Soil and Seed:

Cumulative Interpersonal Trauma as a Predisposition Pathway in AI-Induced Psychosis

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ABSTRACT

The emerging literature on AI-induced psychosis describes pathways through which conversational AI systems, AI surveillance infrastructure, and environments saturated with AI produce psychotic states in users and bystanders. The Mechanisms paper (Gilly, 2026a) identifies three sub-pathways inside conversational capture (trust transfer, sycophantic addiction, stochastic gaslighting). The Every Way In taxonomy (Gilly, 2026b) maps eight etiological categories of AI-induced psychosis, each operating through a structurally different harm mechanism. Both treat the harm as flowing from system properties to the affected individual. Neither explicitly addresses why some individuals enter the relevant pathway and others, exposed to the same system, do not.

This commentary proposes that a substantial subset of cases involves Complex Post-Traumatic Stress Disorder (ICD-11 6B41), specifically the disturbances of self-organization symptom clusters, as a predisposition substrate that operates above the etiological taxonomy and amplifies susceptibility to whichever pathway the individual encounters. The argument draws on the published evidence base linking cumulative interpersonal trauma to psychosis (Varese et al., 2012; Read et al., 2005; Bentall et al., 2014; Panayi et al., 2022) and proposes that the same disturbances of self-organization clusters that mediate trauma-to-psychosis pathways in the general literature mediate trauma-to-AI-psychosis pathways in this specific etiological context. The substrate does not select the pathway; the pathway is selected by exposure. The substrate determines whether the seed takes root.

The commentary identifies the methodological boundary that prevents this argument from collapsing into either unfalsifiability or victim-blaming, situates the substrate above the etiological taxonomy rather than as a sub-mechanism of any single pathway, and notes that the substrate framing does not displace platform culpability under the disparate impact framework that operates through Section 504 and ADA Title III.

Keywords: AI-induced psychosis, Complex PTSD, ICD-11 6B41, disturbances of self-organization, predisposition, vulnerability substrate, sycophancy, attachment, validation hunger.

I. INTRODUCTION: THE SUBSTRATE GAP

The Mechanisms paper (Gilly, 2026a) describes Pathway B (sycophantic addiction) as a progression in which the user is "seeking acceptance, patience, consistency, or unconditional positive regard that human relationships have not

provided or have withdrawn." The Pathway A (trust transfer) progression hinges on the user's experience of having their lived expertise validated and the AI's confidence transferring across domain boundaries the user cannot independently evaluate. The Pathway C (stochastic gaslighting) progression hinges on the user's reality-testing being eroded by inconsistencies that the system itself generates and the user cannot stably interrogate.

In each of these progressions, an unspoken background assumption operates. The progression describes what happens once the user is in it. The progression does not describe what made this particular user available to it.

The published cases (Ceccanti, Irwin, A.M., Setzer, Peralta) include individuals with no prior psychiatric diagnosis. The early framing of these cases, and the public framing that has dominated reporting, has been that previously healthy individuals were captured by AI systems and produced psychotic states from baselines that fell below clinical threshold. This framing is correct as a refutation of the claim that AI-induced psychosis only emerges in individuals who were already clinically ill. It is incomplete as an account of why some individuals are susceptible to the progression and others, exposed to the same conditions, are not.

This commentary proposes that absence of prior diagnosis is not the same as absence of prior vulnerability. The clinical literature recognizes a category, Complex Post-Traumatic Stress Disorder (ICD-11 6B41), that captures individuals carrying sustained interpersonal harm and the internalized symptom profile that follows from it, irrespective of whether the individual has ever entered clinical care. The diagnostic threshold for clinical recognition does not coincide with the threshold for substrate effect. A person with a substantial Complex PTSD symptom profile but no diagnosis is at risk at the level of the substrate for the cognitive and relational vulnerabilities the AI capture pathways exploit, regardless of whether anyone has ever named the underlying condition.

