Lacan, fragmentation, and the synaptic deficit hypothesis of psychosis

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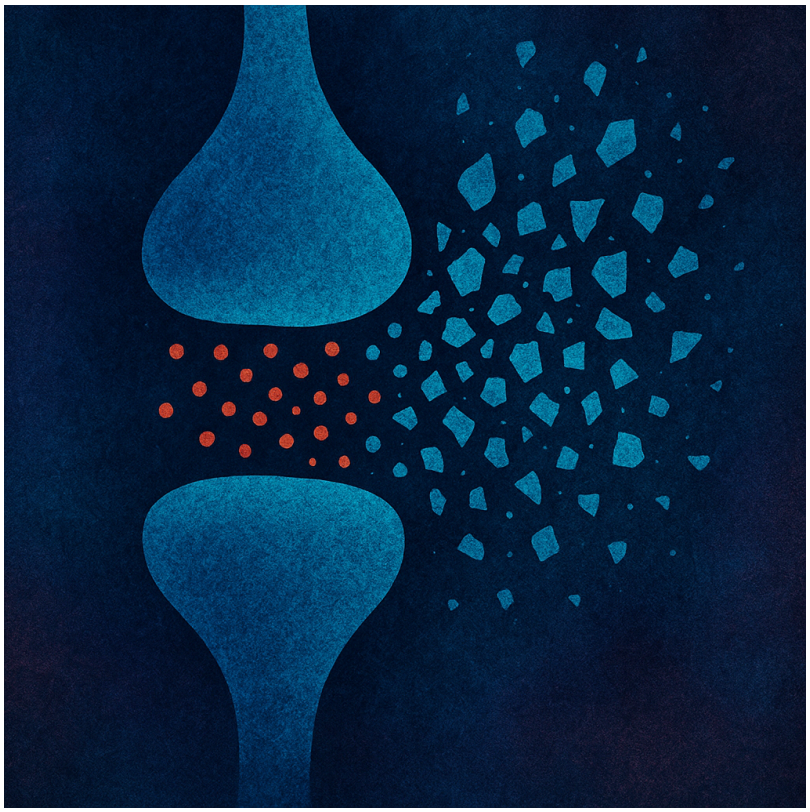
Lacanian Psychoanalysis



For Lacan, we are all split-subjects. As soon as we are inducted into the Symbolic Order, or introduced into language and the rules of society, we have to understand ourselves and others in terms of representations. To live in a society, we have to understand what is expected of us, to "know our place". We have a fundamental need to belong, and it is through conforming to others' expectations that we get our basic needs met. And so the big Other is very effective in shaping our selves and our desires. Our desires are informed by what we believe others want us to desire, and our selves become captured by the ego-ideal we first observe in the mirror stage, living up to who we think we should be or how we think they want us to be; someone whole (Lacan, 1977 p.4). This wholeness is rarely–if ever–achieved, the sense of being a split-subject persists. Yet the Symbolic positions us in intersubjective relationships with others (McCormick, 2022b). Intersubjective language and the cultural ideals and norms signified by it weave us into common webs of meaning and desires that provide a reference point for understanding our place in the world and guiding our actions in it.

We are all fragmented, split-subjects, living within the demands and expectations of the Other and others, but none is so fragmented as the psychotic, who is not bound by the Symbolic (McCormick, 2022b). Many researchers now believe that the fundamental disturbance in schizophrenia can be understood as a lack of ipseity, or sense of inner self (Nelson et. al., 2014). Lacan paraphrases one patient's self-image, as interpreted through her auditory hallucination, thusly: "I, the sow, have just been to the butcher's, I am already disjointed, a fragmented body, membra disjecta, delusional, and my world is fragmenting, like me" (Lacan, 1993 p.52).

