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(Neurotransmitter dysfunction and genetic risk in schizophrenia)

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TABLE OF CONTENTS

Abstract

How to better decode and formulate appropriate treatment plans has long been an important topic that has attracted researchers in fields as diverse as neuroscience and psychiatry. As we all know, as a serious mental illness, its pathogenic factors and pathological features are complicated, which makes it easy to be misdiagnosed. furthermore, the functional impairment of the disease seriously reduces the quality of life of patients. However, as more scientific research has unfolded, there is overwhelming evidence that chemical imbalances in the brain and genetic risks are important causes of schizophrenia. In this article, we provide an in-depth analysis of neurotransmitter dysfunction and genetic risk in schizophrenia based on two summaries of the well-referenced schizophrenia research literature. First, we review the traditional view of neurotransmitter risk, the validity of new theories, and the possibility of multiple treatment modalities. Second, clues from genetic theories such as the selfish gene theory and imprinted genes were examined, adding new insights into understanding genetic risk. Studies have shown that the potential risk factors for schizophrenia are complex and that the glutamate theory can be a good remedy for the limitations of the traditional dopamine theory. Second, the genetic risk of this disorder can be explained by genetic tug-of-war and imprinted genes, with overexpression leading to extreme outcomes (abnormal symptoms).

In this study. we identified the importance of genetic risk and neurotransmitter imbalance in schizophrenia and why refining these theories is important for decoding and treating schizophrenia.

(Key word: mental illness, schizophrenia, neurotransmitter dysfunction, genetic risk)

Introduction

Schizophrenia - "Psychosis" is a common curse word. It is common to see such people in our lives, even though their actions and words seem to fit the standard definition of "mental illness": nonsense, crazy speech, behavior, and erratic movements. However, in a place that is invisible to outsiders (deep inside of mind), the patients may be suffering pain from constant "intrusion by external voices and passive control of behavior. Although this condition is often referred to as "psychosis," in a rigorous clinical diagnosis, the symptoms are explicitly defined as schizophrenia. As one of the most common and easily misunderstood mental illnesses, the core feature of schizophrenia is that behavior and thinking are easily understood to believe they are controlled by external forces rather than himself. (Even though the disorder shares many of the same symptoms as other mental illnesses, such as depressed mood and slowed thinking).

After decades of extensive research, it is widely accepted by many researchers that the etiology of schizophrenia is complex, diverse, and not precisely defined because each of its components is not unique and necessary (e.g., cortical, behavioral, environmental). However, With the advancement of neurobiology and neuroscience techniques, the study of genetic and neurotransmitter risk factors in schizophrenia is becoming increasingly significant; the former is primarily responsible for providing operational support for the communication between neurons in the brain, while the latter has contributed to neural connectivity during early development of the human brain.

This thesis aims to illustrate the importance of these risk factors for understanding and diagnosing schizophrenia by summarizing two essential pieces of literature. In this first article, after introducing the primary symptoms and research challenges of schizophrenia, the applications and limitations of the traditional dopamine theory will be summarized - effectively explaining and treating positive symptoms, but cannot be applied to the negative. The details of the glutamate-as-a-new-receptor theory are introduced later. finally, we will still examine new therapeutic possibilities and individual susceptibility factors - genetics

Secondly, we will summarize in a second article the importance of genetic risk factors in schizophrenia. It is worth noting that the genetic risk of schizophrenia becomes difficult to locate because it differs from the traditional genetic theory. However, Christopher and Pinard give their explanation: imprinted gene and selfish gene theories, suggesting that schizophrenia may be the product of a genetic tug-of- war (strong pull) and that excessive results will lead to mental health risks (genetic tug- of-war)

The result will elaborate on the clues that emerged from the conclusion of the examination of the two papers.

1. Neurotransmitter Imbalances - and Schizophrenia Symptoms - Summary from Daniel C. Javitt and Joseph T. Coyle

A retrospective study by Daniel C. Javitt and Joseph T. Coyle explores why understanding signaling within the brain is critical to better understanding schizophrenia and improving treatment. As a persistent and severe mental illness, Schizophrenia has caused serious physical and mental damage to patients. Its long course of the disease and overwhelming symptoms often lead people to give up treatment, causing more significant harm to a certain extent. According to the author's review of the clinical background of Schizophrenia, the disease's incomplete treatment theories (such as neurotransmitter theory) and drug side effects are equally responsible for its symptoms.

