Scientific Research Publishing

Psychology, 2025, 16(6), 707-733
https://www.scirp.org/journal/psych
ISSN Online: 2152-7199
ISSN Print: 2152-7180

Foreclosure, Repression, and Neurodevelopmental Trauma: Revisiting Symbolization Failure and Its Organic Roots

Giuliana Galli-Carminati[1,2], Federico Carminati[2]

[1]Bundang Hospital, University of Seoul, Seoul, South Korea
[2]International Society of Multidisciplinary Psychoanalysis (SIPsyM), Geneva, Switzerland
Email: giuliana.gallicarminati@gmail.com

How to cite this paper: Galli-Carminati, G., & Carminati, F. (2025). Foreclosure, Repression, and Neurodevelopmental Trauma: Revisiting Symbolization Failure and Its Organic Roots. *Psychology, 16,* 707-733.
https://doi.org/10.4236/psych.2025.166039

Received: April 28, 2025
Accepted: June 9, 2025
Published: June 12, 2025

Abstract

This paper revisits the relations between trauma, repression, foreclosure, and neurodevelopmental disorders from a longitudinal perspective. We propose that failures in symbolization, a central concept in Freudian and Lacanian metapsychology, may stem from underlying neurophysiological vulnerabilities rather than purely psychical conflicts. Using a modified version of Baudouin's septenary model, we explore how developmental disturbances can impair psychic structuration at different stages, particularly through the failure to introduce the third element. Three clinical vignettes illustrate how trauma outcomes may diverge depending on neurodevelopmental substrates. We argue that integrating organic predispositions into psychoanalytic models may inform more targeted diagnostic and therapeutic approaches.

Keywords

Trauma, Repression, Foreclosure, Neurodevelopmental Disorders, Longitudinal Perspective

1. Introduction

This paper examines the relationships between fundamental Freudian and Lacanian mechanisms—repression, foreclosure, and symbolization, and their potential neurobiological substrates, including synaptic function and neurotransmitter activity. The nature and effects of trauma, considered by Freud and Lacan as one of the major etiological factors in mental disorders, serve as the central theme

guiding this exploration.

This paper is exploratory. It proposes a conceptual framework integrating psychoanalysis and neurodevelopmental research to generate new clinical and theoretical hypotheses. We explore the interplay between the organic (neurological structures, neurotransmitter function, and myelination) and the **symbolic-psychic** (subjective experience, unconscious processes, mechanisms such as foreclosure and repression) registers.

We acknowledge that our work remains highly speculative and that many of the links we propose, particularly between Lacanian foreclosure and neurophysiological dysfunction, are currently unprovable. We aim not to assert definitive models but to stimulate interdisciplinary dialogue. Despite the epistemological challenges, we believe that re-examining trauma through both symbolic and neurodevelopmental lenses may open new directions for research and clinical practice.

Both psychoanalysis and neurodevelopmental research investigate the human mind and its pathology, but they often have difficulty communicating because of the fundamental differences in their epistemological assumptions and methodological approaches. Moreover, the demands of their discipline, and academic careers, absorb the professionals on each side, so that they rarely have the opportunity to build bridges with other fields.

These two approaches to the workings of the human mind often seem on opposite sides of the rational-spiritual spectrum. However, it is worth recalling that the scientific positivism (Comte, 1830) of his time profoundly influenced Freud. Trained under the physiological school of Brücke and steeped in the naturalistic assumptions of 19th-century neurobiology, Freud maintained a lifelong ambition to anchor his metapsychology in neurophysiological mechanisms (Borch-Jacobsen, 2023). He envisioned building this bridge with the help of his colleague Wilhelm Fließ (Freud et al., 1995). Although such an integration remained elusive in his time, recent developments in neuroscience offer renewed potential for revisiting this foundational aspiration with tools that Freud could not have imagined.

The guiding hypothesis of this paper is to propose a "shift" of the etiopathological focus of mental disorders from the ψυχή (psyche) to the σῶμα (soma, body). In this view, psychological "dysfunctions" such as the unresolved mother-child dyad, the failed introduction of the third element, or the non-resolution of the Oedipus complex are not the root causes of the observed pathology but rather the symptoms of an underlying neurodevelopmental disorder. We are not, however, suggesting that there is somatic determinism. A specific neurodevelopmental condition has to be seen as a predisposition that can evolve into a disorder due to environmental situations and the subject's personal history. A philosophical reference for our work could be found in Merleau-Ponty's concept of lived body vs. body as object (Merleau-Ponty, 2012).

Psychoanalysis suggests that at the root of several mental disorders, we can

identify a triggering event. Freud and later Lacan have called trauma this triggering event that overwhelms the psychical equilibrium (homeostasis) and causes a pathological condition. Following these authors, we define trauma as one or more events that cause a state of psychic distress, resulting in more or less profound, harmful, and lasting alterations in an individual's psychic functioning—an alteration we refer to, collectively, as traumatic symptoms.

Since the early studies into the nature and effects of trauma, therapists have questioned the quantitative and qualitative link between the traumatic event and the resulting symptoms or pathological outcomes. The clinical observation that different individuals exposed to similar trauma develop different symptoms, which vary significantly in intensity, complicates this inquiry. In this paper, we argue that this variability may be due, at least partially, to organic and genetic differences leading to different neurodevelopmental paths. We consider that, when confronted with trauma, pre-existing neurological "dysfunctions" or fragilities, hitherto "silent," result in thoughts, behaviors, or emotions that are both a response to the traumatic fact and symptomatic of this hidden neurodevelopmental imbalance. The French philosopher Canguilhem has expressed this as: "normal and pathological are not purely biological facts." (Canguilhem, 1991) This fact could explain why, for example, even seemingly minor events during the child's development can function as traumatic triggers. We encounter this very frequently in our clinical experience.

There is a substantial and growing body of evidence linking neurodevelopmental disorders (e.g., autism spectrum disorder (ASD, ADHD, intellectual disability) to altered trauma responses. The literature also shows that the relationship is complex and bidirectional: neurodevelopmental conditions can increase vulnerability to trauma, and trauma can exacerbate or mimic neurodevelopmental symptoms. For instance, children with neurodevelopmental disorders are statistically more likely to be exposed to traumatic experiences such as abuse, neglect, bullying, or medical trauma (Mehtar & Mukaddes, 2011). There is also evidence that neurodevelopmental conditions often involve altered stress physiology, see, for instance, (Carrion et al., 2010).

Relevant to our hypothesis is the finding that early trauma can produce symptoms that resemble those of neurodevelopmental disorders, such as hypervigilance (ADHD-like), social withdrawal (autism-like), or developmental delays, leading to misdiagnosis or dual diagnosis (De Bellis & Zisk, 2014).

Moreover, it is relevant to note that several studies show a strong correlation between neurological findings and childhood trauma (Teicher & Samson, 2016; van der Kolk et al., 2005). This correlation is usually interpreted as trauma causing a lasting neurodevelopmental impact. However, these findings are also consistent with the hypothesis of a pre-existing neurodevelopmental condition leading to increased sensitivity to childhood trauma.

Let us illustrate this with a phenomenon frequently encountered in clinical practice. In the early dyadic phase (Klein, 1946; Lacan, 2006), the child is subject

to a developmental tension in which they simultaneously experience a sense of personal omnipotence and attribute an equal—or even greater—omnipotence to the caregiving figure, particularly the mother. This double attribution generates what we might call a narcissistic polarity (Freud, 1957b; Galli Carminati et al., 2020), a dynamic that plays a foundational role in forming the ego ideal and later structures dependence and rebellion. The perceived omnipotence of the mother evokes admiration and love but may also appear threatening or even abusive. From the child's perspective, the parents possess an almost divine power—inescapable even in thought. This perception alone can be traumatogenic, regardless of any actual mistreatment.

When confronted with such a patient, now an adult, the therapist is faced with a childhood memory that functions as a symptom. In this scenario, the clinician may interpret the parents, who were both deeply loved and experienced as overwhelmingly powerful, as the primary source of the trauma. The child's perception of them as "infinitely abusive" may arise precisely from the intensity of this idealized attachment. However, if the child has an underlying neurodevelopmental vulnerability, their capacity to assess relational reality may be impaired. As a result, their perception of the parents, as powerful, beloved, and threatening, may be distorted, though no less subjectively traumatic. Crucially, the presence of such a developmental condition does not preclude the possibility of actual abuse, which further complicates the therapist's interpretive task.

At this point, the caregiver will try to deploy the classic "remedies" to relieve the traumatized patient's suffering. Depending on the severity of the situation and the patient's personality, this may include different forms of psychotherapy (dynamic, CBT, EMDR, mindfulness) or the use of medication (antidepressants, antipsychotic stabilizers, anxiolytics). There are complementary approaches, such as yoga, art therapy, occupational therapy, social support, and therapeutic groups.

The nature of the trauma also influences the therapeutic approach. We can distinguish "external" and objective trauma, such as physical or verbal violence that can be objectified, and "internal" and subjective, i.e., a "disproportionate"—at least in the eyes of the therapist—reaction to an event that only a highly subjective interpretation can justify. The distinction between internal and external traumas depends on the therapist and the patient. In our practice, we have encountered cases that fall into one of these two categories and several ambiguous ones.

As we mentioned earlier, we believe that the distinction between these different traumas is more apparent than real, since the traumatic outcome, or symptom, results from the interaction between the traumatic "fact," the patient's feelings about the traumatic event, and the patient's somatic and neurobiological substrate.

2. Trauma and Emotions

When considering the subjectivity of the traumatic experience and its consequences, it is interesting to draw a parallel with the theory of emotions in psychol-

ogy, where we can identify two main trends.