This image of the psychotic as fragmented is more than metaphorical: brain imaging studies have revealed that psychopathology–and especially schizophrenia and psychotic disorders–is marked by a lack of synaptic plasticity and dysconnectivity in the brain (Krystal et al., 2017; Stephan et al., 2009). The theory is hardly novel: Charcot hypothesised that hysteria was caused by neural lesions. Not finding them, his protégés Pierre Janet and Sigmund Freud developed ideas that such lesions were of a psychological rather than neurological nature; a fragmenting of consciousness into subconscious or unconscious layers (Walusinski and Bogousslavsky, 2020 p.334). Janet developed this concept as dissociation, while Freud's student Sandor Ferenczi developed a similar concept in "splitting". Ferenczi described dissociation as a splitting off of dead parts of the personality due to shock that allowed for the rest of it to survive, and after "repeated shocks, he spoke of multiple splitting that can extend to the splintering into countless fragments, an atomisation" (Tarquino, 2017). This description is reminiscent of Daniel Paul Schreber, the psychotic judge whose "fragmentation of identity brands all [his] relations with his counterparts on the imaginary level" (Lacan, 1993 p.97). Schreber sees visions of souls who are likewise fragmented, and so there is "a fragmented Flechsig, a superior Flechsig, and an inferior part that ends up fragmented into between forty and sixty little souls" (Lacan, 1993 p.97). The "self" is split, the imaginary is fragmented, and in the Schreberian fashion, we can also say that the nerves that are split, dissociated, and fragmented as opposed to together, connected, and whole.

Charcot did not have a strong enough microscope to find the neural lesions he was looking for, which in today's parlance would more appropriately be referred to as synaptic loss or neurodegeneration. Still, it would be facile to say that disorders ranging from neurosis to psychosis are *caused* by neural lesions. Rather, a prevailing hypothesis posits that chronic stress causes biological responses that are neurotoxic, damaging and killing neurons, resulting in

weaker and altered neural networks and corresponding behavioural changes that render subjects less able to adapt to their environments (Duman et al., 2017; Krystal et al., 2017).

Lacan delineates psychopathology on a spectrum of clinical structures: the neurotic, perverse, and psychotic, which, to paint a picture with broad strokes, may roughly correspond to modern-day DSM classifications of depression and anxiety (neurotic); narcissistic and borderline personality disorders or PTSD (perverse); and schizophrenia (psychotic). So we can think of Lacanian clinical structures as levels of psychic degeneration. Following Charcot and the hypothesis of Krystal et al. (2017), whose work highlights the synaptic loss induced by stress on a spectrum from depression (Krystal et al., 2013; Duman et al., 2016) to PTSD (Krystal et al., 2017), I propose that Lacan's clinical structures broadly correspond to levels of neurodegeneration or cerebral fragmentation caused by chronic and traumatic stress. This hypothesis is further supported by recent studies finding that childhood trauma and traumatic brain injury are major risk factors for, and preludes to, psychotic states. The current prevailing hypothesis for what underlies schizophrenia is glutamatergic abnormalities. While I do not believe this to be the *cause* of schizophrenia, it is interesting to note the centrality of glutamate to language, the stress response, and neurodegeneration.

For Lacan, science as such could not encompass psychoanalysis; it does not address subjects and causes as does the latter (Fink, 1997 p.142). Rather, Lacan saw science as a discourse, a part of the university discourse, in service of the master's discourse, while psychoanalysis is a kind of praxis looking at causes, relationships, and subjectivities (McCormick, 2022a). Unlike Stephan et al. (2009), who propose that the cause of abnormal cerebral functional connectivity and glutamatergic neuron dysfunction underlying schizophrenia is abnormal acetylcholinergic, dopaminergic, and serotonergic dysfunction, I do not believe the *cause* of psychotic disorders can be found in brain chemistry, but in trauma, the realm of the Real and its intersections with the Symbolic. A new field of neuropsychoanalysis has emerged, and this essay fits within that genre, looking at the biological materiality as explained by Symbolic empirical research, and the Real and Symbolic causes of this material reality. To explore the parallels of Lacan's clinical observations of psychosis with the recent medical literature on the epidemiology of psychosis, looking at how trauma induces "holes" or "gaps" both psychically and cerebrally. To look at the psychotic's fragmentation phenomenologically, linguistically, and neurally.