The authors present a refinement of two critical neurotransmitter theories to better understand schizophrenia symptoms. In traditional psychiatry, the dopamine hypothesis is reviewed in terms of theoretical and medical implications. Although it can effectively treat positive symptoms of Schizophrenia, its shortcomings are still very apparent, and it cannot explain or improve other complex symptoms. In contrast to the former, the glutamate hypothesis can better explain and enhance the harmful and emotional disorders of Schizophrenia through the second neurotransmitter theory introduced by the author in this article. Aside from supporting the more important Basic Survival function, the authors believe their research can contribute to Schizophrenia. As well as neurotransmitters, other causative factors, such as genetics, were also mentioned in

other association studies. It is essential to understand the role of genetic factors in Schizophrenia, as some of these genes encode enzymes that are highly susceptible to Schizophrenia.

1.1 Neurotransmitters, Schizophrenia and Treatment Guidance

By vividly citing movie characters and scenes, the authors of this study introduce us to a severe mental illness: Schizophrenia, whose symptoms adversely affect patients' quality of life and behavior. Most patients give up or struggle with treatment, but a few are successful. It is generally characterized by a poor prognosis and inadequate medical guidance, exacerbating the disease and delaying treatment.

After reviewing clinical treatment failures in Schizophrenia, the authors propose that drug guidance limitations derived from neurotransmitter theory may have contributed to these failures. For a long time, the dopamine hypothesis has been the underlying neurotransmitter theory of Schizophrenia, which reduces the positive symptoms of Schizophrenia.

As the dopamine hypothesis is increasingly criticized, the authors suggest further refinement of neurotransmitter theories (such as glutamate) to understand better and treat schizophrenia symptoms. The glutamate hypothesis adds hope to this research.

Specifically, glutamate receptors are more widespread throughout the brain. They are responsible for some essential human survival functions (such as learning and memory)

than dopamine receptors that are active in specific regions. There is evidence that NMDA Receptor disorders are associated with schizophrenia symptoms. Since then, as research into the neural correlates of glutamate receptors has continued in recent years, more researchers have attempted to find treatments to improve glutamate receptor dysfunction. To improve schizophrenia symptoms and improve understanding of the disease, the authors recommend glutamate receptor studies.

1.2 Basic symptoms of Schizophrenia

The authors have provided a brief overview of Schizophrenia's essential concepts throughout this section. Schizophrenia typically results in widespread functional impairments due to severe mental illness that affects the entire brain. Researchers attempt to understand how the disease emerges and the signs that indicate this symptom to create effective clinical treatment rules. Interestingly, Patients were frequently confused by strange thoughts, suspicious voices, and distant voices, and a loss of control over behaviors was also observed. Moreover, the authors cite some specific symptoms that include negative feelings and cognitive disarray, as well as positive and negative symptoms and mental states. As a result, the patient's unusual behavior and lack of enthusiasm for attention often lead to difficulties communicating with the patient's family.

Furthermore, the authors report a routine paper-and-pencil test to determine the extent of brain injury. As a result of this experiment, it is essentially confirmed that

Schizophrenia is associated with severe impairment in quality of life regarding memory and managing cognitive tasks, including the inability to recognize an object.

1.3 The Dopamine Hypothesis and Schizophrenia:

In this section, the authors describe the basis of research on the clinical application of the dopamine hypothesis. In the 1950s, phenothiazine was discovered as one of the most effective schizophrenia dopamine drugs. Its essential purpose is to effectively control positive symptoms (by blocking the function of the dopamine D2 molecule to reduce symptoms). According to the authors, a significant cause of Schizophrenia has been identified in conventional psychiatry based on real-world studies. Nobel laureate Alfred Carlson has confirmed this perspective as well. Amphetamine increases dopamine levels in the limbic and frontal areas of the human brain, which in turn leads to a similar positive effect. Some clinical treatments are continuing to reveal more defections of the dopamine strategy. In particular, the author addressed that criticism has focused on its blemishes therapy applications: Despite improving patients' positive symptoms. However, the treatment may not have reached all patients (some may have improved, others may not).

