

# Explaining Schizophrenia from Medical and Philosophical Perspective

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## Abstract

**Background:** Schizophrenia is a multifactorial two-faced disease with the cognitive impairment being the core of both positive and negative group of symptoms. It is characterized by abnormal behavior, strange speech (word salad) and a decreased ability to understand reality. **Methods:** Using literature review, author's own experience and reflections about the subject, schizophrenia is explained and conceptualized from both medical and philosophical perspective. **Conclusion:** Schizophrenia is a serious illness, treatment of which remains a challenge. Many factors may contribute to the development of schizophrenia including (neuro) immune system dysregulation, genetic susceptibility, pregnancy and birth complications, childhood trauma, social isolation, substance abuse, and many others. Multidisciplinary approach in the treatment of schizophrenia should be preferred and may include, except for psychopharmacology and psychotherapy (psychiatrist, clinical psychologist), nutritional advice (clinical nutritionist), spiritual support (for religious individuals and in the case of religious delusions being present), social support (social worker), cognitive therapy, art and work therapy.

## Keywords

Schizophrenia, Neuroscience, Psychology, Philosophy

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## 1. Introduction

Schizophrenia can be defined as a mental illness characterized by abnormal behavior, strange speech, and a decreased ability to understand reality. Other symptoms may include false beliefs, unclear or confused thinking, hearing voices that do not exist, reduced social engagement and emotional expression, and lack of motivation. It has been estimated that schizophrenia affects circa 24 million people or 1 in 300 people (0.32%) worldwide, with the rate of 1 in 222 people

(0.45%) among adults. Onset is most often during late adolescence and the twenties, but it tends to happen earlier among men [1] [2]. People with schizophrenia often have additional mental health problems such as anxiety, depression, or substance-use disorders. Symptoms typically develop gradually and in many cases, never resolve [3]. Schizophrenia is understood as a multifactorial disease reflecting an interaction between genetic vulnerability and environmental contributors, which might be pregnancy and birth complications, childhood trauma, migration, social isolation, urbanicity, and substance abuse. All of these risk factors may trigger the development of schizophrenia whether acting alone or in combination [4]. According to the two-hit hypothesis for schizophrenia a prenatal genetic or environmental first hit might disrupt some aspects of brain development, what causes increased vulnerability to a second hit, that may occur later in life [5]. An increasing number of clinical, epidemiological, and experimental studies have shown links between schizophrenia and inflammatory conditions. In the context of the two-hit hypothesis the insult at the prenatal or early life stage may prime the nervous system to develop in one of two possible ways. The first way is abnormal development causing the functioning of central nervous system (CNS) being noticeably disordered even before individuals are diagnosed with a schizophrenia-spectrum disorder. The causes of such abnormal development might be alterations to neurons, membranes, dendrites, or neurotransmitter integrity and functioning. The second way is normal development, but such that the CNS is primed so that a second event later in life may disrupt neurological processes [6]. First of these two ways might be associated with earlier onset of schizophrenia, whilst the second way may be associated with the later onset of the disease. It has been also recognized in scientific literature that the (neuro)immune system represents a highly relevant variable in the two-hit hypothesis of schizophrenia, especially the abnormal glutamate signaling [6].

## 2. Novel Insights in the Pathophysiology of Schizophrenia

In a study researchers used Ingenuity Pathway Analysis to analyze canonical and causal pathways of schizophrenia in two different datasets, including patients from Finland and USA. The most significant findings in canonical pathway analysis were observed for glutamate receptor signaling, hepatic fibrosis, and glycoprotein 6 (GP6) pathways. In data-driven causal pathways, ADCYAP1, ADAMTS, and CACNA genes were involved in the majority of the top 10 pathways differentiating patients and controls in both data sets. Results from a Finnish nation-wide database showed that the risk of schizophrenia relapse was 41% lower among first-episode patients during the use of losartan, the master regulator of an ADCYAP1, ADAMTS, and CACNA-related pathway, compared to those time periods when the same individual did not use the drug. Authors emphasized that their results suggest GP6 signaling pathway, ADCYAP1, ADAMTS, CACNA-related purine, oxidative stress, and glutamatergic signaling pathways being among primary pathophysiological alterations in schizophrenia among

patients with European ancestry [7].