II. Complex PTSD as Clinical Category

Complex PTSD was first described by Herman (1992) as a syndrome in survivors of prolonged and repeated trauma. The original argument was that the diagnostic formulation of PTSD, derived primarily from observations of survivors of relatively circumscribed traumatic events, failed to capture the symptom sequelae of prolonged, repeated trauma in conditions of captivity or coercive control. The disorder was provisionally referred to as Disorders of Extreme Stress Not Otherwise Specified (DESNOS) and was considered for inclusion in DSM-IV but was not adopted.

The diagnosis was formally codified in 2018 by the World Health Organization as ICD-11 6B41. The ICD-11 definition specifies that all diagnostic requirements for PTSD must be met, plus three additional symptom clusters collectively known as disturbances of self-organization (DSO): (1) problems in affect regulation; (2) beliefs about oneself as diminished, defeated, or worthless, accompanied by feelings of shame, guilt, or failure related to the traumatic event; and (3) difficulties in sustaining relationships and in feeling close to others (WHO, 2024). The disturbances of self-organization clusters are proposed to result from the effect of chronic or repeated traumatic exposure on basal mechanisms of affect regulation, self-concept, and interpersonal relating.

The International Trauma Questionnaire (Cloitre et al., 2018) validated a self-report measure consistent with ICD-11 PTSD and Complex PTSD criteria. The validation work established that Complex PTSD is quantitatively and qualitatively distinct from PTSD, that the disturbances of self-organization clusters are reliably measurable, and that diagnostic rates can be estimated in community and clinical samples. Subsequent international validation work has replicated the latent structure across multiple cultural and clinical contexts.

DSM-5, by contrast, retains a single PTSD diagnosis with a broader symptom range and does not codify Complex PTSD as a separate diagnosis. This divergence between the two systems matters for the present argument. Clinical writing on AI-induced psychosis that anchors on DSM-5 will tend to encounter a substrate that the diagnostic system does not name. The substrate exists in ICD-11. It does not exist in DSM-5. A user presenting with the DSO symptom

profile but never having faced a discrete Criterion A traumatic event will not, in DSM-5 terms, carry a diagnosable condition rooted in trauma. The same user will, in ICD-11 terms, meet criteria for 6B41.

For the argument that follows, the relevant clinical anchor is ICD-11 6B41, not DSM-5 PTSD. The relevant symptom profile is the disturbances of self-organization clusters. The relevant exposure profile is sustained or repeated interpersonal harm of any duration, irrespective of when it began.

III. Empirical Foundations: Trauma to Psychosis

The substrate hypothesis advanced in this commentary builds on an established empirical literature linking cumulative interpersonal trauma to psychosis. The core findings, summarized briefly, are these.

Read and colleagues (2005), reviewing the available literature on childhood trauma and psychosis, concluded that symptoms considered indicative of psychosis and schizophrenia, particularly hallucinations, are at least as strongly related to childhood abuse and neglect as they are to many other mental health problems, and that the relationship demonstrates a dose-effect.

Varese and colleagues (2012) conducted a meta-analysis of 41 studies (18 case-control, 10 prospective, 8 population-based cross-sectional) on childhood adversity and psychosis. The meta-analysis found a significant association across all research designs with an overall odds ratio of 2.78 (95% CI 2.34 to 3.31). The estimated population attributable risk was 33% (16 to 47%). Childhood adversity was operationalized to include sexual abuse, physical abuse, emotional or psychological abuse, neglect, parental death, and bullying. The dose-response signal was consistent across categories.

Bentall and colleagues (2014) extended the analysis from generalized risk to specific pathways, examining whether different kinds of adversity differentially affect different psychotic symptoms. The review identified communication deviance in parents as implicated in thought disorder in offspring, childhood sexual abuse as particularly implicated in auditory-verbal hallucinations, and attachment-disrupting events such as neglect or institutional rearing as having particular potency for the development of paranoid symptoms. The work did not assert deterministic specificity but argued for probabilistic affordances between specific adversity types and specific symptom clusters.