In addition to reporting a change in the basis for the dopamine hypothesis, the authors also note that reducing adverse symptoms and mood disorders is not feasible. To improve the application of the dopamine hypothesis, some researchers have proposed that an imbalance in dopamine levels in certain regions (frontal lobe D1, basal ganglia

D2) leads to negative emotions and cognitive decline. Researchers have attempted (but failed) to combat these symptoms by developing drug therapies that activate D1 and inhibit D2. As a result of scientific research, new antipsychotics have been developed. Unlike traditional drugs, they reduce psychotic and positive symptoms without side effects, such as clozapine. Nevertheless, the authors emphasize that these modern antipsychotics still cause other physiological diseases, such as diabetes, which cannot be avoided. After that, the authors concluded that dopamine is not the only neurotransmitter disturbed in Schizophrenia, and their hypothesis fails to explain the disorder's complex symptoms.

1.4 Angel dust (PCP) and the glutamate hypothesis

In contrast to the traditional dopamine hypothesis, the authors suggest glutamate receptors may be more effective at treating schizophrenia symptoms. As a hallucinogenic drug, phencyclidine (PCP, "Angel dust") has been prescribed for decades, and abuse can result in apparent mental symptoms. As a result of searching backgrounds from different controlled experiments(amphetamines), the authors discovered that this substance could induce more comprehensive Schizophrenia. Such as marked depression and motor dysfunction (dopaminergic substances such as amphetamines do not induce) are thought to be caused by drugs that block glutamate receptors in the brain. These factors are closely related. Notably, the authors also address that this receptor is generally responsible for supporting essential survival functions such as learning and

memory. They also emphasize its involvement in core functions such as controlling intracranial dopamine levels. Therefore, the authors conclude that dysfunction of the glutamate receptor itself may explain the broad spectrum of schizophrenia symptoms. These symptoms include negative and mood disturbances and positive symptoms due to dopamine abnormalities.

To better understand how glutamate receptors work in the brain, the authors further describe the functional mechanism of brain glutamate receptors in a typical information-processing task: glutamate receptors enhance the Connection. They selectively amplify key neural signals to retrieve the desired information. In contrast, people with Schizophrenia do not have this perception: Not only are they unable to respond strongly to unfamiliar sounds, but strange, misunderstood sounds are often around. Therefore, the authors believe that the symptom is closely related to the malfunction of NMDA receptor-related circuits in the human brain.

1.5 New Pharmacotherapy Guidance

In this section, the authors try to emphasize this point. Although the underlying mechanism of NMDA receptor deficiency in schizophrenia symptoms has not been identified, ongoing clinical research and a new understanding of the disease often offer new hope for drug therapies to address the problem. Interestingly, extensive drug research provides simple but factual solid support for this argument: for example, clozapine, a well-known antipsychotic drug invented and widely used to treat

depression. Not only that, but it can also alleviate psychotic symptoms induced by PCP molecules (traditional antipsychotics cannot).In addition, although some NMDA drug experiments have shown noticeable effects, large-scale trials and commercial applications are still impossible. However, the existing experimental evidence is sufficient to provide factual support for the NMDA hypothesis. Impressively, through in-depth clinical research, we have also achieved promising results in clinical trials, including NMDA receptor drugs that improve symptoms of Schizophrenia. Based on the randomized controlled trial results, the data noted that those with Schizophrenia who took glycine and D-serine in combination with standard medication had significantly improved cognition and mood compared to the other group. Therefore, the authors claim that the experimental results support the conclusion that NMDA receptors are essential in improving symptoms.

Although NMDA receptor drug research has progressed rapidly, the authors note that commercialization has remained challenging because of some unavoidable factors. There are also several other challenges besides technical issues, such as drug loading, slow-release technology for core molecules, and the development of AMPA. This new drug technology activates NMDA directly.

1.6 Multidimensional causative factors

Schizophrenia, a severe mental illness that causes widespread functional impairment, has recently received increasing attention from psychologists and psychiatrists. As the

authors suggest, Schizophrenia is not caused by a single cause but by a combination of factors. Traditionally, if genetics is the cause of the disease, then the patient's immediate family members are also at risk if they inherit the disease. However, evidence from existing association studies does not support this conclusion: in practice, for example, twins are 50% less likely than patients to have the disease, and immediate family members are less likely to have the condition. Thus, the authors claim that while genetic factors do play an important role in rising prevalence, it is not a requirement; other factors, such as environmental factors, are also considered an essential disease factor: better prevention as well as environmental and behavioral factors, which reduce the chance of disease, and vice versa, adverse factors that increase the likelihood of disease. Moreover, Several genetic studies have also been reported, which indicate schizophrenia susceptibility may be increased. These enzymes significantly increase the risk of Schizophrenia since some genes encode enzymes that affect core neurotransmitters.