In another study, researchers performed a hypothesis-driven analysis to examine the association and enrichment of immune system-related genes and transcripts in the available datasets and found limited evidence for a role of microglia and immune processes among genetic risk variants for schizophrenia. Complement factor 4 gene is linked to schizophrenia and synaptic refinement (greater expression of C4A in the brain is associated with an increased risk of schizophrenia). Transcriptome and methylome studies suggest that immune-related genes and pathways have been shown to be involved in neurodevelopment and neuronal functioning, such as microglia, complement, nuclear factor- $\kappa$ B, toll-like receptor and interferon signaling pathways [8].

Roomruangwong and coworkers proposed a novel theoretical framework, namely the compensatory immune-regulatory reflex system (CIRS), which was previously developed for major depression and bipolar disorder, to be applied to the neuro-immune pathophysiology of schizophrenia and its phenotypes, including first-episode psychosis, acute relapses, chronic and treatment-resistant schizophrenia, comorbid depression and deficit schizophrenia. Researchers argue that these schizophrenia phenotypes and manifestations are accompanied by increased production of positive acute-phase proteins, including haptoglobin and  $\alpha$ 2-macroglobulin, complement factors, and macrophagic M1 (IL-1 $\beta$ , IL-6, and TNF- $\alpha$ ), T helper (Th)-1 (interferon- $\gamma$  and IL-2R), Th-2 (IL-4, IL-5), Th-17 (IL-17), and T regulatory (Treg; IL-10 and transforming growth factor (TGF)- $\beta$ 1) cytokines, cytokine-induced activation of the tryptophan catabolite (TRYCAT) pathway, and chemokines, including CCL-11 (eotaxin), CCL-2, CCL-3, and CXCL-8. Immune profiles in the different schizophrenia phenotypes indicate the activation of the immune-inflammatory response system, but there are also simultaneous signs of CIRS activation, including increased levels of the IL-1 receptor antagonist (sIL-1RA), sIL-2R and tumor necrosis factor- $\alpha$  receptors, Th-2 and Treg phenotypes with increased IL-4 and IL-10 production, and increased levels of TRYCATs and haptoglobin,  $\alpha$ 2-macroglobulin, and other acute-phase reactants [9].

### 3. Two Faces of Schizophrenia

Schizophrenia may be defined as a disease with two faces, meaning positive and negative symptoms, with cognitive impairment being the core of both group of symptoms. Positive symptoms of schizophrenia are delusions, defined as mistaken beliefs (implausible, bizarre, patently untrue) that are held with strong conviction (certainty) even in the presence of superior evidence to the contrary (incorrigibility) [10], disordered thoughts and speech, then tactile, auditory, visual, olfactory and gustatory hallucinations (typically related to the content of the delusional theme). Positive symptoms generally respond well to medication [11] [12]. Delusions may be of persecutory nature (feeling of being stalked, hunted, framed, or tricked without any touch points with objective reality), referential

(wrong beliefs that public forms of communication are a special message for psychotic individual), somatic (wrong beliefs of having a terrible illness or bizarre health problem), erotomanic (wrong beliefs that a celebrity is in love with delusional individual), religious (misinterpretation of religion with distortion of moral values and religious laws, such as Satanism and clinical vampirism [13]) and grandiose (megalomania, wrong belief of having great powers or very significant role without any touch points with reality). Unlike mental constructions (unrealistic, obsessive thoughts organized in a false reality that can exist in a non-psychotic individual), delusions are serious psychotic elements that are causing severe detachment from the objective reality. Mental constructions are changeable by compelling counterarguments or proof to the contrary, but delusions are not. Mental constructions are not completely false and have some touch points with the objective reality, whilst delusions are always patently untrue. Mental constructions are never held with an absolute conviction, unlike delusions, which are always held with an absolute conviction. Individuals who suffer from schizophrenia sometimes have severe logorrhea (excessive wordiness and repetitiveness) and unlike neurotic individuals who are sometimes logorrheic, but still coherent in speech most of the time, patients who suffer from schizophrenia have incoherent speech most of the time, the so-called, word salad (confused or unintelligible mixture of seemingly random words and phrases) with neologisms (new words without clear derivation and without logical meaning). Word salad often contains elements of delusions and hallucinations (usually associated with delusional theme). Some neologisms might reveal what is the theme of delusion.