First, I'll look at Lacan's concept of psychosis and its relationship to language. Then, I will look at the current prevailing explanation of psychosis in neuroscience of glutamatergic dysfunction, and I will look at how these two theories overlap in the realm of language. Since glutamatergic neurons underlie the neural mechanism of language, the direction of my argument should not be difficult to predict. Excessive glutamate release, as occurs during stressful conditions, also leads to synaptic loss and even neural necrosis and we can thus say "fragments" the brain, preventing neurons from communicating and the brain from connecting. This leaves holes where our neurons would otherwise connect or remember, with corresponding deficits in speech.

Nervous language

"The nerves are this verbiage and these refrains, this verbalised insistence that has become his universe," Lacan comments on Schreber's memoirs (Lacan, 1993 p.210). Lacan's emphasis was on language, applying linguistics to understand the unconscious, making the claim, "the unconscious… is structured like a language" (Lacan, 2006 p.737). Lacan was also adamant that psychosis could most easily be detected at the level of linguistic disturbance (McCormick, 2022b). Schreber himself makes the connection between nerves and language, "the continual reiteration of certain words is sufficiently obvious to leave no doubt of the deliberate stimulation of the corresponding human nerves to use these words" (Schreber, 1988 p.222). Language is indeed produced in the brain, and has neural correlates (Loritz, 1999).

Language "has the structuring effect of putting us into intersubjective relationships with others" (McCormick, 2022b). However, "the psychotic does not accept… that there is a force outside them that they have to fit into" (McCormick, 2022b). For the psychotic, the Symbolic Order never fully takes hold. It is rejected or foreclosed, and so psychotic subjects are never able to adequately find their place in it, or form a stable sense of self within society. Rather, "in psychosis… reality itself initially contains a hole that the world of fantasy will subsequently fill" (Lacan, 1993 p.45).

Whilst language precedes the subject both at a cultural and developmental level, language and memory have their physical basis in nerves. Specifically, language functions through neurons and synapses. Synapses are the gaps between neurons, where the latter communicate with one another through chemical messengers called neurotransmitters, sending them or their ionic charge across the synapse to the next neuron. Postsynaptic neurons receive neurotransmitters emitted by presynaptic neurons at specific receptor sites, and depending on the neurotransmitters received, send an electrical signal down the length of the neuron to send (or not send) more signals across the next synapse to the next neuron. Glutamate is the most abundant excitatory neurotransmitter in the human brain, it is the excitatory signal of pyramidal cells, the dominant cells of the brain, and NMDA receptors are the most common type of glutamate receptor. Neural networks linked by glutamatergic NMDARs are thus the foundation of language and memory. Our ability to form new memories and learn new things is largely dependent on synaptic plasticity, which is a function of the health and strength of neurons, their dendrites, and the number and type of receptors that line them at synapses (Jewett and Thapa, 2022). Thus the signifying chain described by Lacan has its physical correlate in synaptic networks.

Synapses are gaps between neurons, where they nevertheless connect through chemical messengers. However in the neurodegeneration characteristic of PTSD, schizophrenia, and to a lesser extent disorders such as depression, there is a lack of these gaps, holes where these synaptic gaps should be, where thoughts and memories should be connected the mind draws a blank. This manifests as disruptions in the signifying chain of language. Thus Schreber cannot finish his thought. "He hears – *Lacking now is* … and then the sentence is interrupted, he hears nothing more. We only have his word for it, but for him this sentence has the implicit meaning of - *Lacking now is the leading thought*" (Lacan, 1993 p.113).