Cognitive theories emphasize the role of cognitive processes in generating emotions. According to researchers of this orientation, emotions depend on a cognitive assessment of the objective situation and our physiological activation (Lazarus, 2006; Schachter & Singer, 1962).

Physiological (or peripheralist) theories, on the contrary, postulate that emotion stems primarily from the body's physiological reactions. According to the theory of James and Lange (1884-1885) (Barrett, 2017; Dewey, 1894), emotion is a consequence of bodily changes. To use an example from James: when we see a bear, our body reacts (racing heart, tremors), and then we interpret these reactions as fear. So "we are afraid because we are trembling" and not vice versa.

If we consider these two tendencies, we observe that a large part of the literature approaches trauma from a cognitivist viewpoint, i.e., focusing on the—conscious or unconscious—cognitive elaboration of the traumatic fact. According to some authors (Brewin et al., 1996), trauma results from a disturbance in the integration of memories and emotions associated with the event. Other authors (Janoff-Bulman, 2002) have proposed that trauma destroys our fundamental cognitive patterns. In cognitive behavioral therapies (CBT, EMDR) (Shapiro, 1989) trauma treatment is based on the idea that trauma can be cognitively "reprogrammed."

Psychoanalysis offers us an intermediate position. It shares with the cognitivists the primacy of the psychic elaboration of trauma and trauma as a problem of representation while introducing elements foreign to cognitivism, such as the unconscious and the drive (lust, death). Freud and the psychoanalysts agree with cognitivism in attributing a secondary role to immediate physiological reactions concerning their psychic effects.

A physiological (peripheralist) interpretation of trauma may shed light on certain clinical features that remain opaque under purely cognitive or representational models. In this approach, trauma is understood primarily as the interpretation we give to our neurophysiological reaction. This neurophysiological response ultimately depends more on our function, or dysfunction, at the level of synapses and neurotransmitters than on any cognitive process. This interpretation can help to understand the personal variability of responses to trauma: it is not the event itself that is traumatic, but our ability (or inability) to interpret our own reaction to that event.

One of the fundamental concepts in the development of dynamic psychology is the repression of trauma and its consequences on mental health. We will, therefore, take this element as a starting point for our reflection, first trying to understand the difference between the traumatic fact in itself and the psychic symptom that results from it as an effect.

As we said before, in Lacanian terms, trauma is an event we fail to symbolize (Lacan & Miller, 1998). In a peripheralist interpretation, this failure will be more at our neurophysiological and physical reaction to the event than at the actual level of facts experienced. In other words, it is not the experience of reality that

escapes our symbolic understanding but our reaction to it. Clinically, we often encounter the consequences of this. While some of our traumatized patients manage to tell us "what happened," for a significant number, there is an amnesia of their feelings related to the facts. When we ask them to describe their reaction, it is practically impossible, even if they recall the facts about the trauma.

The fact that some patients, for example, "remember nothing of their childhood" must sound like a "flashing red light" because this amnesia often conceals the trauma. In this case, we should not try to remove this amnesia from the outset. If it is there, even if it entails a significant loss of psychic energy, it has a protective function, and it is better to remain very careful.

In addition, we now know that our "unspeakable" reaction is directly inscribed "in the soma," which is probably also covered by amnesia. Research in neurophysiology shows that traumatic memory is physically inscribed in the nervous system, leading to lasting changes in neural circuits. The amygdala, a key structure in the limbic system, is overactive in people who have experienced trauma, which intensifies the emotional response and reinforces the inscription of the memory (LeDoux, 2003; Rauch, 1996; Van der Kolk, 2014). At the same time, the hippocampus, which is responsible for contextualizing memories, is often atrophied in people with posttraumatic stress disorder, which prevents the normal integration of the memory into narrative representation (Bremmer et al., 1995).

In addition, the prefrontal cortex, which regulates emotional reactions, is hypoactive in the presence of traumatic stimuli, explaining the inability to rationalize the event (Shin et al., 2006). Trauma also alters neurotransmitter levels, including increased cortisol and norepinephrine, which disrupts memory consolidation processes (Kim & Kim, 2023; Yehuda et al., 1990).

This interpretation also helps explain the phenomenon of retraumatization: when a patient attempts to recall a traumatic event, they may not simply revisit a memory but re-experience a neurophysiological reaction that remains imprinted in the nervous system. When we recall—or try to remember—the trauma, we also recover—or try to—our neurophysiological reaction that has been "encoded" forever in our nervous system. Our response remains unsymbolizable, and this inability renews the trauma. It is essential to note here that this may lead to a much stronger reaction to a debriefing. We are not dealing with the inevitably fading memory of an event but with our response to it, which we have inscribed in our soma, and it is still present. Our inability to face it brings us to relive the trauma "as if it were now."

This view also erases the difference between internal and external trauma and explains why the triggering event is so personal and variable in "objective" intensity and severity. What counts is more our "dismay" in the face of our neurophysiological reaction to the "traumatic fact" than the fact itself.

Recent meta-analyses appear to support this hypothesis, casting doubt on the clinical effectiveness of early debriefing interventions (Ancarani et al., 2025; Stileman & Jones, 2023) and even suggesting that such approaches may consolidate,

rather than mitigate, the traumatic imprint (McNally et al., 2003).

Our primary emotional response likely depends on our neurophysiological function. We can thus weave a direct link between our neurophysiological features, and fragilities, responsible for our neurophysiological reaction and the effect of trauma, which in this case should be seen as a symptom and not the root cause of our reactions. A fact is traumatic if we cannot interpret, symbolize, as Lacan would say, our response. An event is traumatic also because of the emotional reactions and sensations that we are unable to imagine.

This interpretation may explain the difficulty of Freud and psychoanalysis in assessing the importance of trauma in the mechanism of repression, especially in the face of the observation that not all patients have experienced objectively identifiable traumas.

If a traumatic event occurs, its inscription in the soma by neurophysiological mechanisms creates an uninterpretable nucleus that metaphorically forms a kind of "cyst," resulting in a tear in the psyche. The effect on the development of a possible mental illness probably depends on the moment in the psychic life at which the trauma occurs. It would, therefore, be helpful to work on the concept of trauma by considering it from a longitudinal "ontogenetic" perspective in time, chronological, and developmental. According to the interpretation proposed here, trauma is a permanent condition in the face of a neurophysiological disorder that persists in the conscious or the unconscious.

This interpretation agrees with the "classical" organicist vision in recognizing that an external adverse physical event can often be traumatic: mistreatment, injuries, illnesses, and accidents. In our opinion, the element that standardizes the nature of the trauma—whether internal or external—is that the psyche has not had the means to go beyond its physiological reaction to the event and has encysted the trauma, more or less skillfully, without being able to elaborate. This neurophysiological dysfunction may crystallize into a non-analyzable nucleus—an 'emotional event'—that resists integration and impairs adaptive psychic development at the personal and social level.

3. Baudouin's Septenary

In the following, we will concentrate on the mother-child dyad and the potential traumatic and pathogenic effects of its non-resolution. For this, we will use a model of child development taken from C. Baudouin's "Septenary" schema of the psyche (topic), modified for our purposes. We will introduce the Septenary here and explain the modifications we propose.

In 1950, the Swiss psychoanalyst Charles Baudoin attempted an ambitious and subtle synthesis between the theories of Freud and Jung, intending to show their practical and theoretical complementarity. Faithful to the Freudian concept of topics, he combined Freudian and Jungian topics in the structure of *Stoker's Cone* (Baudouin, 1950). Stoker's Cone is a graphical representation of Baudouin's Septenary, illustrating the organization of psychic instances in concentric layers sur-

rounding the Ego. In this model, the different topoi are arranged around the conscious Ego and aspire to a harmonious functioning in the spirit of the Freudian economic principle, orchestrated by a Self, resulting from a Jungian individuation process (see Figure 1 on the left).

Baudouin's innovation was to introduce a seventh instance – the Automaton – representing Freud's principle of repetition. Faithful to the psychoanalytic tradition, Baudouin attributes a degree of consciousness/unconsciousness to the different topoi.

According to Baudouin, the different instances develop progressively during the child's growth. The Automaton is a vital function that has existed since birth. The child experiences very early the pulsion and desires of the Primitive, equivalent to the Freudian Es, and also develops the "social mask," the Persona, to regulate their interaction with the social environment, reducing the socially unacceptable aspects of the Primitive. The Ego develops progressively as the child builds a sense of their individuality. The introduction of the Father and the Law symbolically castrates the child via Oedipus's complex. The psyche relegates the unavowable and forbidden desires to the Shadow and integrates the Paternal Law into the Superego. The Self is the final objective of human development and individuation since it will promote the harmonious collaboration of all the instances (see Figure 1 on the left).

In our opinion, the development of the Persona, the social mask, follows, rather than precedes, the symbolic castration and the development of the Shadow-Superego polarity; therefore, we will use the modified Septenary where the Persona and Superego are inverted. Figure 1 (on the left) illustrates our proposed inversion of the Superego and Persona within Stoker's Cone. This modification reflects our hypothesis that social adaptation (Persona) emerges only after the internalization of Law (Superego).