Hallucinations can be auditory (hearing voices), visual (seeing lights, objects, people or patterns), olfactory (good and bad nonexistent smells), gustatory (good and bad nonexistent tastes) and tactile (for example, delusional parasitosis). Auditory hallucinations might be understood as a distortion of inner voice. Individuals who suffer from schizophrenia might perceive their own reflection, which is typically incoherent, illogical and *too* loud (strong thoughts echo), as a foreign voice (voice of a mind intruder). Their mind tries to compensate this cognitive impairment by rejecting entire cogitation and perceiving it as a *voice of a stranger*. In the context of translational approach auditory verbal hallucinations implicate speech perception areas in the left temporal lobe, impairing perception of and attention to external sounds. Amygdala could be implicated in the emotional *colouring* of the voices [14]. Visual hallucinations may be related to disruption of functional connectivity networks, with underlying biochemical dysfunction, such as decreased cholinergic activity. Structural abnormalities in primary and higher order visual processing areas have also been found in patients suffering from visual hallucinations [15]. Olfactory hallucinations associated with schizophrenia are argued as a prognostic factor of a poorer outcome. Authors of a phenomenological survey found no relationship between disease severity measures and type or frequency of olfactory hallucinations. They found

the predominance of negative olfactory hallucinations and interestingly, significant relationship between frequency of olfactory hallucinations and severity of tactile hallucinations [16]. Based on the available scientific literature impaired cortical excitation-inhibition balance, cortical plasticity and motor resonance have all been reported in schizophrenia. Cortical connectivity impairments have been demonstrated in motor and prefrontal brain regions. Transcranial magnetic stimulation (TMS) has been investigated in the therapy of schizophrenia. The available evidence best supports 1-Hz TMS to the left temporoparietal cortex for the short-term treatment of persistent auditory hallucinations. High-frequency TMS to the left prefrontal cortex might improve negative and cognitive symptoms (small effects). TMS combined with diverse brain mapping techniques and clinical evaluation can unravel critical brain-behavior relationships relevant to schizophrenia, which supports the conceptualization of schizophrenia as a connectopathy with anomalous cortical plasticity [17].

Negative symptoms are deficits of normal emotional responses or of other thought processes, and are less responsive to medication [18]. These include flat expressions or little emotion, poverty of speech, inability to experience pleasure, lack of desire to form relationships, and lack of motivation. Negative symptoms appear to contribute more to poor quality of life, functional ability, and the burden on others than positive symptoms do [19] [20]. DSM-V defines the negative symptoms as affective flattening, alogia (poverty of speech), and avolition (inability to initiate and persist in goal-directed activities) [12]. Neuronal networks dysfunction related to negative symptoms can be divided into neuroanatomical (abnormalities involving the frontal and subcortical structures; decrease in the prefrontal, especially orbitofrontal region white matter volume; volume loss in anterior cingulate, insular cortex and left temporal cortex; ventricular enlargement; reduced perfusion in frontal, prefrontal, posterior cingulate, thalamus, some parietal and striatal regions), neurochemical (affecting lower cortical dopamine transmission-mesocortical pathway; reduced noradrenergic transmission; reduced serotonergic transmission; hypo-functioning NMDA; altered neuroplasticity; reduced serum brain-derived neurotrophic factor), electrophysiological (reduced alpha power; increased delta activity; reduced Mu desynchronization; reduced gamma power and synchrony; defective P50; decreased N200; abnormal P300) and genetic. Psycho-social abnormalities associated with negative symptoms are impairment in outward emotional expression with spared internal experience, significantly less anticipatory pleasure for goal-directed activities, an underlying impairment in the ability to convert experience into action, fails to initiate behaviors by themselves, lesser subjective value of reward, impoverished environment, lack of coherent stimulation and ambiguous rewards, under-stimulation, social breakdown [21]. Cognitive deficits associated with schizophrenia are found in working memory, long-term memory, verbal declarative memory, semantic processing, episodic memory, attention and learning, especially verbal learning [22] [23] [24] [25] [26].