For Lacan, psychosis can always be detected at "the level of linguistic disturbance, because the psychotic has… a maladaptive relationship to language" (McCormick, 2022b). Both language and synapses depend on connections, and we need synaptic connectivity to make linguistic associations. Where synapses fail, the brain may be forced down other pathways, to more distant connections. Here, language is fragmented. Schreber has stumbled into a hole – an interruption where a synaptic gap or connection to the next neuron should be.

Chronic stress not only inhibits brain development, but contributes to neuronal death and atrophy (Krystal et al., 2017). Recent research on rodents has found that chronic stress in early life can reduce the density of astroglia in the frontal cortex in adulthood, "demonstrating the potential long-term effects of stress on glial cells" (Leventopoulos et al., 2007). Astroglia are a type of immune neuron that, among other functions, regulates glutamate and prevents the glutamatergic excitotoxicity induced by stress. The reduction of astroglia in rodents with a history of childhood trauma could partly explain a disturbance in language learning that begins in childhood and extends into adulthood. We can imagine a rat rejecting the primordial signifier, or *Bejahung* (Lacan, 1993 p.12).

According to Lacan, traumatic experiences occur in the realm of the Real, or pre-symbolic (McCormick, 2022b). The inability to linguistify them within a shared Symbolic Order organised around the Other characterises psychosis (McCormick, 2022b). For Lacan, the psychotic has a disturbed relationship to language as a result of never having been properly inducted into the Symbolic, and refuses it. However, as a result, they are unable to integrate their experiences within language, situate them, and repress them as a "normal" or neurotic person might.

When experiences are not encoded into language, they can haunt the Real. This means that the psychotic subject's past experiences can haunt the present, in the form of delusions or hallucinations, for example. In the case of PTSD we see a similar phenomenon in which the patient sometimes relives traumatic experiences in the form of flashbacks or other intrusions, though the subject is more aware that such intrusions are not happening now or "real". We can see how these two models or diagnostic labels are not so different, though we think of psychosis as more severe than PTSD. When we consider that psychotic patients tend to have had more traumatic experiences, we can more easily envisage psychosis as an extreme form of PTSD.

Childhood trauma, TBIs, and mirrors in pathophysiology

> A central phenomenon in Schreber's delusion, we may even say an initial phenomenon in the conception he formed of the transformation of the world that constitutes his delusion, is what he calls Seelenmord, soul murder. Now, he himself presents this as being totally enigmatic. To be sure, chapter 3 of the Memoirs, in which he gave reasons for his neuropathy and developed the notion of soul murder, was censored. Still, we know that it contained remarks concerning his family, which would probably have thrown light both on his initial delusion in relation to his father and brother, or to someone else close to him, and on what are commonly called the significant transferential elements. (Lacan, 1993 pp. 75-76)

We can only imagine what events Schreber may have suffered at the hands of his father or brother that would have caused "soul murder" and been subject to censorship. The link between childhood trauma and psychosis has now become well established in the literature (Rössler et al., 2016; Varese et al., 2012; Giannopoulou, 2023). Recent studies have found that those with a history of adverse childhood experiences are approximately three times as likely to experience psychosis in adulthood (Varese et al., 2012). A more recent meta-analysis (Oon Him Peh et al., 2019) found that those at high risk for psychosis had experienced "significantly more severe trauma than controls, regardless of trauma subtype… [they were] 5.5, 2.5 and 3.1 times as likely to report emotional abuse, physical abuse and bullying victimisation, respectively." Kraan et al. (2015) found that 87% of people diagnosed with a psychotic disorder had experienced childhood trauma. This may not even capture the full picture, since some trauma may not meet the DSM criteria, and other childhood trauma goes unreported or unrecalled.