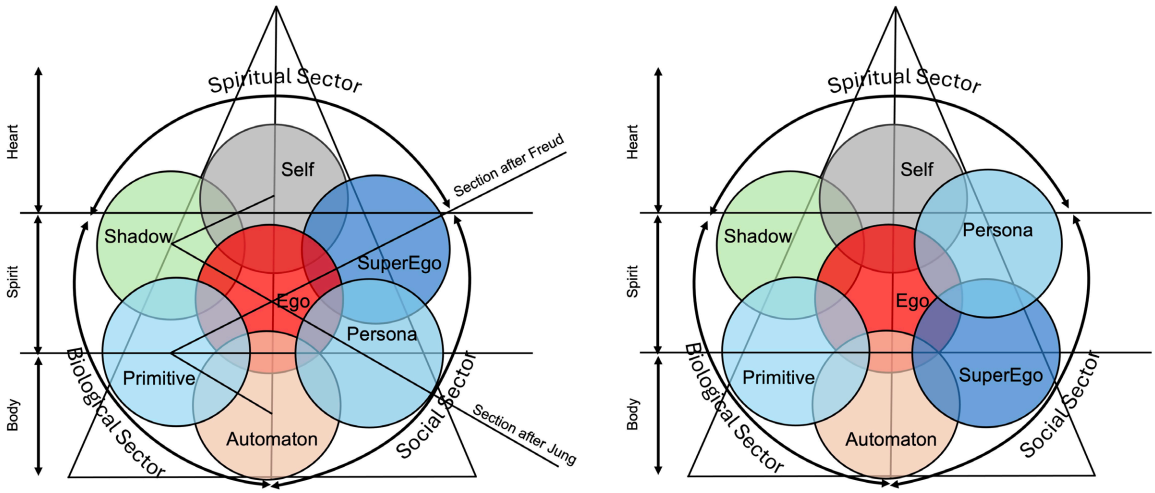

Figure 1. On the left is Stoker's Cone according to Baudouin, and on the right is our proposal for the inversion of the topical Superego and Persona.

In the description of psychic evolution in connection with the development, we will use the modified schema of Baudoin's Septenary, as we proposed in a previous paper of ours (Baudouin, 1950; Carminati & Carminati, 2019): "*The child is certainly influenced by the social context and the collective unconscious. However, the first source of frustration is the presence of the separating third element. This yanks him from the state of completeness of the child-mother dyad and forces him to begin, or better, to continue his path of individuation beyond the Automaton and the primitive.* […] *We could say that the upper corner of Stocker's triangle, the spiritual sector, is probably more related to our Persona and that our social attitude strongly depends on how in the early stage of our development, we integrated in our Superego, the third element/Law/Totem."* (see **Figure 1** on the right).

This temporal inversion of the development of the SuperEgo and the Persona is more consistent with the actual child's evolution. Placing the development of the SuperEgo during the Oedipal phase and that of the Persona after the latency period is more consistent with an early foreclosure and a later "neurotic" trauma and their effects.

4. Neurodevelopmental Mechanisms and Trauma

As we said in the previous Section, we will take the non-resolution of the mother-child dyad as an example of a potentially traumatic event. This mechanism and its dysfunctions are critical since they concern a crucial moment in the child's development. The effects of failing to resolve the dyad by introducing the third element, the Father or the Law, can be severe and long-lasting. In our clinical practice, we often encounter patients suffering from the consequences of this failure.

The mother-child dyad is a natural and necessary stage of child development (Bowlby, 1997; Dolto, 2014; Klein, 1997; Stern, 2017; Winnicott, 2021). Lacan analyzes the place of the all-powerful mother and the structuring role of the Father (le nom-du-Père) in the progressive separation of the mother-child dyad (Lacan, 1984). When the introduction of the separating third element does not occur, we have the permanence of the mother-child dyad, which can seriously harm the psychological and social development of the individual. If we want to introduce a computer analogy, we could say that the mother provides the "motherboard" of the child, while the father introduces the CPU, the heart of the "computer" that will analyze reality. If, in this construction phase, a "bug" is introduced in the "hardware," it is not easy to correct it afterward (Freléchoz, 2025).

Early trauma may come from the fact that the Mother (in capital letters because it is a concept and not a person) was unable to nurture and support the child adequately. Winnicott would say she was not "good enough" (Winnicott, 2021). This inadequacy can occur, for example, if the mother is depressed, has postpartum depression, is psychotic, has autistic traits, or is somatically ill and therefore not available, and the family and environment do not compensate for her shortcomings.

However, it can also be the case that a problem in the child's neurodevelopment, such as neurological disease or disability, may alter the evolution of their relationship with the mother. In this case, the child's inability to interpret their feelings in the period of the maternal dyad could be in itself a traumatic experience. The child's impasse in moving past the dyadic phase that they cannot interpret, symbolize, would say Lacan, generates what we described before as "a psychic cyst" that is often identified and treated as the cause and not a symptom of the trauma.

Suppose we are confronted with a patient in a state of psychological suffering, for example, due to their inability to enter adult life, because they persist in inadequate childish attitudes leading to painful social isolation and often to economic precariousness. For the therapist, it is challenging to decide whether the non-resolution of the dyad that caused an early trauma is due to the mother's inadequacy or the father's absence or is a symptom of a neurodevelopmental disorder.

According to Lacan, contact with the Real is the source of the trauma (Lacan & Miller, 1998). For Lacan, the Real is what escapes any symbolization or representation in the Symbolic register of language. It is the impossible, which can be integrated neither into the imaginary nor the symbolic, marking an irreducible lack (Lacan, 2006). When this trauma occurs during the dyadic phase, the introduction of the third element, i.e., the name of the Father (le nom-du-Père), the psyche withdraws into foreclosure[1] (Lacan & Lacan, 1997).

On the other hand, if the early trauma resulting in the non-resolution of the maternal dyad occurs during the formation of the Superego, as described in the reworked Septenary (see **Figure 1**), but the effects will manifest only after the latency period, the psyche still withdraws into foreclosure, (see **Figure 2**). In a harmonious development, around the age of two or three, the introduction of the separating third element provides the child with enough confidence to move away from the mother and open to the external world. A developmental disorder during this phase can cause a specific symptom or remain silent during the latency period.

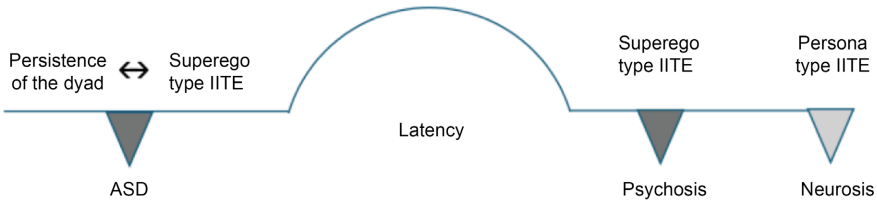

Figure 2. Longitudinal diagram of the impasse of introducing the third element (IITE) in the mother-child dyad. As explained in the text, a Superego type IITE before the latency period (about 5 - 10 years) creates a foreclosure with the appearance of an autism spectrum disorder (ASD). A Superego-type IITE before the latency period may reappear after it as a psychosis. A later IITE during the construction of the social Persona will lead to neurotic disturbances.

[1]In this article, the term *foreclosure* is used in its psychoanalytic sense, following Lacan's concept of *forclusion*—the exclusion of a fundamental signifier from the symbolic order, typically the Name-of-the-Father. This usage should not be confused with the legal definition of foreclosure, which refers to the repossession of property due to loan default.

When it reappears in adolescence, it is disruptive and may generate severe disorders such as psychosis. In this case, the individual does not reach adolescence with a post-oedipal sexualized psyche but is still steeped in the deep anxieties of early childhood.

Although early trauma typically occurs during the prelinguistic phase (Chiantaretto et al., 2022), it is nonetheless possible to conceptualize a form of failed proto-symbolization occurring even at this primitive stage. Since, according to Lacan, the unconscious is structured like a language, symbolization is a primary function that precedes language acquisition. Thus, the symbolic process is already at work, even before the child learns to speak.

The Symbolic generates the Imaginary as the ensemble of the signified forms and reveals the Real as a non-symbolizable rest. Nevertheless, even if the Real becomes perceptible through the articulation of the Imaginary and the Symbolic, it pre-exists them, i.e., it does not wait for language to exist.

The mother-child dyad spans the presymbolic and symbolic phases, but this does not mean the child is not already in the symbolic order. From birth, or even before, they are inscribed in the network of parental signifiers, which structure their relationship with the world and themselves. Therefore, language acquisition is not the condition of symbolization but rather its mature mode of expression. The process of symbolization thus precedes language and manifests differently according to the stages of development.

To return to our example of the mother-child dyad, we cite Freléchoz again (Freléchoz, 2024): *"Piera Aulagnier posits the hypothesis that in the development of the child's psyche before what she calls the Primary, there is a phase that she calls the Original. There would thus be the Originary* (*Aulagnier's pictogram*)*, the Primary* (*Freud's notion of the fantasy or word-image*)*, and the Secondary* (*representation and speech*)*."* (Aulagnier, 1964, 1981, 2009, 2016). The child is entirely dependent on the M-other/mother (the caregiver), and this encounter can be invigorating, viable, deficient, or downright deadly, the actual stages of functioning being much more detailed than described by these very simplistic registers.

Let us go back to the resolution of the dyad: we said that the source of the trauma could be an inadequacy of the maternal or paternal element, respectively, but also a developmental inadequacy of the subject affected by the trauma. In any case, we have an impasse in the introduction of the third element, the subject and source of the Superego of very early childhood. Because of this impasse, the Father (in capital letters because it is a concept and not a person) fails to support the distancing from the Mother, leading to foreclosure. The Father cannot separate the dyad, and the psyche remains included in the mother-child dyad, a Real situation that is not symbolizable and is the source of trauma and excessive jouissance (Lacan, 1996).

Lacan had studied psychoses in depth and especially in practice, and his concept of "foreclosure of the Father's name" (*forclusion du nom-du-Père*) is enlightening. He identifies the source of psychosis in the foreclosure, causing an early

blockage of the Symbolic in the construction of the Imaginary.