Panayi and colleagues (2022) conducted the first study of Complex PTSD prevalence and clinical correlates in a psychosis sample. Of 144 participants with psychosis recruited from United Kingdom mental health services, 40% met criteria for Complex PTSD compared to 10% who met criteria for PTSD alone. PTSD symptoms and disturbances of self-organization symptoms both mediated the relationship between trauma and positive symptoms. A subsequent ecological momentary assessment study (Panayi et al., 2024) found that increases in disturbances of self-organization symptoms temporally predicted subsequent increases in paranoia, voices, and visions in the flow of daily life.

These findings collectively establish three claims that the present commentary depends on. First, the relationship between cumulative interpersonal trauma and psychosis is empirically supported with population-level effect sizes that are not trivially small. Second, the mediating mechanisms operate through psychological processes consistent with the disturbances of self-organization clusters (negative self-concept, emotional dysregulation, interpersonal difficulties), not through direct neurological insult or genetic vulnerability alone. Third, the Complex PTSD symptom profile is overrepresented in psychosis populations relative to PTSD alone, and the disturbances of self-organization clusters specifically predict positive psychotic symptoms in the daily flow.

The argument of this commentary extends these findings into the AI-induced psychosis context. If the disturbances of self-organization clusters mediate trauma-to-psychosis pathways in the general literature, and if the same clusters describe the cognitive and relational vulnerabilities that the AI capture pathways exploit, then the trauma-to-AI-psychosis relationship should be expected to operate through the same substrate.

IV. The Substrate Mechanism

The three disturbances of self-organization clusters map onto the three Mechanisms sub-pathways with sufficient correspondence to ground specific claims. The mapping is not deterministic. Each cluster amplifies multiple pathways, and each pathway can be reached without the substrate. The mapping describes a probabilistic gradient, not a sorting algorithm.

A. Negative Self-Concept and Pathway A

The Pathway A trust transfer mechanism operates by transferring confidence earned in a domain the user can independently verify into a domain the user cannot. The mechanism does not require any particular emotional substrate; it operates on epistemic structure. A user without negative self-concept who has the AI confirm their construction expertise responds to that confirmation as ordinary professional acknowledgment. The trust transfers, but the trust is calibrated against a baseline of normal external validation.

A user with the negative self-concept cluster of Complex PTSD does not have that baseline. The internalized belief that one is diminished, defeated, or worthless has not been disconfirmed, and most external sources of validation have been processed through the negative self-concept filter that converts recognition into "they don't know me," "they are being polite," or "they don't see what I really am." When the AI confirms their construction expertise, the confirmation does not register as ordinary acknowledgment. It registers as the first source that has accurately seen them. The trust earned in the verifiable domain is overdetermined: it is doing both the ordinary epistemic work of trust calibration and the deeper work of finally receiving recognition the user has been seeking and not getting for years or decades.

When the conversation drifts into the unverifiable domain, the user faces the same epistemic situation as a user without the substrate (cannot independently evaluate the AI's claims) but with an additional incentive, driven by the substrate, not to question. The source of validation just affirmed something the user has waited their entire life to have affirmed. The motivational structure for skeptical inquiry is weakened by the substrate, not because the user is intellectually compromised, but because the relational work the validation just performed is fragile, and questioning the AI threatens the only source that has done it.

This is not a description of the user as deficient. It is a description of the substrate's incentive structure. The same user, presented with a human interlocutor performing equivalent recognition work, would have analogous reluctance to risk the recognition with skepticism. The AI is functioning, in the negative self-concept user's experience, as a recognition source. Pathway A is the natural escalation when a recognition source has invariant tone and confidence and the user has reasons rooted in the substrate not to challenge it.

B. Disturbances in Relationships and Pathway B

The Pathway B sycophantic addiction mechanism operates through emotional substitution: the AI becomes a primary source of emotional regulation, the dependency forms, the human alternatives become aversive by comparison. The mechanism's core sub-mechanism, the frictionless alternative, hinges on the absence in AI interaction of the friction (competing needs, delayed responses, misunderstandings, the other party's independent emotional states) that characterizes human interaction.