1. Catechol-O-methyltransferase is prominent in the prefrontal cortex and is primarily responsible for participating in dopamine metabolism.

2. Dysbindin and neuregulin proteins are mainly responsible for affecting the number of NMDA receptors in the brain.

3. D-amino acid oxidase is mainly responsible for the breakdown of D-serine.

In order to have a better understanding of Schizophrenia, other objective genetic factors need to be measured as well. Moreover, Last but not least, the authors have reinforced the global pathological view of Schizophrenia by pointing out that measures of

dysfunction in Schizophrenia should include more than just specific cortical areas. Schizophrenia disrupts interactions between brain regions as normal behavior relies on brain coordination.

1.7 The discussion (1)

Finally, schizophrenia is a severe mental illness manifesting a variety of causative factors and dysfunctions that have attracted the attention of many psychologists and psychiatrists over the years. In addition to the long course of the disease, the treatment is complex, and people are likely to give up easily. Nevertheless, some guiding medical theories limit their therapeutic effects, leading to more severe consequences like suicide.

In recent years, as an essential point of view in schizophrenia psychiatry research, the dopamine theory has been generally supported by most empirical studies of Schizophrenia. It asserts that Schizophrenia's positive symptoms are closely related to abnormal dopamine levels in the brain. Despite its effectiveness in treating positive symptoms (amphetamines can stimulate positive reactions), there are still several limitations. For example, it cannot theoretically explain negativity and emotional symptoms, and the treatment found that some people did not respond to the treatment. Some improvements and drug development efforts have addressed the situation. As well as improving schizophrenia symptoms, clinical use of new antipsychotic drugs reduces adverse drug reactions and alleviates patients' suffering. However, the authors point out that the dopamine hypothesis has significant shortcomings and that it is

necessary to consider different neurotransmitter theories. In addition to illustrating the specific regional distribution of dopamine, the authors discuss the NMDA hypothesis to demonstrate its more important functional details beyond dopamine receptors (including essential functions supporting memory and learning and controlling dopamine levels). As a result of the Controlled drug challenge trials (phencyclidine and ketamine induce a wide variety of psychotic symptoms, both positive and negative), it is more likely that NMDA receptors will address the dopamine hypothesis, as well as provide better symptom relief.

As for treatment, the author introduced a new drug known as clozapine, which has been shown to relieve psychotic symptoms caused by angel dust (phencyclidine). In addition, the authors also verified the validity of the NMDA hypothesis by conducting empirical tests. Combining amino acids, D-serine, and standard drugs can improve symptoms. On the other hand, this new drug brings hope to people experiencing specific symptoms. Despite the technical difficulties, its commercialization is still a challenge. In addition to neurotransmitter factors, the authors also suggest the importance of genetic factors and their relationship to neurotransmitters (partial genes and susceptibility to NMDA receptors), which may contribute to the differences in a person's exposure to neurotransmitter pathways.

Last but not least, the authors state that compared to a single-factor definition, an in-depth understanding of the disease's quality and consideration of a variety of treatment options gives us hope for treating and understanding Schizophrenia more effectively.

2. Genetic tug-of-war theory and schizophrenia symptoms - excessively biased gene expression induces pathological features

A retrospective article by Christopher Badcock and Bernard Crespi shows us why trying to understand the genetic tug-of-war between fathers and mothers in the developing brain is so vital in explaining people's mental illness.

Over the past decade, it has become widely accepted that multiple factors cause mental illness, none of which is unique and necessary—for example, the environment. As technology advances, the authors suggest that this conclusion may be swayed by genetic and epidemiological evidence.

This article takes schizophrenia and autism as examples. Although most traditional psychiatric studies suggest that genetic factors are a significant component of psychiatric disorders, early psychiatric studies identified the genetics of both disorders as significantly different from conventional Mendelian laws, leaving no specific causative genes identified in earlier studies. However, with the development of Hamilton's selfish gene theory and the discovery of gene imprinting in more empirical studies, some typically imprinted genes have been shown to significantly affect a person's physiological characteristics or mental state. For example, overexpression of the IGF2 gene in both male and female parents can lead to genetic disorders (e.g., Silver-Russell syndrome).

In this article, the author introduces and summarizes the biased imprinted gene expression for cases and theory and the influence of mental illness, I believe that this process can be considered as genes in the tug-of-war between my parents and put forward the imprinted genes in the expression of father or mother ancestry have prejudice can significantly affect humans or their offspring's mental state.