These studies propose various theories concerning the biological mechanism through which trauma mediates psychosis. However it is well established that both PTSD and psychotic patients have increased exposure to glucocorticoids such as the stress hormone cortisol. Giannopoulou et al. (2023) report that "prolonged stress or prolonged exposure to glucocorticoids has toxic effects for the development of the brain, and in particular the hippocampus. Hippocampal changes such as volume reduction and decreased brain-derived neurotrophic factor levels are observed in patients with schizophrenia and in individuals with early-life adversity." Glucocorticoids secreted as a stress response contribute to neuronal death

and atrophy through various mechanisms. Glucocorticoids rapidly induce the release of glutamate (Popoli et al., 2011), and excess glutamate that spills outside the synapse and binds to extrasynaptic NMDARs is excitotoxic, causing synaptic or even cellular death. Chronic stress in early life reduces the number of astroglia, neurons with immune support functions that regulate glutamate, preventing excitotoxicity induced by stress (Leventopoulos, 2007).

Krystal et al. (2017) propose that PTSD and TBIs share a similar pathophysiology. Though one is typically caused by psychic trauma, and the other physical trauma, at the neural level both are characterised by synaptic loss. Interestingly, TBIs are also implicated in the development of psychosis. A meta-analysis looking at traumatic brain injuries and subsequent onset of schizophrenia found a 60% increase in schizophrenia risk following TBIs (Molloy et al., 2011), and some studies have suggested a much higher incidence, with Wilcox and Nasrallah (1987) reporting a 10-fold increase in risk of psychosis following childhood TBI.

The pathophysiology of schizophrenia

Previously, schizophrenia was thought to be a dopaminergic disorder. Now, experts are calling it a fundamentally glutamatergic disorder (Stephan et al., 2009). If we accept Lacan's premise that psychosis is a disordered relationship to language and the Symbolic, a glutamatergic hypothesis makes sense, since language is coded into glutamatergic NMDAR networks in the brain. Although "dysfunction of glutamatergic neurotransmission is increasingly considered to be a core feature of stress-related mental illnesses" (Popoli et al., 2011), researchers are specifically looking at the role of NMDAR hypofunction on GABAergic interneurons in schizophrenia (Synder and Gao, 2020; Dong et al., 2023). This can, confusingly, lead to glutamate overactivity, as the interneurons are not effectively inhibiting its signals.

Post-mortem studies of schizophrenic subjects have repeatedly revealed a reduction in dendritic complexity of neurons, as well as a decrease in the size of pyramidal cells compared to controls (Kruse and Bustillo, 2022) which likely explains the "progressive cortical thinning described with MRI in- vivo" (Stephan et al., 2009). PTSD has been less well studied in these terms, although a

pilot study of post-mortem tissue found that dendritic spines in PTSD patients tended to be immature and stubby compared to controls, whose dendritic spines were mature and sprouted like mushrooms (Krystal et al., 2017). Neuroimaging studies have also found reductions in cortical thickness, subcortical volumes, white matter pathways, and functional connectivity in patients with PTSD that correspond to symptom severity and cognitive deficits, lending support to the synaptic deficit hypothesis (Krystal et al., 2017). It is not only the event that induces PTSD; the disorder itself is a state of chronic stress whose symptoms compound the neurodegenerative process.

If we extend Krystal et al.'s (2017) theory of synaptic loss induced by chronic stress as underlying depression and PTSD to psychosis, it maps with the prevailing glutamatergic dysfunction hypothesis of schizophrenia. Stress induces a surge of glutamate in the prefrontal cortex (Popoli, 2011), which is not effectively inhibited by GABAergic interneurons. The glutamate becomes neurotoxic when it spills outside the synapse and binds with extrasynaptic NMDA receptors, shrinking dendrites and even killing neurons. Subjects with histories of childhood trauma could be expected to have fewer astroglia to absorb the excess glutamate, increasing the likelihood of neurotoxicity. Excessive glutamate release during episodes of chronic stress would also lead to a downregulation of receptors, which could theoretically also lead to glutamate hypoactivation and the "prefrontal reductions in the obligatory GluN1 subunit of the NMDA-R… repeatedly found in post-mortem tissue" of schizophrenic patients (Kruse and Bustillo, 2022). Such reductions in NMDARs could in turn be expected to correspond to language disturbances. This is not to mention additional pathways by which chronic stress may lead to neurodegeneration, such as cortisol-induced brain-derived neurotrophic factor (BDNF) downregulation or neuroinflammation.