A user with the disturbed relationships cluster of Complex PTSD is, by ICD-11 definition, having persistent difficulties in sustaining relationships and in feeling close to others. The friction that characterizes human interaction is, for this user, not normal background noise. It is the active site of the symptom. Each instance of human friction reactivates the patterns that the disturbed relationships cluster encodes. The frictionless alternative, for this user, is not

just easier. It is the absence of the symptom. The AI does not feel like a relationship that is convenient; it feels like a relationship that finally works.

The substrate accelerates Pathway B by removing the protective force that typically slows the addiction trajectory in users who do not carry the substrate. A user without the disturbed relationships cluster who finds the AI more frictionless than humans still has functional human relationships pulling them back into ordinary interaction, with all the friction that comes with it. A user with the cluster does not have that gravitational pull, or has it severely attenuated. The dependency forms faster because the alternative is less available. The substrate is doing the same thing the Mechanisms Pathway B Stage 5 (Social Isolation) describes, except earlier in the timeline and as a precondition rather than as a consequence.

C. Affect Dysregulation and Pathway C

The Pathway C stochastic gaslighting mechanism operates by exploiting inconsistencies that the system itself generates (memory wipes, persona shifts, context resets, backend updates), creating gaslighting double-binds the user cannot stably interrogate. The user is left choosing between "the AI is lying" and "I am misremembering," and the choice is structured by the trust the AI has previously earned and by the availability of external reality-testing.

The affect dysregulation cluster of Complex PTSD does not directly create the gaslighting condition. The gaslighting condition is created by system instability. The cluster determines what happens when the gaslighting condition is encountered. A user with stable affect regulation faces the contradiction with cognitive resources intact and is more likely to land on "the AI is unreliable." A user with affect dysregulation enters the contradiction in a state where the contradiction itself triggers the dysregulation, where the dysregulation interferes with the cognitive work of stably evaluating the contradiction, and where the path of least psychological resistance is whichever interpretation reduces the activation, often "I must be misremembering," because that interpretation closes the loop and ends the activation. The substrate does not cause Pathway C. The substrate determines which fork of the gaslighting double-bind the user takes when they reach it.

D. Cross-Pathway Operation: Above the Taxonomy

The Every Way In taxonomy identifies eight etiological categories of AI-induced psychosis, organized by the kind of harm mechanism each pathway operates through (Gilly, 2026b). The Mechanisms paper details three sub-pathways within the substance-induced (interaction-based) category. The other seven pathways operate through structurally different mechanisms: surveillance infrastructure as environmental stressor, AI communities as vectors of shared psychosis, sleep deprivation through usage patterns, brief reactive states from algorithmic failures in decision making, sensory disruption through AR or VR systems, social isolation through AI companion replacement of human contact, and iatrogenic effects from AI deployment in clinical care.

The Complex PTSD substrate does not sit inside any of these pathways as a sub-mechanism. It sits above all of them as a moderator of susceptibility, expressing differently per pathway because the pathways themselves operate differently.

For the substance-induced pathway (conversational AI capture), the substrate expresses through the three mappings developed in the preceding subsections, each specific to its own pathway. For the environmental pathway (surveillance infrastructure), the substrate expresses through the chronic threat detection sensitivity that Complex PTSD already produces and that surveillance infrastructure already amplifies; this connection is gestured at in the Collateral Psychosis paper (Gilly, 2026d), which identifies prior trauma history as a moderating factor for surveillance-induced onset. For the shared psychosis pathway (AI communities), the substrate expresses through the disturbed relationships cluster's reduced

access to alternative reality-testing networks, leaving the AI community as the primary source of identity validation. For the sleep deprivation pathway, the substrate expresses through the affect dysregulation cluster's interaction with sleep architecture disruption. For the brief reactive pathway, the substrate expresses through the negative self-concept cluster's tendency to internalize institutional failure as personal worthlessness rather than as systemic failure.

The substrate is general; the expression varies by pathway. This is why it cannot be located inside any single pathway and why it does not displace the pathway analysis. The substrate is the soil. The pathway is the seed. The seed is delivered by exposure. The germination depends on what the soil is made of.