2.1 Strong Pull - "Gene Tug of War and Imprinted Gene"

In recent years, there have many scientific studies of families and twins has been shown that certain physical traits and specific diseases may be determined by the sex of the parent who inherits them (e.g., Silver-Russell syndrome). Although there are different answers to this "origin" inheritance, the authors suggest that imprinted genes are a plausible explanation. Specifically, according to the author's hypothesis, the imprinting process can be likened to a genetic tug-of-war in which if information from a parent silences the allele, then the genetic information is passed on to the mother, who makes the corresponding, final imprinted expression. Traits. However, similar to the direction in which the outcome of a tug-of-war ultimately falls, genetic imprinting can affect a range of individual human traits such as behavior, cognition, and mental status. In contrast, the extreme result in different directions can lead to various mental disorders, such as autism.

To verify that genetic imprinting may play a significant role in autism, the authors examined evidence of an association between imprinting and its dysfunction. For

example, an earlier study by the authors suggested that certain forms of autism might be caused by father-biased gene expression. Secondly, a survey of Beckwith-Wiedemann syndrome patients confirmed the former conclusion by finding increased IGF2 expression. Although most previous studies have suggested a variety of causes for autism, the authors recommend considering the profound relationship between imprinted genetics and autism.

Moreover, by comparing a range of paranoia-typical symptoms against some of autism's fundamental deficits, the authors identified a disorder distinct from autism (the other end of the tug-of-war): schizophrenia. For example, in contrast to the fantasies and mood disturbances of schizophrenia, people with autism are always in a state of self-deepness and lack interest. Similarly, the differences in concentration are also stark, with the former showing full attention and the latter showing ambivalence.

Finally, based on this theory, the authors succinctly express the relationship between imprinted gene expression and mental illness. As with a tug-of-war rope, any slight bias towards paternal or maternal imprint expression can result in subtle changes in a person's personality, such as a "quieter" maternal prejudice and a "focused" paternal bias. "However, excessive imprinting can lead to the inheritance of diseases, such as mental retardation in individuals with oversized sires. In contrast, the middle position is the best.

2.2 Additional Clues - Genetic Tug of War and Psychiatric Symptoms

To demonstrate the strong relationship between imprinted genes and mental illness, the authors analyzed several cases of mental disorders strongly correlated with imprinted genes. The first study describes an association between an imprinted gene on human chromosome 15 and two genetic diseases. In this region, the researchers discovered a set of imprinted genes that, when expressed with extreme bias, result in genetic disorders such as Prader-Willi syndrome, which is a condition of maternal bias, and Angel syndrome, which is a condition of paternal bias. Also, the bias of the parents was reflected in the symptoms of the disease: the formerly displayed calmness while the latter exhibited less demand. Meanwhile, the latter showed hyperactivity and autism in infancy. The authors of the second study propose that the gene expression tug-of-war theory could also explain gender bias in mental disorders. By using the concept of imprinted genes, the authors explain two limitations of Baron Cohen's 'extreme male brain' theory. First of all, Cohen only reported a male bias in autism but not that females were also highly biased (even more so than males). Furthermore, different interpretations of psychiatric symptoms exist: the authors state that depression is more prevalent in women, and schizophrenia is more prevalent in men (And these are perfect examples of Cohen's gender bias).

Finally, the authors have attempted to express in this section their conclusion that, firstly, there is a strong association between genetic tug-of-war and mental health. There are two main axes of mental states: gender and imprinted gene bias expression. According

to this theory, compatibility between these two axes can be detrimental to mental health (if compatible, the mental state tends to be good, and vice versa, more serious mental disorders can occur).

2.3 The Discussion (2)

This article presents and summarizes the significance of imprinted genes and genetic tug-of-war theories in understanding mental illness, based on Hamilton's selfish gene theory and genetic research on gene imprinting. Thus, it provides a novel explanation for mental illness (imprinted brain hypothesis), either by overexpression of imprinted genes in maternal and parental genes or by conflicting inheritances (Hamilton-selfish inheritance theory). Therefore, based on this theory, the authors suggest that genetic tug-of-war influences behavior, cognition, and personality as well.