Suppose the trauma occurs in the Persona phase, during which the individual develops the social mask. The child has emerged from the mother-child dyad, but they have now to move past the family and into the social. If this further separation fails, we have what Freud calls repression, a notion first introduced by Freud (*Verdrängung*) in his 1894 essay "Die Abwehr-Neuropsychosen" (Freud, 1962). We can suppose that if the trauma occurs in a later phase of development, and it is not a resurgence of an early trauma muted during the latent phase, we will have a pathology in the spectrum of neuroses and not psychoses.

We remember here that we hypothesize that the psychological trauma resulting in the non-resolution of the mother-child dyad has a neurodevelopmental component. Depending on when, during the child's development, this condition disrupts the resolution of the maternal dyad, we can have different pathological outcomes. If the disruption happens very early in the pre-verbal phase, it can result in an autism spectrum disorder. Previous nomological classifications of ASD defined it, probably not without reason, as infantile psychosis (American Psychiatric Association, 1968; Bettelheim, 1967; Kanner, 1943) of varying severity depending on the neurological characteristics. We can also have ASD followed by early-onset schizophrenia, for which we proposed the name "Passerella Syndrome" (Galli Carminati et al., 2018) or a classic psychosis. We recognize that, after years of clinical and scientific work pursuing the goal of separating autism from psychosis, it is a bold proposition to bring them back closer. Still, in the present paper, we are reflecting on the links between the time of insurgence of trauma, as defined before, and the psychological development disorders. We mention ASD as an example of a deficiency in the normal development of the person, focusing more on the symptomatology related to this disorder than on its precise diagnosis.

Let us consider a psychological development along Freudian lines and suppose that the latency period could inhibit ("cover") the effects of the trauma, resulting in the non-introduction of the third element from the age of three to the onset of adolescence. In this case, the early foreclosure will be silent until the onset of adolescence, when it will manifest with psychotic symptomatology. In other words, we make the supposition that classical psychosis appears in adolescence when an impasse of the introduction of the third element (Superego Type IITE) in early childhood remains latent until adolescence.

Let us now suppose that the IITE happens in a more advanced phase, when the person confronts the (sexualized) social environment, i.e., during the development of the Persona (see **Figure 1**). There, the third element introducing the Law will be the social environment with its rules and conventions rather than the Superego tasked with allowing the separation from the Mother and the exit from the dyad. An IITE in this phase, again according to a Freudian-inspired longitudinal analysis, will cause repression with neurotic symptoms. These symptoms have a later onset and are less severe, even if they can introduce a painful destructuring of the psyche and have serious practical consequences (see **Figure 2**).

Considering the definition of borderline personality disorder (BPD) or borderline state (American Psychiatric Association, 2013; MSD, 2025) we can suppose that these disorders, which straddle psychotic and neurotic disorders, find their origin in early traumas. BPDs are mental conditions characterized by pervasive and persistent patterns of thoughts, perceptions, reactions, and relationships that result in significant suffering for the person and/or significantly impair their ability to function. Personality traits correspond to relatively stable patterns of thought and behavior in perceiving, reacting, and relating to each other. Some patients tend to be moody or closed, while others tend to be more outgoing and sociable. These disorders manifest as indelible scars of trauma as an inability to adapt to the internal and external reality in social situations.

BPDs are a psychiatric pathology when the severity of the personality traits becomes a source of maladaptive problems at work, at school, and in personal and social relations. Rigid and maladaptive attitudes characteristic of BPD prevent people from adjusting their behavior to the circumstances of life, which can lead to significant suffering for those affected and their family and professional environment

Obsessive-Compulsive Disorders (OCDs), which often accompany ASDs, psychoses, but also neurotic depressive states and personality disorders, can debut as an early depressive state already in childhood and are not specific to foreclosure or repression. The comorbidity between OCD and BPD is estimated at 5% (Thamby & Khanna, 2019). In an article by the authors in 2023, we can read: *"This case confirmed our observation, also found in other similar situations, that disorders combining ADHD, OCD, and Asperger's Syndrome with an epileptic-like syndrome, especially if present since childhood, produce an adaptation of the production of thought that leads to considering pathological functioning as normal."* (Galli Carminati et al., 2023). OCD, therefore, has a non-specific pervasiveness, and it is characterized, as Freud says in The *Rat Man* (Freud, 1957a), by a very early onset of compulsion anxiety.

Freud proposes a sexual origin of this pathology, as in the definition of anxiety hysteria (Breuer & Freud, 2022). According to the authors, the origin is much earlier and probably linked to a developmental disorder, which can occur during the period of the introduction of the Superego type third element. In a recent paper, the authors have speculated on the possibility that at the root of developmental disorders such as ASD and probably ADHD, as well as for less well defined developmental disorder, there is an epileptic-like syndrome (Galli Carminati et al., 2023) with obsessive thoughts characteristic of OCD as symptoms. This hypothesis is not in contradiction with a psychoanalytic interpretation of developmental disorders because the IITE processes described above, the failure to resolve the mother-child dyad, the superego type IITE, and its reactivation after the latency period, may be the apparent symptoms of an underlying organic problem.

In people with ADHD, the behaviors caused by the disorder are severe enough to create difficulties in daily activities. The inability to focus on their tasks causes

people with ADHD to be easily distracted, forget what they need to do, and avoid tasks that require sustained attention. It is also challenging for them to organize their activities, even leisure ones. They also tend to lose items or have frequent accidents. Hyperactivity, which is very common, manifests through frequent movements and difficulty sitting still. Impulsivity can lead to rash decisions followed by hasty actions. More than two-thirds of people with ADHD have other syndromes: mood or anxiety disorders, learning disabilities, or substance use disorders. The concomitant presence of ASDs, but also Tourette's syndrome, is also possible. We observe that the hyperactivity associated with ADHD, characterized by repetitive and often uncontrollable movements, can present superficial similarities to the compulsive behaviors seen in OCD.

OCD could appear not only as a symptom of repression but also of foreclosure and be the visible part of a trauma whose chronology may be challenging to establish without a longitudinal and organicist approach, as proposed in our hypothesis.

We suppose that the root cause of this difficulty/incapacity could be the same organic substrate of trauma, i.e., a problem at the neurophysiological level. Several authors have identified neurotransmitters at the center of this problem (Løkhammer et al., 2024; Maihofer et al., 2022; Omopo, 2024; Peel et al., 2022; Sabé et al., 2024) with deficiencies or dysregulations of the dopaminergic or serotonergic systems (Galli Carminati et al., 2022).

It is possible to hypothesize a dynamic interaction between the psychological functioning associated with the disorders of ASD, ADHD, and OCD, which are partially influenced by genetic factors, and the environmental influences that may affect the psyche and epigenetic expression. For ADHD, according to Oades (Oades, 2010), the genetics of ADHD influences the functioning of specific neurotransmitter systems, in particular that of serotonin and noradrenaline (Sonuga-Barke et al., 2009). Other studies have observed disturbances in the reuptake of this neurotransmitter or its availability. In addition, neuroscience and neurobiological research on ASD indicates the presence of abnormalities within the serotonergic system, more specifically in the circuits connecting the frontal and limbic areas (Cadman et al., 2015; Derksen et al., 2020; Girgis et al., 2011; Zandt et al., 2007).

We could also ask ourselves if the proposed pseudo-epilepsy mechanism is not "simply" a problem of defective myelination in early childhood, as discussed by Gzielo *et al.* (Gzielo & Nikiforuk, 2021). In this paper, the authors posit that oligodendrocytes and their precursors may be involved in the pathogenesis of ASD via white matter alterations and myelination. Human studies and animal models show structural changes in the corpus callosum and prefrontal cortex associated with social and behavioral deficits (Ameis et al., 2016). BTBR mice, a model of autism, exhibit increased immunoreactivity of NG2 cells, suggesting disruption of interactions between neurons and oligodendrocytes, which may be a key factor in neuronal dysfunction in ASD (Bergles & Richardson, 2016; Filley & Fields, 2016; Graciarena et al., 2019; Makinodan et al., 2012).

Reduced density of myelinated axons connecting the anterior cingulate cortex (ACC) to neighboring cortices has been observed in children with ASD (3-10 years of age), indicating early alteration (Zikopoulos et al., 2018). In addition, the proportion of thin axons (local connections) was significantly higher, while that of thick axons (long-distance connections) decreased with age in autism, unlike the control group. In addition, Trutzer et al. (Trutzer et al., 2019) have highlighted a disorganization of cortical circuits in layer one of the lateral prefrontal cortex (LPFC), reinforcing the hypothesis of altered neuronal connections in ASD.

Again, it is worth repeating that we are not simply suggesting that foreclosure *comes from* myelination failure or neuronal disorganization, but that a neurodevelopmental fragility may predispose the psyche to difficulties in symbolic elaboration, possibly aggravated by certain traumatic conditions.

The following Section will indicate possible therapeutic approaches based on the abovementioned hypothesis.

5. Therapeutic Approach

The therapeutic approach is highly dependent on the diagnosis, and it is sometimes necessary to observe the patient for some time before initiating a specific treatment. It is essential to understand whether the symptomatology is rooted in a trauma that can be early (dyadic period, Superego Type IITE in the early Superego phase, reactivation of the Persona type IITE, after the period of latency) or later (phase of social confrontation, phase of the Persona).

