufficient; the translation from computational model to clinical intervention is still in the early stage, and mature clinical assessment tools or intervention plans have not been developed. At the intervention level, pharmacological treatment may take effect by adjusting the precision weighting mechanism, and at the same time, it inspires new ideas such as using computational models to predict response to intervention.

6. Cross-theoretical comparison

There are significant differences in the interpretation of the formation mechanism of delusions among the three theories, but they also show a certain level of complementarity. From the perspective of information processing, the cognitive model emphasizes the core role of cognitive bias in the process of transforming anomalous experiences into delusional beliefs, which has strong empirical support. The psychodynamic model goes deep into the psychological history and internal conflict level of the individual, understands delusions as the product of defense mechanisms and meaning construction, and focuses on the individual meaning and psychological function of delusions. The predictive processing model provides the most basic explanation from the perspective of computational systems, regards delusions as disturbances in belief updating caused by dysregulation of precision, and provides a unified framework for understanding the neural basis of delusions.

In terms of maintenance mechanism, the three theories also show complementary characteristics. The cognitive model emphasizes the role of cognitive bias and safety behaviors in maintaining delusions; the psychodynamic model attributes persistence to the unresolved state of unconscious anxiety and the continuous operation of the defense mechanism; the predictive processing model explains why it is difficult to change delusional beliefs from the perspective of precision imbalance. These three maintenance mechanisms are not mutually exclusive, but may describe different analytical levels of the same phenomenon: the imbalance of precision leads to systematic deviations in individual information processing, and these deviations are used to manage and defend against internal psychological distress [20]. Table 1 provides a systematic comparison.

Table 1. Comparison of cognitive, psychodynamic, and predictive processing models

Dimension	Cognitive Model	Psychodynamic Model	Predictive Processing Model
Core Assumption	Cognitive biases in interpreting anomalous experiences	Defensive externalization of inner conflicts	Dysregulated precision weighting of prediction errors

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Table 1. (continued)

Formation Mechanism	External attribution, jumping to conclusions, selective attention	Regression and projection convert conflicts into external threats	Low prior precision: sensory noise overtrusted, aberrant errors emerge
Maintenance Mechanism	Confirmation bias, hypothesis protection, safety behaviors block disconfirmation	Paranoid-schizoid fixation, continuous anxiety externalization	Low prior precision persists: disconfirming evidence ignored, confirming evidence overvalued
Limitations	Limited specificity for delusional content; long-term intervention efficacy wanes	Unfalsifiable core constructs; limited cross-cultural and cross-diagnostic generalizability	Computational intractability in clinical settings; mechanistic claims require prospective validation

Based on the above comparative analysis, this paper proposes a multi-level integrated framework to understand the formation and maintenance of delusions. The framework contains three interrelated levels, as illustrated in Figure 1. The first layer is the neural computational layer, which corresponds to the predictive processing model. This layer describes how precision dysregulation produces abnormal prediction error signals at the neural level. The second layer is the cognitive processing layer, which corresponds to the cognitive model. This layer describes how cognitive biases transform abnormal signals into systematic delusional beliefs. The third layer is the psychodynamic layer, which corresponds to the psychodynamic model. This layer describes how the psychological history and internal conflicts of individuals shape the specific content of delusions and give them psychological functions. In this framework, the imbalance of the lower layer provides the neural basis for the deviation of the upper level. The deviation of the upper layer also provides maintenance conditions for the imbalance of the lower layer, forming a dynamic interaction cycle between the three levels.

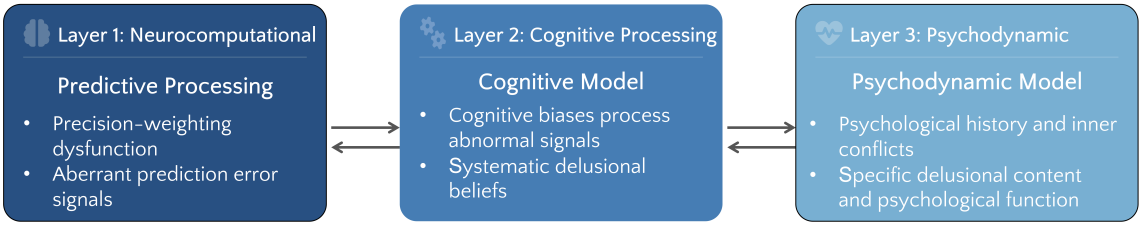


Figure 1. An integrative multilevel framework for delusion formation and maintenance

7. Conclusion

7.1. Main findings

Through systematic cross-theoretical comparison, this paper conducts an in-depth analysis of the formation and maintenance mechanisms of delusions in schizophrenia. The analysis is based on three perspectives: the cognitive model, psychodynamic model and predictive processing model. Focusing on the three dimensions of delusion formation mechanism, maintenance mechanism and intervention implications, the study found significant differences between the three theories. These differences are reflected in explanatory level, unit of analysis and core mechanism assumptions. However, the three theories are also complementary in emphasizing the interaction between cognition and experience. The cognitive model focuses on systematic deviations in information

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processing. It provides a framework for understanding the cognitive mechanisms of delusions and the most direct clinical intervention strategy. The psychodynamic model focuses on the individual meaning and psychological function of delusions. It provides a unique perspective for understanding the content formation and underlying motivation of delusions. The predictive processing model provides a unified theoretical framework from the perspective of computational systems. It helps to understand the neural basis and precision imbalance of delusions.

7.2. Limitations and future directions

As a narrative literature review and cross-theoretical comparative research, this article has certain limitations. Cross-theoretical comparison involves the dialogue of different paradigms. The differences in conceptual systems and research methods of the three theories make strict comparisons difficult. Moreover, the integrated framework proposed in this article is still conceptual, and its effectiveness needs to be tested through empirical research.