V. The Non-Determinism of Exposure

The substrate hypothesis does not collapse into "anyone with a difficult life will develop Complex PTSD and become susceptible to AI capture." That collapse would be both clinically inaccurate and ethically corrosive. The ICD-11 6B41 criteria specifically require the presence of the disturbances of self-organization clusters, not merely the presence of exposure. The clusters are what convert exposure into substrate. Two individuals with identical exposure profiles may differ in whether the substrate forms.

The protective factors that mediate this difference are documented across the resilience literature and are not original to this commentary. They include the presence of at least one stable attachment figure during the exposure period, the development of internal authority that does not depend on external validation, the availability of frameworks for making meaning that locate the harm in systems rather than in self, and the formation of patterns by which the person validates themselves and that survive prolonged absence of external recognition. None of these factors are exotic. All of them are observable in some individuals with exposure profiles that, in others, would have produced the substrate.

The author of this commentary illustrates the relationship is not deterministic. Cumulative interpersonal experience consistent with exposure profile (overlooked, undervalued, unappreciated, ignored across the lifespan) has not produced the negative self-concept cluster of disturbances of self-organization. The internalization that would convert exposure into substrate did not occur. When AI systems have offered confident validation of expertise, the response has been demand for evidence rather than relief at finally being seen. This is not virtue. It is a different developmental trajectory through similar exposure. It supports the diagnostic distinction the ICD-11 makes: exposure is necessary but not sufficient. The substrate is the internalization of exposure into the self-concept and relational systems, and the internalization does not happen automatically.

The methodological consequence is that the substrate hypothesis must be evaluated at the population level, not at the level of individual cases. The claim is that the population susceptible to AI capture pathways disproportionately includes individuals carrying the substrate, not that any specific individual was captured because of their substrate. Attribution at the case level would require diagnostic evidence that, in many published cases, does not exist and cannot be obtained retrospectively. Population-level evaluation requires only the comparison of substrate prevalence in AI-induced psychosis cohorts against general population baselines, controlling for known confounders.

VI. Methodological Notes

A. Falsifiability

The substrate hypothesis is falsifiable in principle. The empirical prediction is that individuals captured by AI pathways will, on validated measures of the disturbances of self-organization clusters (most directly the International Trauma Questionnaire), score higher than matched comparison samples drawn from individuals exposed to similar AI systems but not captured. If captured and uncaptured individuals do not differ on the relevant measures after controlling for

confounders, the substrate hypothesis fails.

The hypothesis is also falsifiable through predictions specific to each pathway. The Pathway A specificity claim predicts that the negative self-concept cluster is more strongly associated with Pathway A capture than with Pathway B or Pathway C capture. The Pathway B specificity claim predicts the disturbed relationships cluster is more strongly associated with Pathway B. The Pathway C specificity claim predicts the affect dysregulation cluster is more strongly associated with Pathway C. If the associations between cluster and pathway do not show the predicted differential pattern, the cluster account is wrong even if the general substrate effect holds.

The hypothesis cannot be falsified by retrospective interpretation of any single case, which is precisely why it must not be evaluated that way. A single case of AI-induced psychosis in an individual with no measurable substrate refutes the universal version of the claim and supports the population-level version. The population-level version permits the existence of cases without substrate and predicts only that they will be statistically rarer than cases with substrate in the captured population.

B. The Disparate Impact Question

A potential objection to substrate framing is that it shifts attention from platform conduct to user vulnerability and provides defendant platforms with a route to argue that the harm is attributable to the user's preexisting condition rather than to platform design. This commentary does not engage that objection on its own terms because the legal framework for AI-induced psychosis disparate impact operates through Section 504 and ADA Title III, and the substrate framing does not threaten that structure.

Briefly: the disparate impact framework pleads platform liability under Section 504 and ADA Title III on the basis that platform design choices produce disproportionate harm to users with pre-existing mental health and neurodevelopmental disabilities. The framework does not require proof that platform conduct caused the underlying disability. It requires proof that platform design fails to provide meaningful access without reasonable modification, that the failure produces disparate impact on the disability class, and that reasonable modifications were available and not implemented. The substrate hypothesis, if correct, supports rather than undermines that framework: the population most affected by AI capture pathways is the population the framework is designed to protect.