#### 4. Philosophical Perspective

Philosophically, schizophrenia can be defined as a state of mind in which mind contradicts to itself. In Christian theology and philosophy, Devil (Satan, former Lucifer) is the symbol of spiritual schizophrenia because of being in absolute contradiction with oneself. Lucifer (the Light Bearer) became Satan (the darkness) because of vanity-driven madness and grandiose delusions. Moral schizophrenia, as here-proposed new explanation of such term, differing from Michael Stoker's terminology (moral schizophrenia defined as a split between one's motives and one's reasons) [27], can be defined as a state of mind in which moral inversion occurs (what is good is bad, what is bad is good) and it is also characterized by moral ambivalence (insisting in high moral standards for oneself, but at the same time be ready to commit crimes against others without showing any remorse, and considering oneself as a moral person). Deep and persistent contradictory beliefs and actions may cause schizophrenic rip in an individual's mind.

For some patients suffering from schizophrenia, religion instills hope, purpose, and meaning in their lives, whereas for others it might induce spiritual despair. Patients suffering from schizophrenia might exhibit religious delusions and hallucinations. Religion and religious practices influence social integration, risk of suicide attempts, and substance use, however religion might also serve as an effective method of coping with the illness as it may influence the treatment compliance in some patients [28]. Religious delusions and hallucinations have a direct reference to organized religious themes (prayer, sin, possession) or religious figures (God, Jesus, Devil, Prophet). The supernatural delusions and hallucinations have more general mystic references (black magic, spirits, demons, being bewitched, mythical forces, ghosts, sorcery, voodoo) [29]. Studies, which have evaluated the delusional themes of various religious/spiritual delusions, report that the common themes are that of persecution (by malevolent spiritual entities), influence (being controlled by spiritual entities), and self-significance (delusions of sin/guilt or grandiose delusions) [30] [31]. Positive religious coping has been associated with higher quality of life in the domain of psychological health [32]. Positive religious coping strategies include religious purification/forgiveness, religious direction/conversion, religious helping, seeking support from clergy/members, collaborative religious coping, religious focus, active religious surrender, benevolent religious reappraisal, spiritual connection, and marking religious boundaries [33]. Negative religious coping (spiritual discontent, demonic reappraisal, passive religious deferral, interpersonal religious discontent, reappraisal of God's powers, punishing God reappraisal, and pleading for direct intercession) has been associated with lower quality of life and higher distress [32] [33] [34]. Longitudinal studies have shown that higher salience of religion and use of positive religious coping at the baseline are predictive of lesser negative symptoms, better quality of life, and better clinical global impression [35]. Participation in spiritual activities has been shown to be associated with better social

functioning and dealing with negative symptoms [36].

Some artists described schizophrenia as both mental illness and spiritual problem (demonic possession). Example is a psychological horror film *Daniel Isn't Real* (2019) directed by Adam Egypt Mortimer, from a screen-play by Mortimer and Brian DeLeeuw, based upon the novel *In This Way I Was Saved* by DeLeeuw. The film is a mystical travel into the unknown where demonic entity and schizophrenia intertwine. Artistic pictures made by people who suffer from schizophrenia often reveal the artistic display of evil spirit (demonic entity, devil, incubus) [37]. Science cannot prove either the existence or absence of evil spirits, but either way, the existence of the demonic entity in the mind of a troubled patient cannot be ignored and therefore, it is very important to consider the patient's spirituality and religiosity during the psychotherapy.

## 5. Special Entities Associated with Schizophrenia

### 5.1. Psychopathic Schizophrenia

In a study, sixty-one male forensic patients who met the DSM-IV criteria for schizophrenia were categorized into high and low psychopathic trait groups using the Psychopathy Checklist: Screening Version (PCL: SV). The groups were compared on their criminal history, symptom profile, personality style, risk scores and subsequent institutional violence. Patients with high scores on the PCL: SV had a greater number of previous convictions and were more likely to have a family history of criminality. The high psychopathy-scoring group had higher levels of Positive and Negative Syndrome Scale grandiose and hostile symptomatology, and higher scores on trait impulsivity and aggression. They also had a more coercive, less compliant interpersonal style than the low-psychopathy scoring group. The high-psychopathy scoring group was more likely to be involved in institutional aggression and had higher levels of risk for violence. Authors concluded that patients with schizophrenia and high levels of comorbid psychopathy have a distinctive interpersonal style that may contribute to their greater risk of disruptive institutional behavior [38]. Psychopathy (sociopathy) is a personality disorder characterized by persistent antisocial behavior, impaired empathy and remorse, and bold, disinhibited, and egotistical personality traits [39] [40] [41]. Individuals who have psychopathic personality traits and schizophrenia (schizopaths) may manifest extreme aggression that has both impulsive features (psychotic agitation) and premeditated qualities, which are delusion-driven [42].