A hypothesis of organic dysfunction must corroborate claims about the defective– or absent – ability to recover psychic energy. As a bridge between the organicity of the trauma, and the psychoanalytic approach, it could be helpful to consider the use of an antiepileptic stabilizing and anxiolytic medication, but also to introduce into the diet substances that promote myelination such as phospholipids and sphingomyelins. Recent studies from nutritional trials in children to clinical research in psychiatry and neurology indicate that optimizing the intake of these lipids can have positive effects on myelin and various mental health indicators (cognitive development, emotional stability, stress reduction) (Schneider et al., 2019; Slykerman et al., 2024).

These results pave the way for new nutritional or pharmacological therapeutic approaches aimed at supporting proper myelination and neurological health and highlight the importance of a balanced diet of essential lipids throughout life (Fond, 2025; Kister & Kister, 2023; Mari et al., 2024; Oades et al., 2010; Stimpfl et al., 2023).

The pharmacological treatment in childhood, especially early childhood, is complex and potentially dangerous since, even if the trauma or developmental defect is clinically evident, it can be challenging to pose a precise diagnosis. If we retain the hypothesis of a problem of poor myelination, it would be much more prudent to add to the diet elements helping myelination[2].

[2]https://www.eurofins-biomnis.com/referentiel/liendoc/precis/PHOSPHOLIPIDES.pdf

Moreover, we can ask ourselves if a therapeutic attempt to recall the trauma to try re-elaborating and overcoming it is beneficial. The pathological situation has been generated by the psyche's inability to elaborate and overcome the traumatic event, preventing proper and adaptive personal and social development. The re-evocation of the trauma may simply lead to retraumatization with the risk of depressive or psychotic decompensation.

A colleague psychoanalyst thus expressed our hypothesis while commenting on an ancient statue of the Virgin and Child accidentally decapitated: *"The Mother-Child dyad after trauma: the 'dependence' child in disarray* (Winnicott, 2021) *is projected outside. The dyad is composed only of the cleaved Mother* (*Protector/Monstrous*)*, and the child is symbolically foreclosed* (*head cut off*) *while the body remains. The suffering body of the dyadic couple maintains the memory of the foreclosed* (*rather than repressed*) *head. And that is the problem: treating repression does not solve foreclosure. Therefore, we agree that if the work on repression continues without relieving the discomfort, the therapist must question the presence of a possible foreclosure and change their approach.*

Virgin and Child. Salle des États of the Musée des Beaux-Arts de Brou, collection from the time of Margaret of Austria (1480-1530)

If the therapist's vocation as a caregiver comes from the foreclosure of 'their' child in distress with projection onto the patient, the omnipotence of his mother-protector dyad/foreclosed child projected (*'felt' or 'sensed' in the Real - in fact on the patient*) *can blind them. As long as the therapist does not realize their blind spot* (*due to foreclosure and not repression*)*, there is only an encounter of two mother/child-foreclosed dyads in a mirror, and the patient's dependent child does not* (*still*) *emerge..."* (Mirafiori, 2025)

The authors wonder whether long-term psychotherapeutic care, without an adequate – or at least considered – developmental diagnosis and psychopharmacological (and dietary?) support, is not almost iatrogenic as it risks exhausting the

subject's already reduced energy-recovery capacities.

While remaining very cautious about hypotheses widely publicized in the recent past about the "dietary" causes of mental disorders, such as heavy metal poisoning (Galli-Carminati et al., 2006; Richmond & Goldblatt, 1998; Wakefield et al., 2002; Wakefield & Montgomery, 1999) or disorders related to subclinical candidiasis, it is interesting to note that Avoidant/Restrictive Food Intake Disorder (ARFID) and ASD frequently coexist. In addition, people with ARFID are more likely to have autism than those who do not have this eating disorder (Osetrova et al., 2024; Yap et al., 2023).

The primary clinical consequence of our hypothesis is to be very careful when treating patients with traumatic experiences. The possible presence of a neurodevelopmental congenital co-factor should lead the therapist to be wary of "one size fits all" approaches. Debriefing, cognitive-behavioral therapies, EMDR, and support groups are all valid instruments, but they could be ineffective or even dangerous for some patients. Dynamic psychotherapy and psychoanalysis are less invasive and, in a sense, safer options, but they tend to be slow, and sometimes the patient's disadaptive behaviors or mood disorders need to be addressed urgently. Pharmacology is often necessary.

As examples, we will now describe clinical situations syntheses of real cases. These clinical vignettes illustrate how trauma, whether situated in foreclosure or repression, can shape psychic organization and symptom formation.

We have rigorously anonymized the three clinical vignettes presented. According to the regulations in force in Switzerland, the presentation of individual clinical cases does not require the approval of an ethics committee as long as no identifiable data is used and the authors ensure compliance with fundamental ethical principles.

6. Persistence of Dyad in Superego Type ITTE Clinical Vignette: Mrs. Robin

Mrs. Robin, a woman in her sixties, consults because she continues to experience severe relational difficulties at work and suffers from chronic exhaustion that worries her. Ms. Robin has a long history of psychotherapy, having experienced a problematic adolescence with a conflictual bond with her mother and stepfather. Her biological father had left Switzerland immediately after Mrs. Robin was born. Ms. Robin has a high-level university education but did not pursue an academic career because she considered it too competitive and stressful. After a few sessions, mainly about work, Mrs. Robin begins to talk about her mother, who represents the "big question" of her life.

On the one hand, she gets angry with her mother almost every time she sees her, which happens frequently since they live in the same town. On the other hand, she cannot live without thinking about what her mother would say or do in this or that situation she encounters. Mrs. Robin never married and lives alone without children. She says she never wanted to have a child, and this proves her wis-

dom, as her mother gave her a very negative perspective of motherhood Ms. Robin was diagnosed with dysthymia and Asperger's syndrome in her thirties.

The situation at work worries her so much that we wonder if work and relationship difficulties do not hide a much deeper disorder. Mrs. Robin is highly projective, feels poorly judged by colleagues and superiors, and periodically experiences intense fatigue that she attributes only partially to work. Mrs. Robin says she has "a bike in her head" (recurrent obsessive thoughts), which exhausts her and sometimes makes it difficult for her to fall asleep. When she is on leave, the "bike" bothers her much less; she "manages to forget herself" and relax during her hikes.

Mrs. Robin oscillates between an almost childlike satisfaction with her life, her hobby as a solo hiker, and her long weekends due to reduced working hours and deep feelings of isolation and emptiness that trigger severe anxieties.

Mrs. Robin thinks with terror of the moment when her mother will grow old and die and cannot imagine her life continuing after her disappearance. For Mrs. Robin, life is meaningless without her mother; relationships with men end with reciprocal rejections and almost with relief.

We realize that the many psychotherapeutic attempts aimed, according to Mrs. Robin, at overcoming her relational difficulties with her mother and, more generally, with everyone, had a fate parallel to her relationships with men. After one year, she experiences a gradual but rapid loss of interest and a need to drop everything and return to the eternal fundamental problem of the relationship with her mother.

Mrs. Robin does not present any psychotic symptoms. Still, in addition to mood disorders, she suffers from severe OCD, which she does not like to discuss but which invades her days and is partly the cause of her sleep disorders.

After a period of follow-up at our practice, Mrs. Robins will continue her follow-up and her antidepressant treatment in another town, where she will live with her mother after having taken early retirement.

The absence of overt psychotic symptoms suggests a structure leaning toward neurosis, though the unresolved dyadic entrapment hints at an underlying foreclosure mechanism rather than classic repression.

7. Post-Latency Superego Type IITE Clinical Vignette: Antoine

Antoine is a 23-year-old young man who consults after leaving his previous therapist. His father, a well-known political figure, directed him to our office, worried about his son's social difficulties at school and feeling utterly helpless in the face of rebellions and crises, which, in his opinion, vastly exceed normal adolescent behavior. After a relatively successful primary and secondary education, Antoine suddenly left high school because he experienced the school atmosphere as extremely stressful and, simultaneously, suffered a paralyzing feeling of boredom. He will try to attend General Education Studies, but he leaves after a year. He

cannot fit into an apprenticeship, and he does not have a clear idea of what he wants to do.

After one more failure in the workshop, which accepted him for a trial internship, he spent a few days in a rehabilitation clinic. Following a brief improvement, Antoine resumed his follow-up but quickly relapsed into severe insomnia and profound fatigue. Antoine arrived at our first appointment in deep sadness and anger against the family, the teachers, the caregivers, the course managers, and finally, against the whole world. In the initial sessions, he describes a sense of sidereal emptiness. On our part, we note an alerting state of almost mental confusion. During a calmer session, Antoine reported that, from the age of sixteen, he had experienced a subjective sense of cognitive decline – a disturbing and panic-inducing phenomenon that he found difficult to acknowledge. He described it with a facade of nonchalance, leading to misunderstanding by his family and teachers alike.

When reviewing his file, we find a list of diagnoses that range from ADHD with autistic traits to borderline personality disorder. These diagnoses were not "wrong," as they correctly identified attention deficit, impaired theory of mind, and relational distancing. However, they remain partial and fail to capture the overarching structure of Antoine's condition. With a more in-depth anamnesis, we realize that his vision of his family, and especially of the father, remains extremely childish as he saw him as an all-powerful being, without any possibility of re-elaborating this vision of his father. The bond with the mother was also very infantile: on the one hand, Antoine wanted to become independent; on the other, he felt a panicked fear of loneliness at the idea of not living with her.

Upon further examination of his childhood, we discovered that, after a normal birth, Antoine had significant nutritional difficulties and intolerances to different foods. Already in kindergarten, he alternated between profound passivity and anger, probably underestimated by teachers and parents. When his younger brother, five years his junior, was born, Antoine went through a period marked by intense whims and emotional instability. The two brothers were never able to establish a sense of complicity. Nevertheless, Antoine described a rather serene childhood and a challenging and troubled school career.