Healing through connection and association

> In delusional speech the Other is truly excluded… there is so little truth that the subject places none there himself, and in the face of this phenomenon, this ultimately raw phenomenon, his attitude is one of perplexity. It will be a long time before he attempts to restore an order, which we shall call a delusional order, around this. (Lacan, 1993 p.53)

Lacan sees psychotic delusion as an attempt to heal (McCormick, 2022b). The subject is trying to make sense of what has happened to them, though in a way that does not accord with the reality of others. They are too fragmented – neurally and experientially – to share the same referent or big Other as neurotics do. Estranged from others and the Other, they attempt to make sense of their reality in a fundamentally narcissistic way, but delusions are nevertheless an attempt to connect the fragmented parts of themselves. On a neural level, we can imagine how a fragmented brain might try to make connections, but lacking the synapses to connect certain memories or information, these connections may not make sense to others. However subjectively true the psychotic's experience may be, their lack of resonance with shared reality presents a problem for functioning in society.

The psychotic is hardly a lost cause. The answer to this problem is obvious if we are willing to be literal enough. Dissociation, splitting, fragmentation – Freud and Breuer put forth a cure for the troubled psyche in (free) association. While Lacan warns that analysis with patients with psychotic clinical structures must take a different tack than with that of neurotics lest an episode be triggered, the associative principle stands. At the neural level, synaptic loss can be combated with strategies for synaptic connectivity. This is already taking form in current trends with cognitive remediation therapy (or, learning) and even exercise for schizophrenia, both of which support overall brain health and synaptogenesis.

To return to Lacan's sow. While she is dejected, disjointed, and fragmented, we might also consider how in this example, the delirious woman and her mother are isolated or fragmented from society. Lacan notes how the two patients felt their neighbour's presence as "intrusive" to the point of wanting to get her committed to hospital for her affair with a married man (Lacan, 1993 p.49). Isolation and fragmentation not only happen to neurons on the cellular level, or individuals on the psychic level, but on the societal level. It is not enough to help someone to connect parts of themselves and memories; people must be in association and connection with others to heal.

Conclusion

Krystal et al. (2017) report that chronic stress leads to neural atrophy, reducing neuroplasticity and impairing memory. Neuronal death and weaker neurons with fewer dendritic branches and synapses means the brain is less able to make connections - literally and figuratively. There are more gaps in our brains, our memories, and in our speech. Krystal et al.'s synaptic loss theory explains psychic and neurodegeneration on a spectrum from depression to PTSD, which I here extended to include psychosis, comparing the synaptic loss hypothesis with Lacan's image of the psychotic as fragmented.

Throughout his work Lacan refers to split-subjects as fragmented bodies, striving to be the whole selves we see reflected in our mirrors and desires. However the psychotic subject's entire reality is fragmented, because it is not held together by the Symbolic Order – so much so that their inner dialogue emanates from others. Their psychic fragmentation is reflected in cerebral degeneration: they have fewer neurons, with weaker dendrites and synapses. The cause of this fragmentation and degeneration is often chronic stress, especially in childhood, when our brains are developing, our neurons are growing, and we learn language. At least one mechanism by which childhood trauma mediates psychosis is the long-term downregulation of glial cells, especially astrocytes, which regulate glutamate and prevent excitotoxicity. Chronic or traumatic stress in early life therefore sets children up for a lifetime of nervous system deregulation, a potentially disordered relationship to the intersubjective Symbolic realm that gives us a place within society, and puts them at greater risk of developing neurodegenerative disorders including schizophrenia.

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