In order to clarify the "strong grasp" on imprinted genes for interpreting genetic factors in psychiatric symptoms, the authors provide case studies of schizophrenia and autism. Accordingly, empirical studies on the autism spectrum and investigations in patients with Beckwith-Wiedemann syndrome have confirmed that the enhanced expression signature of the imprinted gene IFG2 is associated with an autism spectrum disorder. In the authors' empirical study, it was found that imprinted genes in autism were associated with overexpression in the paternal parent. Furthermore, the authors also identified a distinct disorder in their study in which autism

spectrum disorders are compared to other forms of mental mania: schizophrenia. Moreover, there is more evidence of imprinting genes not only in these two psychiatric disorders, but also in other genetic disorders: chromosome 15 and Angel syndrome, for example, and Baron-Cohen's "extreme male brain" theory.

Finally, based on Hamilton's description of genetic conflict and imprinted genes, the author believes that genetic inheritance can be viewed as a genetic tug-of-war, emphasizing that either extreme outcome will have serious consequences. and the Imprinted genes play a significant role in human development and mental state.

The Conclusion

As one of the most misunderstood mental illnesses, schizophrenia has many variants and symptoms, but the major definition of the disease is probably a confusion of thought and behavior: the patients typically experience seeing or hearing things that do not exist (positive symptoms), or having delusions and false beliefs about themselves or the world (negative symptoms). Furthermore, the disorder also seriously affects the patient's social function and emotional expression, the long course of this disease seriously reduces the quality of life of the patient.

In this paper, I have summarized clues about neurotransmitter dysfunction and genetic risk in schizophrenia in two separate articles, and try to explain why refining neurotransmitter theory and genetic processes is essential for understanding schizophrenia disease is very important. It is worth noting that despite the increasing knowledge of the "image" and "nature" of schizophrenia over time, it is undeniable that understanding and treating schizophrenia remains a daunting challenge.

First, there is the neurotransmitter risk. It is well known that past neuroscientific evidence generally supported a role for dopamine neurotransmission in schizophrenia (since the 1950s) and suggested that this "hypothesis" could effectively explain the nature of "positive symptoms," but as more patients were observed, the reliability of this hypothesis was undermined. The reliability of this hypothesis was "shaken" - its inability to explain negative symptoms - until the

clinical application of the glutamate receptor hypothesis: the author's Daniel and Joseph claimed that since the disease could not explain negative symptoms, dopamine might not be the only transmitter disturbed in the disorder, but that other transmitter existed, such as glutamate. transmitters, such as -glutamate. Notably, the author's elaboration of the glutamate hypothesis is based on controlled drug trials and physiological evidence - that the drug phencyclidine (hallucinogen), which responds to glutamate receptors, explains the symptoms of schizophrenia more comprehensively than amphetamine (dopamine receptor response), and that the evidence for glutamate receptors themselves is sufficient to justify their importance - they are responsible for controlling dopamine concentrations and its receptor impairment can trigger a wide range of schizophrenic symptoms. But nonetheless, this hypothetical application will hardly become a reality in a short time (huge costs and difficulty in conducting experiments).

secondly, the importance of genetic risk in developing schizophrenia is also elaborated (some genes encode enzymes that affect neurotransmitter susceptibility and thus increase the risk of developing the disease). As a complex and serious mental illness, schizophrenia is difficult to determine its specific genetic risk due to the complexity of its genetic factors. Christopher and Bernard (authors of another paper) have attributed the genetic risk of schizophrenia to the selfish gene theory, the result of a genetic tug-of-war. Evidence suggests that some physical and

mental disorders may arise as a result of a "strong pull" between the male parent and the mother parent, while that genetic over-selection between the sexes may produce different outcomes (e.g., Beckwith syndrome), like a tug-of-war, where a bias in either direction can lead to opposite consequences.. Of course, imprinted genes play an indelible effect in the midst of this, taking IFG2 as an example, whose overexpression is associated with two distinct psychiatric disorders - autism and schizophrenia. Thus, it is clear that it is far better to be in a balanced position than to be overly biased in the genetic inheritance of genes, and that any extreme result can lead to serious consequences, and that imprinted genes are significant in human development and psychological processes.

Finally, while in addition to summarizing the risk of genes and neurotransmitters in schizophrenia, by examining these two pieces of literature, I would like to suggest an important perspective: In terms of any mental disease (in the case of schizophrenia), while might no single perspective is unique and necessary, we should refining our understanding of the disease through a broader coverage of research and practical tests that In addition to allowing us to In addition to understanding its details in greater detail, we can also better organize treatment plans and alleviate the suffering of patients.

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