After about six months of follow-up, we proposed to Antoine a clinical evaluation because we had the feeling of facing a psychotic disorder dominated by negative symptomatology and requiring a more structured follow-up than possible in private practice.

The evaluation confirmed a diagnosis of simple schizophrenia, aligning with the clinical picture of post-latency decompensation after a failed separation process. Antoine will pursue his follow-up in a psychiatric institution, continuing the psychotropic treatment we prescribed, which he tolerated well.

In Antoine's case, a failed introduction of the third element during the Superego phase remained silent till after the latency period. The result was the development of a classical psychosis during adolescence.

8. Persona Type ITTE Clinical Vignette: Mr. Panolis

Mr. Panolis is a 55-year-old professional cellist. He consults under the advice of a girlfriend with whom he lived for two years as a couple and with whom he maintains a friendship. He lives alone, has never been married, and has no children. Following prolonged interpersonal conflict within the first violin, Mr. Panolis resigned from the orchestra after 20 years, triggering a profound professional crisis and social withdrawal.

He says he is tired and unmotivated and would like to leave Switzerland to live "differently" in the south of France. This project has no practical anchor because he lives on his savings and refuses to file for unemployment benefits. M. Panolis finds society unbearably deceiving. He does not want to thank or owe anyone anything, so he refuses to use what we constantly remind him of as his rights.

He describes himself as a shy child with good school grades but a constant fear of others. He was taller than his schoolmates, but he never wanted to show his strength and was, therefore, too obedient, too kind, and too polite a kid to have friends.

His parents had fled Latin America for political reasons, and he was born shortly after they arrived in Europe, in very precarious conditions. Two sisters were then born, and little by little, the family found a relatively comfortable economic and social situation. Mr. Panolis describes his father as a true hero, idealizes his political struggle, and suffers from a constant sense of inferiority towards him, a feeling that increased significantly after his death when the patient was in his thirties. The mother, with whom he had a profound and very exclusive bond, died five years ago. This second death triggered a deep crisis with depressive and obsessive symptoms, as well as a worsening of his social problems. Mr. Panolis realizes that his conflict with the first violin, who reminded him of his father, is related to his repressed aggressiveness towards the father's image, which is so perfect and untouchable. Mr. Panolis spends a significant portion of the sessions discussing politics, social injustice, and the folly of humanity that lives only for money. He is also steadfast in his opinions about music, which he finds too commercially exploited, with no room for new acoustic techniques. He says he is glad to have left the orchestra that only offered old things, but at the same time, with savings dwindling, he finds himself faced with inevitable compromises to find work.

Mr. Panolis was distraught by this; his dream of music beyond the notes was crumbling because the participants of the group "L'Archetto Virtuale," with which he wanted to start a new work activity, had all left for various reasons. One died, another abandoned music, and two others had other commitments. His former partner, although supportive and on good terms with him, has started a new relationship and is distancing herself. Mr. Panolis finds himself alone with his unrealistic dreams of success in experimental music. He is in a difficult financial situation caused by his categorical refusal to file for unemployment and find work, overwhelmed by his inability to sell himself in classical music.

After almost four years of follow-up with psychopharmacological support that

he finally accepted, Mr. Panolis decided to leave for Canada to work as a music teacher in a private high school in Montreal.

Mr. Panolis has overcome the maternal dyad but remains oppressed by an unrealistic and threatening paternal figure, preventing him from finding his place in society, we are in a Persona like IITE situation. He sees accepting social rules and conventions as an unacceptable treason of his "higher values." Mr. Panolis' persistent inability to integrate into social structures reflects a failure in the formation of a functional social Persona rooted in unresolved repression of paternal authority and unmanageable internalized demands of perfection. This failure is the cause of his neurotic inability to find his place in society.

These three vignettes, which are, we repeat, syntheses of different cases and highly anonymized, can guide clinicians to a diagnostic reflection and show the emergence of varying psychodynamics. In the case of Mrs. Robin, the dyad is not resolved, and the separating third of the Superego type is not integrated into the psyche of the patient, who remains frozen in a very primitive development period. Antoine, too, has not been able to move past the maternal dyad during the phase of the formation of the Superego, but he has been able to traverse the latency period almost without severe symptoms, to collapse disruptively in the first adolescence. In the case of M. Panolis, we observe significant difficulty in introducing the normative third element of the Persona type, preventing him from living a satisfactory adult social life. From a psychoanalytic angle, the first two cases are characterized by the foreclosure of the name of the father/separating third element of the dyad, the third by the repression of the aggressiveness towards the father/authority/law. While these hypotheses concern a possible somatic predisposition, the patient's subjective history and symbolic elaboration remain irreducible and unique.

9. Conclusions

We have revisited the concept of trauma and its effects from a psychoanalytic and neurodevelopmental perspective. We propose that distinguishing early from late trauma and the resulting mechanisms of foreclosure versus repression, offers significant clinical utility.

The psychoanalytic perspective is all the more relevant because it allows us to observe symptoms through the lens of psychodynamic mechanisms. The integration of Lacanian concepts, particularly the failure of symbolization and the foreclosure of the Name-of-the-Father, is especially relevant in a reflection on psychoses and developmental disorders, as is a reading of development with the common thread of Freudian theory.

On the clinical and therapeutic level, the debate on posttraumatic debriefing remains open. Recent studies dealing with it question its effectiveness, evoke a risk of trauma consolidation, and emphasize that forcing a patient to verbalize a traumatic event too early can strengthen the neural connections associated with stress rather than attenuate it and, therefore, reactivate the traumatic experience.

We have situated the trauma resulting in the failed resolution of the dyads, Superego type IITE, before and after the latency period, and Persona Type IITE in the process of neuropsychic development, which can suggest avenues for better understanding individual differences in the face of traumatic experiences. We believe it would be helpful not to exclude consideration of concomitant causes related to deficiencies in food components that support myelination, especially since access to these substances is easy, safe, and not prohibitively expensive.

Our hypothesis should be substantiated by experimental evidence. It would be necessary to perform longitudinal studies correlating the presence of neurodevelopmental disorders with the severity of the reaction to trauma and the period in the child's development when the trauma has occurred. We recognize that such studies would be extremely challenging to conduct. The very idea of collecting a representative sample of individuals who "could be subject" to trauma and testing them for neurodevelopmental problems before the expected trauma is both practically and ethically problematic. The possibility of performing tests on animals is ethically sensitive and poorly representative of the human condition. The systematic observation of children and the description of case reports could provide precious indications on the nature and effects of trauma.

Another issue with our hypothesis is that the current status of the studies linking trauma and neurodevelopmental disorders does not allow us to indicate which neurodevelopmental trait is correlated with a specific outcome (foreclosure or repression). We hope that, with the accumulation of experimental evidence in this area, we will be able to refine, or falsify, our proposition in this sense.

We argue that understanding trauma requires listening not only to what was lived, but to what the body itself could not interpret. A developmental and neurophysiological reading of trauma may thus inform more targeted, phase-specific therapeutic interventions, combining psychoanalytic insight with neurodevelopmental support. Alternative interpretations could argue that the link between neurophysiological vulnerability and foreclosure is too indirect to establish. Future studies must avoid biological reductionism and maintain the irreducible complexity of psychic structures. Controlled longitudinal studies would be necessary to validate or invalidate our hypothesis of an organic cause at the root of trauma during development.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

Ameis, S. H., Lerch, J. P., Taylor, M. J., Lee, W., Viviano, J. D., Pipitone, J. et al. (2016). A Diffusion Tensor Imaging Study in Children with ADHD, Autism Spectrum Disorder, OCD, and Matched Controls: Distinct and Non-Distinct White Matter Disruption and Dimensional Brain-Behavior Relationships. *American Journal of Psychiatry, 173,* 1213-1222. https://doi.org/10.1176/appi.ajp.2016.15111435

American Psychiatric Association (1968). *Diagnostic and Statistical Manual of Mental Dis-*

orders: DSM-II (2nd ed.). American Psychiatric Association.

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). American Psychiatric Association. https://doi.org/10.1176/appi.books.9780890425596

Ancarani, F., Añaños, P. G., Gutierrez, B., Pérez-Nievas, J., Vicente-Rodríguez, G., & Marco, F. G. (2025). The Effectiveness of Debriefing on the Mental Health of Rescue Teams: A Systematic Review. *International Journal of Environmental Research and Public Health, 22,* Article 590.

Aulagnier, P. (1964). Remarques sur la structure psychotique. *Psychanalyse: Revue de La Société Française de Psychanalyse, 8,* 47-66.

Aulagnier, P. (1981). Du langage pictural au langage de l'interprète. *Topique, 26,* 29-54.

Aulagnier, P. (2009). *Les destins du plaisir: Aliénation, amour, passion/monograph* (3rd éd.). Presses universitaires de France.

Aulagnier, P. (2016). *Un interprète en quête de sens.* Éditions Payot & Rivages.

Barrett, L. F. (2017). *How Emotions Are Made: The Secret Life of the Brain.* Mariner Books.

Baudouin, C. (1950). *De l'instinct à l'esprit.* Desclée de Brouwer.

Bergles, D. E., & Richardson, W. D. (2016). Oligodendrocyte Development and Plasticity. *Cold Spring Harbor Perspectives in Biology, 8,* a020453. https://doi.org/10.1101/cshperspect.a020453

Bettelheim, B. (1967). *The Empty Fortress: Infantile Autism and the Birth of the Self.* Free Press.