The future research directions include carrying out integrated empirical research, while measuring cognitive biases, precision weighting and the quality of early object relations, and testing the predictive power of multi-level frameworks. Future studies may also design and evaluate randomized controlled trials of multi-level integrated intervention plans to test whether multi-level intervention is better than a single approach. In addition, future research can pay attention to the content dimension of delusions, and explore whether different delusional subtypes correspond to different cognitive and neural mechanisms. Through these efforts, the academic community is expected to make greater progress in understanding delusions in schizophrenia, and ultimately provide more effective help to patients.

References

[1] Sheffield, J. M., Suthaharan, P., Leptourgos, P., & Corlett, P. R. (2022). Belief updating and paranoia in individuals with schizophrenia. *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging, 7*(11), 1149-1157. https://doi.org/10.1016/j.bpsc.2022.03.013

[2] Jauhar, S., Johnstone, M., & McKenna, P. J. (2022). Schizophrenia. *Lancet, 399*(10323), 473-486. https://doi.org/10.1016/S0140-6736(21)01730-X

[3] World Health Organization. (2025). Schizophrenia. https://www.who.int/news-room/fact-sheets/detail/schizophrenia

[4] Friston, K. (2023). Computational psychiatry: from synapses to sentience. *Molecular Psychiatry, 28*, 256-268. https://doi.org/10.1038/s41380-022-01743-z

[5] Corlett, P. R., & Fletcher, P. (2021). Modelling delusions as temporally-evolving beliefs. *Cognitive Neuropsychiatry, 26*(4), 231-241. https://doi.org/10.1080/13546805.2021.1938984

[6] Garety, P. A., Kuipers, E., Fowler, D., Freeman, D., & Bebbington, P. E. (2001). A cognitive model of the positive symptoms of psychosis. *Psychological Medicine, 31*(2), 189–195. https://doi.org/10.1017/S0033291701003312

[7] Beck, A. T., & Rector, N. A. (2005). Cognitive approaches to schizophrenia: Theory and therapy. *Annual Review of Clinical Psychology, 1*, 577–606. https://doi.org/10.1146/annurev.clinpsy.1.102803.144205

[8] McCutcheon, R. A., Keefe, R. S. E., & McGuire, P. K. (2023). Cognitive impairment in schizophrenia: Aetiology, pathophysiology, and treatment. *Molecular Psychiatry, 28*, 1902–1918. https://doi.org/10.1038/s41380-023-01949-9

[9] Vita, A., Barlati, S., Ceraso, A., Nibbio, G., Ariu, C., Deste, G., & Wykes, T. (2021). Effectiveness, core elements, and moderators of response of cognitive remediation for schizophrenia: A systematic review and meta-analysis of randomized clinical trials. *JAMA Psychiatry, 78*(8), 848–858. https://doi.org/10.1001/jamapsychiatry.2021.0620

[10] Freud, S. (1914). On narcissism: An introduction. *Standard Edition, 14*, 67–102.

[11] Freud, S. (1911). Psycho-analytic notes on an autobiographical account of a case of paranoia (dementia paranoides). *Standard Edition, 12*, 9-82.

DOI: 10.54254/2753-7048/2026.35678

[12] Petrovic, P., & Sterzer, P. (2023). Resolving the delusion paradox. *Schizophrenia Bulletin, 49*(6), 1425-1436. https://doi.org/10.1093/schbul/sbad084
[13] Garrett, M. (2026). Psychoanalytic notes on psychosis, disturbances in perception, delusional narratives, and the Bayesian predictive processing model of the brain. *Psychoanalytic Inquiry, 46*(4), 379-396. https://doi.org/10.1080/07351690.2025.2501504
[14] Rao, R. P. N., & Ballard, D. H. (1999). Predictive coding in the visual cortex: A functional interpretation of some extra-classical receptive-field effects. *Nature Neuroscience, 2*(1), 79-87. https://doi.org/10.1038/4580
[15] Jiang, L. P., & Rao, R. P. N. (2022). Predictive coding theories of cortical function. *Oxford Research Encyclopedia of Neuroscience.* https://doi.org/10.1093/acrefore/9780190264086.013.328
[16] Katthagen, T., Fromm, S., Wieland, L., & Schlagenhauf, F. (2022). Models of dynamic belief updating in psychosis: A review across different computational approaches. *Frontiers in Psychiatry, 13*, 814111. https://doi.org/10.3389/fpsyt.2022.814111
[17] Fletcher, P. C., & Frith, C. D. (2009). Perceiving is believing: A Bayesian approach to explaining the positive symptoms of schizophrenia. *Nature Reviews Neuroscience, 10*, 48–58. https://doi.org/10.1038/nrn2536
[18] Adams, R. A., Vincent, P., Benrimoh, D., Friston, K. J., & Parr, T. (2022). Everything is connected: Inference and attractors in delusions. *Schizophrenia Research, 245*, 5–22. https://doi.org/10.1016/j.schres.2021.07.032
[19] Goodwin, I., Kugel, J., Hester, R., & Garrido, M. I. (2023). Bayesian accounts of perceptual decisions in the nonclinical continuum of psychosis: Greater imprecision in both top-down and bottom-up processes. *PLoS Computational Biology, 19*(11), e1011670. https://doi.org/10.1371/journal.pcbi.1011670
[20] Erdmann, T., & Mathys, C. (2022). A generative framework for the study of delusions. *Schizophrenia Research, 245*, 42–49. https://doi.org/10.1016/j.schres.2020.11.048