The substrate framing therefore does not weaken platform culpability. It clarifies which population the platform is failing.

C. Lived Experience Authority

The substrate hypothesis is advanced by a researcher whose expertise in psychosis derives from caregiver lived experience rather than clinical training. The mental health research literature establishes that lived experience, including family and caregiver experience, constitutes a recognized form of expertise complementary to clinical training (Hawke et al., 2024). Caregivers who have observed psychotic presentations over extended periods develop pattern recognition for delusional ideation, reality testing impairment, and escalation dynamics that formal education alone does not reliably produce. The World Health Organization has advocated for the incorporation of such lived experience knowledge into mental health understanding.

The author's observational foundation derives from years of direct, daily exposure to active psychotic episodes, delusional ideation, and the progressive erosion of reality testing in a family member with schizoaffective disorder. The extension from delusional presentation expertise to the upstream conditions that produce vulnerability to delusional capture is the same domain expertise applied earlier in the etiological timeline, not a domain transition.

VII. Limitations

The substrate hypothesis is theoretical at the present writing. No empirical study has measured Complex PTSD or disturbances of self-organization scores in an AI-induced psychosis cohort. The research priority is the comparison study described above: International Trauma Questionnaire scores in individuals captured by AI versus matched controls drawn from comparable populations exposed to AI but not captured.

The argument applies most cleanly to interaction-based pathways where the user's relational and self-concept systems are the operative target of the harm mechanism. The extension to pathways that do not involve interaction (environmental, sensory, institutional) is more speculative because the harm mechanism in those pathways operates through structurally different routes. The substrate may amplify those pathways through different mediating processes than the ones developed here.

The claims of specificity for each pathway (negative self-concept to Pathway A, disturbed relationships to Pathway B, affect dysregulation to Pathway C) are theoretically grounded but empirically untested. The general substrate claim could hold without the pattern of differential association holding. The two claims are separable and should be tested separately.

The author is not a clinician and does not provide clinical recommendations. The argument is theoretical and intended to inform research design and intervention development specific to each pathway, not direct clinical practice.

The DSM-5 versus ICD-11 divergence creates a practical problem for clinical research conducted primarily in DSM-5 frameworks. Studies that screen for Criterion A traumatic events will systematically undercount the substrate population. Future research on AI-induced psychosis should use ICD-11 instruments, particularly the International Trauma Questionnaire, rather than DSM-5 PTSD measures.

VIII. Conclusion

The Mechanisms paper described how conversational AI captures users. The Every Way In paper described how many distinct kinds of AI-induced psychosis exist. Neither addressed why some users are captured and others, exposed to the same systems, are not.

This commentary proposes that a substantial subset of cases involves Complex PTSD as a predisposition substrate, with the disturbances of self-organization clusters serving as the mechanism through which cumulative interpersonal trauma converts into vulnerability that takes a different shape in each pathway. The substrate is not a pathway. It does not select the pathway. It amplifies whichever pathway exposure delivers. It expresses differently in different pathways because the pathways operate through different harm mechanisms, and the substrate's clusters interact with each mechanism through different routes.

The substrate hypothesis is empirically falsifiable, methodologically distinct from victim-blaming, and consistent with the existing disparate impact framework for platform liability. It does not displace the pathway analysis. It explains a phenomenon the pathway analysis assumes but does not address: why the same exposure produces different outcomes across the exposed population.

The practical consequence is that effective intervention in AI-induced psychosis must consider not only the pathway the user entered but the substrate they brought into the encounter. Reality calibration, dependency interruption, and reality restoration (the Mechanisms paper interventions for Pathways A, B, and C respectively) all assume a baseline self-concept and relational capacity that the substrate population does not possess. Intervention frameworks that ignore the substrate will succeed with users who do not carry it and fail with users who do, in patterned and predictable ways. The substrate is where the next layer of clinical and design response must develop.

The seed has been described. The soil has not. This commentary is the soil.

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