Borch-Jacobsen, M. (2023). *Freud's Thinking: An Introduction.* Cambridge University Press. https://doi.org/10.1017/9781009371117

Bowlby, J. (1997). *Attachment: Vol. 1 of the "Attachment and Loss" Trilogy.* Pimlico.

Bremmer, J., Randall, P., Bronen, R., Seibyl, J., Southwick, R., Delaney, R., McCarthy, G., Charney, D., & Innis, R. (1995). MRI-Based Measurement of Hippocampal Volume in Patients with Combat-Related Posttraumatic Stress Disorder. *American Journal of Psychiatry, 152,* 973-981. https://doi.org/10.1176/ajp.152.7.973

Breuer, J., & Freud, S. (2022). *Studies in Hysteria.* Legare Street Press.

Brewin, C. R., Dalgleish, T., & Joseph, S. (1996). A Dual Representation Theory of Posttraumatic Stress Disorder. *Psychological Review, 103,* 670-686. https://doi.org/10.1037/0033-295x.103.4.670

Cadman, T., Spain, D., Johnston, P., Russell, A., Mataix-Cols, D., Craig, M. et al. (2015). Obsessive-compulsive Disorder in Adults with High-functioning Autism Spectrum Disorder: What Does Self-report with the OCI-R Tell Us? *Autism Research, 8,* 477-485. https://doi.org/10.1002/aur.1461

Canguilhem, G. (1991). *The Normal and the Pathological.* Zone Books.

Carminati, G. G., & Carminati, F. (2019). The Marauding Paths of a Group Training. *Psychology, 10,* 1959-1983. https://doi.org/10.4236/psych.2019.1014126

Carrion, V. G., Haas, B. W., Garrett, A., Song, S., & Reiss, A. L. (2010). Reduced Hippocampal Activity in Youth with Posttraumatic Stress Symptoms: An fMRI Study. *Journal of Pediatric Psychology, 35,* 559-569. https://doi.org/10.1093/jpepsy/jsp112

Chiantaretto, J. F., Cohen de Lara, A., Houssier, F., & Matha, C. (2022). *Aux origines du je: L'œuvre de Piera Aulagnier.* Ithaque.

Comte, A. (1830). *Cours de philosophie positive (Vols. 1-6).* Bachelier.

De Bellis, M. D., & Zisk, A. (2014). The Biological Effects of Childhood Trauma. *Child and*

Adolescent Psychiatric Clinics of North America, 23, 185-222. https://doi.org/10.1016/j.chc.2014.01.002

Derksen, M., Feenstra, M., Willuhn, I., & Denys, D. (2020). The Serotonergic System in Obsessive-Compulsive Disorder. *Handbook of Behavioral Neuroscience, 31,* 865-891. https://doi.org/10.1016/b978-0-444-64125-0.00044-x

Dewey, J. (1894). The Theory of Emotion: I: Emotional Attitudes. *Psychological Review, 1,* 553-569. https://doi.org/10.1037/h0069054

Dolto, F. (2014). *L'image inconsciente du corps.* Éd. Points.

Filley, C. M., & Fields, R. D. (2016). White Matter and Cognition: Making the Connection. *Journal of Neurophysiology, 116,* 2093-2104. https://doi.org/10.1152/jn.00221.2016

Fond, G. (2025). *Bien nourrir son cerveau: Contre le stress, l'anxiété, la dépression, le déclin cognitif.* Odile Jacob.

Freléchoz, T. (2024). *Balade avec mes « influenceurs », Tome II. Cahiers de la SIPsyM N. 52.* http://www.sipsym.com/images/CahiersSIPsyM/N52-Balade-II.pdf

Freléchoz, T. (2025). *Private Communication.*

Freud, S. (1957a). Five Lectures on Psycho-Analysis. In J. Strachey (Ed.), *The Standard Edition of the Complete Psychological Works of Sigmund Freud (vol. 11)* (pp. 1-56). Hogarth Press and the Institute of Psycho-Analysis.

Freud, S. (1957b). On Narcissism: An Introduction. In J. Strachey (Ed.), *The Standard Edition of the Complete Psychological Works of Sigmund Freud (Vol. 14)* (pp. 67-102). Hogarth Press.

Freud, S. (1962). The Neuro-Psychoses of Defence. In J. Strachey (Ed.), *The Standard Edition of the Complete Psychological Works of Sigmund Freud (Vol. 3)* (pp. 41-61). Hogarth Press.

Freud, S., Masson, J. M., & Fliess, W. (1995). *The Complete Letters of Sigmund Freud to Wilhelm Fliess, 1887-1904.* Belknap Press of Harvard University Press.

Galli Carminati, G., Buttex, A., Zecca, G., & Carminati, F. (2023). Epileptic-Like Disorder as an Underlying Condition in ASD, OCD, and ADHD. *Psychology, 14,* 1573-1583. https://doi.org/10.4236/psych.2023.1410090

Galli Carminati, G., Carminati, F., Zecca, G., & Zecca-Tagan, C. (2022). Asperger's Syndrome, Obsessive-Compulsive Disorder and Attention Deficit Comorbidity. *Psychology, 13,* 964-978. https://doi.org/10.4236/psych.2022.137066

Galli Carminati, G., Freléchoz, T., & Carminati, F. (2020). *Dans les tréfonds de l'Œdipe. Cahiers de la SIPsyM N. 31.* http://www.sipsym.com/images/CahiersSIPsyM/N31-LesTrefonds.pdf

Galli Carminati, G., Tagan, C., Zecca, G., & Carminati, F. (2018). Between Autistic Spectrum Disorder (ASD) and Childhood Onset Schizophrenia (COS): A Proposal for a Passerella Syndrome. *Neuropsychiatry, 8,* 1239-1248. https://doi.org/10.4172/neuropsychiatry.1000453

Galli-Carminati, G., Chauvet, I., & Deriaz, N. (2006). Prevalence of Gastrointestinal Disorders in Adult Clients with Pervasive Developmental Disorders. *Journal of Intellectual Disability Research, 50,* 711-718. https://doi.org/10.1111/j.1365-2788.2006.00833.x

Girgis, R. R., Slifstein, M., Xu, X., Frankle, W. G., Anagnostou, E., Wasserman, S. et al. (2011). The 5-HT 2A Receptor and Serotonin Transporter in Asperger's Disorder: A PET Study with [^{11} C]MDL 100907 and [^{11} C]DASB. *Psychiatry Research: Neuroimaging, 194,* 230-234. https://doi.org/10.1016/j.pscychresns.2011.04.007

Graciarena, M., Seiffe, A., Nait-Oumesmar, B., & Depino, A. M. (2019). Hypomyelination

and Oligodendroglial Alterations in a Mouse Model of Autism Spectrum Disorder. *Frontiers in Cellular Neuroscience, 12,* Article 517. https://doi.org/10.3389/fncel.2018.00517

Gzielo, K., & Nikiforuk, A. (2021). Astroglia in Autism Spectrum Disorder. *International Journal of Molecular Sciences, 22,* Article 11544. https://doi.org/10.3390/ijms222111544

Janoff-Bulman, R. (2002). *Shattered Assumptions.* Free Press.

Kanner, L. (1943). Autistic Disturbances of Affective Contact. *Nervous Child, 2,* 217-250.

Kim, E. J., & Kim, J. J. (2023). Neurocognitive Effects of Stress: A Metaparadigm Perspective. *Molecular Psychiatry, 28,* 2750-2763. https://doi.org/10.1038/s41380-023-01986-4

Kister, A., & Kister, I. (2023). Overview of Myelin, Major Myelin Lipids, and Myelin-Associated Proteins. *Frontiers in Chemistry, 10,* Article 1041961. https://doi.org/10.3389/fchem.2022.1041961

Klein, M. (1946). Notes on Some Schizoid Mechanisms. *The International Journal of Psycho-Analysis, 27,* 99-110.

Klein, M. (1997). *The Psycho-Analysis of Children.* Vintage.

Lacan, J. (1984). *Les complexes familiaux dans la formation de l'individu.* Navarin.

Lacan, J. (1996). *Le séminaire de Jacques Lacan. 3: Les psychoses, 1955-1956.* Éd. du Seuil.

Lacan, J. (2006). *Écrits: The First Complete Edition in English* (Translated by B. Fink). W. W. Norton & Company.

Lacan, J., & Lacan, J. (1997). *The Psychoses 1955-1956.* W.W. Norton & Company.

Lacan, J., & Miller, J. A. (1998). *The Four Fundamental Concepts of Psychoanalysis.* W.W. Norton & Company.

Lazarus, R. S. (2006). *Stress and Emotion: A New Synthesis.* Springer Publishing Company.