### 5.2. Monosymptomatic Schizophrenia

*Anorexia nervosa*, often referred to simply as anorexia [43], is an eating disorder characterized by low weight, fear of gaining weight, and a strong desire to be thin, resulting in food restriction [44]. Many people with anorexia see themselves as overweight even though they are in fact underweight [44] [45] and usually deny they have a problem with low weight [12]. They weigh themselves

frequently, eat only small amounts, and only eat certain foods. Some will exercise excessively, force themselves to vomit, or use laxatives to produce weight loss [44]. Symptoms of *anorexia nervosa* are a low body mass index for one's age and height, *amenorrhea* in women, fear of even the slightest weight gain, rapid and continuous weight loss, an obsession with counting calories and monitoring fat contents of food, preoccupation with food, recipes and cooking, food restrictions despite being underweight or at a healthy weight, food rituals (cutting food into tiny pieces, refusing to eat around others, hiding or discarding of food), purging (laxatives, diet pills, ipecac syrup, water pills), induced vomiting (more common symptom in bulimia), excessive exercise, perception of self as overweight in contradiction to an underweight reality, intolerance of cold and frequent complaints of being cold, hypotension, bradycardia or tachycardia, depression, anxiety disorders, insomnia, solitude, chronic fatigue, rapid mood swings, self-harming and self-loathing, admiration of thinner people and many other [46] [47] [48] [49].

Although schizophrenia and *anorexia nervosa* are seemingly two very distinct psychiatric disorders, symptomatology of these two diseases is interconnected by various types of relationships. Symptoms of anorexia may precede the onset of psychosis, evolve in the active phase of psychosis or more rarely manifest during remission, and conversely, psychotic symptoms may occur transiently in the course of anorexia. When anorexia precedes the manifestation of psychosis, symptoms of anorexia can be treated as a component of the prodromal phase of schizophrenia. Another possibility of co-existence of a psychosis with anorexia is when the eating disorder syndrome manifests at the same time as the full-blown psychotic syndrome. In such cases, when the symptoms of these two disorders occur simultaneously, it is often difficult to distinguish whether patient suffers from schizophrenia, in the course of which anorexia has arisen secondary to psychotic symptoms, or from anorexia during which psychotic symptoms developed, usually thematically associated with eating [50]. There is a high prevalence of comorbidity between schizophrenia and eating disorders, which may occur together with or independent of psychotic symptoms. Binge eating disorders and night eating syndromes are frequently found in patients with schizophrenia, with a prevalence of circa 10%. *Anorexia nervosa* seems to affect between 1% and 4% of patients suffering from schizophrenia [51].

Anorexia might be associated with low self-esteem, lack of self-confidence and psychological trauma related to beauty bullying. Patients who suffer from anorexia might experience the broken mirror syndrome with deformed picture about one's own body image, internalization of abuser's negative emotions and thoughts about one's own body, mind knots (negative emotions and thoughts about body might reflect negativism about oneself as a person), destructive self-criticism with or without self-harming and self-loathing, low self-esteem and strong inferiority complex (feeling of being less worthy than others, admiration of thinner people and perceiving them as superior), strong somatization of emotional pain (gastrointestinal disturbances) and increased suggestibility (inability



to resist the unwanted influence of other people, such as influence of mass media that often promote anorexia as beauty standard). *Anorexia nervosa*, in its severe form with delusions and visual hallucinations about one's own body, might be understood as a monosymptomatic schizophrenia, which may develop as a type of schizophrenia and/or represent its prodromal phase, it may evolve in the active phase of schizophrenia or it can be a completely independent entity. Schizophrenia and *anorexia nervosa* share similar symptomatology: delusions about one's own body (perception of self as overweight in contradiction to an underweight reality), visual hallucinations (seeing one's own body as obese, which is contradictory to underweight reality), co-occurrence of depression and anxiety disorders, social withdrawal and abnormal behavior.