LeDoux, J. (2003). The Emotional Brain, Fear, and the Amygdala. *Cellular and Molecular Neurobiology, 23,* 727-738. https://doi.org/10.1023/a:1025048802629

Løkhammer, S., Koller, D., Wendt, F. R., Choi, K. W., He, J., Friligkou, E. et al. (2024). Distinguishing Vulnerability and Resilience to Posttraumatic Stress Disorder Evaluating Traumatic Experiences, Genetic Risk and Electronic Health Records. *Psychiatry Research, 337,* Article ID: 115950. https://doi.org/10.1016/j.psychres.2024.115950

Maihofer, A. X., Choi, K. W., Coleman, J. R. I., Daskalakis, N. P., Denckla, C. A., Ketema, E. et al. (2022). Enhancing Discovery of Genetic Variants for Posttraumatic Stress Disorder through Integration of Quantitative Phenotypes and Trauma Exposure Information. *Biological Psychiatry, 91,* 626-636. https://doi.org/10.1016/j.biopsych.2021.09.020

Makinodan, M., Rosen, K. M., Ito, S., & Corfas, G. (2012). A Critical Period for Social Experience-Dependent Oligodendrocyte Maturation and Myelination. *Science, 337,* 1357-1360. https://doi.org/10.1126/science.1220845

Mari, J., Dieckmann, L. H. J., Prates-Baldez, D., Haddad, M., Rodrigues da Silva, N., & Kapczinski, F. (2024). The Efficacy of Valproate in Acute Mania, Bipolar Depression and Maintenance Therapy for Bipolar Disorder: An Overview of Systematic Reviews with Meta-analyses. *BMJ Open, 14,* e087999. https://doi.org/10.1136/bmjopen-2024-087999

McNally, R. J., Bryant, R. A., & Ehlers, A. (2003). Does Early Psychological Intervention Promote Recovery from Posttraumatic Stress? *Psychological Science in the Public Interest, 4,* 45-79. https://doi.org/10.1111/1529-1006.01421

Mehtar, M., & Mukaddes, N. M. (2011). Posttraumatic Stress Disorder in Individuals with Diagnosis of Autistic Spectrum Disorders. *Research in Autism Spectrum Disorders, 5,* 539-546. https://doi.org/10.1016/j.rasd.2010.06.020

Merleau-Ponty, M. (2012). *Phenomenology of Perception* (Translated by D. A. Landes).

Routledge.

Mirafiori, G. (2025). *Private Communication.*

MSD (2025). *Présentation des troubles de la personnalité-Troubles mentaux.* Manuels MSD pour le grand public. https://www.msdmanuals.com/fr/accueil/troubles-mentaux/troubles-de-la-personnalité/présentation-des-troubles-de-la-personnalité

Oades, R. D. (2010). The Role of Serotonin in Attention-Deficit Hyperactivity Disorder (ADHD). *Handbook of Behavioral Neuroscience, 21,* 565-584. https://doi.org/10.1016/s1569-7339(10)70101-6

Oades, R. D., Dauvermann, M. R., Schimmelmann, B. G., Schwarz, M. J., & Myint, A. (2010). Attention-deficit Hyperactivity Disorder (ADHD) and Glial Integrity: S100B, Cytokines and Kynurenine Metabolism—Effects of Medication. *Behavioral and Brain Functions, 6,* Article No. 29. https://doi.org/10.1186/1744-9081-6-29

Omopo, O. E. (2024). Exploring Post-Traumatic Stress Disorder: Causes, Diagnostic Criteria, and Treatment Options. *International Journal of Academic Information Systems Research, 8,* 35-44.

Osetrova, M., Tkachev, A., Mair, W., Guijarro Larraz, P., Efimova, O., Kurochkin, I. et al. (2024). Lipidome Atlas of the Adult Human Brain. *Nature Communications, 15,* Article No. 4455. https://doi.org/10.1038/s41467-024-48734-y

Peel, A. J., Purves, K. L., Baldwin, J. R., Breen, G., Coleman, J. R. I., Pingault, J. et al. (2022). Genetic and Early Environmental Predictors of Adulthood Self-Reports of Trauma. *The British Journal of Psychiatry, 221,* 613-620. https://doi.org/10.1192/bjp.2021.207

Rauch, S. L. (1996). A Symptom Provocation Study of Posttraumatic Stress Disorder Using Positron Emission Tomography and Script-Driven Imagery. *Archives of General Psychiatry, 53,* 380-387. https://doi.org/10.1001/archpsyc.1996.01830050014003

Richmond, P., & Goldblatt, D. (1998). Autism, Inflammatory Bowel Disease, and MMR Vaccine. *The Lancet, 351,* 1355-1356. https://doi.org/10.1016/s0140-6736(05)79081-4

Sabé, M., Chen, C., El-Hage, W., Leroy, A., Vaiva, G., Monari, S. et al. (2024). Half a Century of Research on Posttraumatic Stress Disorder: A Scientometric Analysis. *Current Neuropharmacology, 22,* 736-748. https://doi.org/10.2174/1570159x22666230927143106

Schachter, S., & Singer, J. (1962). Cognitive, Social, and Physiological Determinants of Emotional State. *Psychological Review, 69,* 379-399. https://doi.org/10.1037/h0046234

Schneider, N., Hauser, J., Oliveira, M., Cazaubon, E., Mottaz, S. C., O'Neill, B. V. et al. (2019). Sphingomyelin in Brain and Cognitive Development: Preliminary Data. *ENEURO, 6,* ENEURO.0421-18.2019. https://doi.org/10.1523/eneuro.0421-18.2019

Shapiro, F. (1989). Efficacy of the Eye Movement Desensitization Procedure in the Treatment of Traumatic Memories. *Journal of Traumatic Stress, 2,* 199-223. https://doi.org/10.1002/jts.2490020207

Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, Medial Prefrontal Cortex, and Hippocampal Function in PTSD. *Annals of the New York Academy of Sciences, 1071,* 67-79. https://doi.org/10.1196/annals.1364.007

Slykerman, R., Davies, N., Fuad, M., & Dekker, J. (2024). Milk Fat Globule Membranes for Mental Health across the Human Lifespan. *Foods, 13,* Article 1631. https://doi.org/10.3390/foods13111631

Sonuga-Barke, E. J. S., Oades, R. D., Psychogiou, L., Chen, W., Franke, B., Buitelaar, J. et al. (2009). Dopamine and Serotonin Transporter Genotypes Moderate Sensitivity to Maternal Expressed Emotion: The Case of Conduct and Emotional Problems in Attention

Deficit/hyperactivity Disorder. *Journal of Child Psychology and Psychiatry, 50,* 1052-1063. https://doi.org/10.1111/j.1469-7610.2009.02095.x

Stern, D. N. (2017). *Le monde interpersonnel du nourrisson: Une perspective psychanalytique et développementale* (4e éd.). Presses univercity de France.

Stileman, H. M., & Jones, C. A. (2023). Revisiting the Debriefing Debate: Does Psychological Debriefing Reduce PTSD Symptomology Following Work-Related Trauma? A Meta-analysis. *Frontiers in Psychology, 14,* Article 1248924. https://doi.org/10.3389/fpsyg.2023.1248924

Stimpfl, J. N., Mills, J. A., & Strawn, J. R. (2023). Pharmacologic Predictors of Benzodiazepine Response Trajectory in Anxiety Disorders: A Bayesian Hierarchical Modeling Meta-Analysis. *CNS Spectrums, 28,* 53-60. https://doi.org/10.1017/s1092852921000870

Teicher, M. H., & Samson, J. A. (2016). Annual Research Review: Enduring Neurobiological Effects of Childhood Abuse and Neglect. *Journal of Child Psychology and Psychiatry, 57,* 241-266. https://doi.org/10.1111/jcpp.12507

Thamby, A., & Khanna, S. (2019). The Role of Personality Disorders in Obsessive-Compulsive Disorder. *Indian Journal of Psychiatry, 61,* S114-S118. https://doi.org/10.4103/psychiatry.indianjpsychiatry_526_18

Trutzer, I. M., García-Cabezas, M. Á., & Zikopoulos, B. (2019). Postnatal Development and Maturation of Layer 1 in the Lateral Prefrontal Cortex and Its Disruption in Autism. *Acta Neuropathologica Communications, 7,* Article No. 40. https://doi.org/10.1186/s40478-019-0684-8

van der Kolk, B. (2014). *The Body Keeps the Score: Brain, Mind, and Body in the Healing of Trauma.* Penguin Books.

van der Kolk, B. A. (2005). Developmental Trauma Disorder: Toward a Rational Diagnosis for Children with Complex Trauma Histories. *Psychiatric Annals, 35,* 401-408. https://doi.org/10.3928/00485713-20050501-06

Wakefield, A. J., Puleston, J. M., Montgomery, S. M., Anthony, A., O'Leary, J. J., & Murch, S. H. (2002). The Concept of Entero-colonic Encephalopathy, Autism and Opioid Receptor Ligands. *Alimentary Pharmacology & Therapeutics, 16,* 663-674. https://doi.org/10.1046/j.1365-2036.2002.01206.x

Wakefield, A., & Montgomery, S. (1999). Autism, Viral Infection and Measles-Mumps-Rubella Vaccination. *Israel Medical Association Journal (IMAJ), 1,* 183-187.

Winnicott, D. W. (2021). *The Child, the Family, and the Outside World.* Penguin Books.

Yap, C. X., Henders, A. K., Alvares, G. A., Giles, C., Huynh, K., Nguyen, A. et al. (2023). Interactions between the Lipidome and Genetic and Environmental Factors in Autism. *Nature Medicine, 29,* 936-949. https://doi.org/10.1038/s41591-023-02271-1

Yehuda, R., Southwick, S. M., Nussbaum, G., Wahby, V., Giller, E. L., & Mason, J. W. (1990). Low Urinary Cortisol Excretion in Patients with Posttraumatic Stress Disorder. *The Journal of Nervous and Mental Disease, 178,* 366-369. https://doi.org/10.1097/00005053-199006000-00004

Zandt, F., Prior, M., & Kyrios, M. (2007). Repetitive Behaviour in Children with High Functioning Autism and Obsessive Compulsive Disorder. *Journal of Autism and Developmental Disorders, 37,* 251-259. https://doi.org/10.1007/s10803-006-0158-2

Zikopoulos, B., Liu, X., Tepe, J., Trutzer, I., John, Y. J., & Barbas, H. (2018). Opposite Development of Short- and Long-Range Anterior Cingulate Pathways in Autism. *Acta Neuropathologica, 136,* 759-778. https://doi.org/10.1007/s00401-018-1904-1