## 6. Discussion

Schizophrenia is a multifactorial complex disease with two faces, meaning positive and negative symptoms, and the cognitive impairment is the core of both group of symptoms. Positive symptoms are delusions, hallucinations, disordered thoughts and speech (word salad). Negative symptoms are deficits of normal emotional response or of other thought processes, such as affective flattening, alogia and avolition. Negative symptoms are more difficult to treat in comparison with positive symptoms that usually respond better to medication. Cognitive impairment associated with schizophrenia refers to deficits in working memory, long-term memory, verbal declarative memory, semantic processing, episodic memory, attention and learning, especially verbal learning. According to the two-hit hypothesis for schizophrenia a prenatal genetic or environmental first hit might disrupt some aspects of brain development, what causes increased vulnerability to a second hit, that may occur later in life. The first hit can cause the abnormal development of the CNS to be noticeably disordered before an individual is diagnosed with schizophrenia (earlier onset of the disease), but on the other the CNS may develop normally in a way that is primed so that a second hit may cause the disease later in life. (Neuro)immune system dysregulation, especially abnormal glutamate signaling may be very relevant factor for the development of schizophrenia. In addition to that, hepatic fibrosis and glycoprotein 6 (GP6) pathways, as well as ADCYAP1, ADAMTS, and CACNA genes and their associated pathways, are found to be relevant in the pathophysiology of schizophrenia. Complement factor 4 gene is linked to schizophrenia and synaptic refinement (greater expression of C4A in the brain is associated with an increased risk of schizophrenia). Immune-related genes and pathways have been shown to be involved in neurodevelopment and neuronal functioning, such as microglia, complement, nuclear factor- $\kappa$ B, toll-like receptor and interferon signaling pathways and therefore, disruption of these genes and pathways may represent the first hit in the development of schizophrenia. Some studies have provided evidence to support the conceptualization of schizophrenia as a type of connectopathy with anomalous cortical plasticity. Philosophically, schizophrenia may be

conceptualized as a state of mind in which mind contradicts to itself, and moral schizophrenia as a state of mind characterized by moral inversion and ambivalence. For some patients, spirituality and religion has a great impact on their disease. Some patients who suffer from religious delusions experience very distressful mind adventures involving maleficent and dangerous evil spirits torturing them. Although science cannot prove either the existence or the absence of evil spirits, the presence of demonic entity in the mind of a troubled patient cannot be ignored and therefore, it is very important to consider the patient's spirituality and religiosity during the psychotherapy. Studies have shown that positive religious coping is associated with better quality of life and improvement of negative symptoms. Therefore, spiritual and/or religious support might be a helpful complementary treatment of schizophrenia for religious patients and/or those suffering from religious delusions. One of the most disturbing schizophrenia-related comorbidities is psychopathic schizophrenia because such individuals are more likely to commit serious crimes in comparison with non-psychopathic schizophrenia patients. Individuals suffering from psychopathic schizophrenia (schizopaths) may manifest extreme aggression that has both impulsive features (psychotic component) and delusion-driven premeditated qualities (mixed psychotic-psychopathic element). *Anorexia nervosa*, in its severe form might co-occur with symptoms of psychosis, represent the prodromal phase of schizophrenia or might be conceptualized as an independent entity, meaning mono-symptomatic schizophrenia characterized by delusional body image and hallucinations about one own's body. In such severe cases, treatment of anorexia as both eating disorder and a psychosis might be necessary.

## 7. Conclusion

Schizophrenia is a serious illness, treatment of which remains a challenge. Many factors may contribute to the development of schizophrenia including (neuro)immune system dysregulation, genetic susceptibility, pregnancy and birth complications, childhood trauma, social isolation, substance abuse, and many other. Multidisciplinary approach to the treatment of schizophrenia should be preferred and may include, except for psychopharmacology and psychotherapy (psychiatrist, clinical psychologist), nutritional advice (clinical nutritionist), spiritual support (for religious individuals and in the case of religious delusions being present), social support (social worker), cognitive therapy, art and work therapy. It is also very important to treat co-existing disorders that may be present in patient suffering from schizophrenia, such as anxiety, depression, substance abuse and eating disorders, especially *anorexia nervosa*.

## Conflicts of Interest

The author declares that there are no conflicts of interests. Author did not receive any funding for this article.

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