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SPECIAL ARTICLE

Modelling disruptions of intentionality in psychosis

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ABSTRACT

In philosophy, *intentionality* involves directedness, aboutness, or reference of mental states. It seems to have intense connections with mental representation, consciousness, as well as evolutionary selected functions. *Naturalizing intentionality*, in terms of tracking or functional roles, is one of the most important goals in philosophy of mind. Such *what-matters models* would be useful, employing a combination of the principles of intentionality and causality. For example, the brain contains a *seeking system* that is responsible for its capacity of having an *instinct-like* urge towards something or towards wanting. *Reward circuits* are linked with emotional learning, reward seeking, reward learning, as well as with the homeostatic system and the hedonic system. We may suggest that such brain systems reflect components of a broad *intentional system*, whereas non-linear dynamics can explain the complex behavior of such chaotic or fuzzy systems. Historically, the *cusp catastrophe model* has been used to predict health behaviors. It can explain why relatively small changes in a parameter can result in catastrophic changes in the state of a system. If distal risk is low, then proximal risk will be linearly related to psychopathology. If distal risk is high, then proximal risk is nonlinearly related to a severe psychopathology and small changes in proximal risk predict a sudden lapse. The phase of *hysteresis* can explain how a network stays active long after the events in the external field that triggered its activation have waned. It seems that in psychotic patients there is a failure of intentionality, due to the inappropriateness of an *intentional object* or connection, or due to the absence of an intentional object altogether. In psychosis, these failures seem to occur through a non-linear and multifactor fluctuating pattern of intentionality. The ultimate goal is to provide a better understanding of relapse. The sudden collapse can be explained by an already fragile *intentional system* rather than by a novel stressor. The *catastrophe model* may help individuals remove themselves from a *hysteresis cycle*, and strategies for sustainable management of such cases should focus on maintaining resilience. Focusing on *disruptions of intentionality* can deepen and enrich our understanding of radical disturbances involved in different psychopathologies, including psychosis.

KEYWORDS: Intentionality, psychosis, aboutness, directedness, cusp catastrophe model, non-linear modelling.

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Intentionality

The term *intentionality* is derived from the Latin verb *intendo* ("aim," "hold out," or "stretch"), and refers to the way consciousness can be *about* things. It is the feature of the mind through which mental states are *directed at*, or are *about* or *of*, or *refer to*, states of affairs in the world. Intentionality plays a central role in subjective experience and permeates all human activities. Dennett¹ proposes *intentional stance* as so powerful that it can be developed into a valid *intentional theory*. For Brentano² all psychological phenomena and only them are intentional. The crucial concept in Husserl's³ theory of intentionality is the *noema* of consciousness, while for Heidegger,⁴ intentionality is a feature of *Dasein's* entire way of being-in-the-world. Merleau-Ponty⁵ extended to *motor intentionality*, the intentional constitution of the body and its role in perceptual experience.

Intentionality seems to have essential connections with both consciousness and evolutionary selected functions.⁸ Searle^{6,7} extended to *collective intentionality*, a biologically primitive phenomenon that we humans share with other social animals, adding that without a *pre-intentional sense of community* there would not have been collective intentionality and social reality. Our ability to participate with others in collaborative activities with common goals is called *shared intentionality*. Kesebir⁹ used the *superorganism* (such as ants or bees) metaphor to reflect on human sociality. According to her, cultural meaning systems, shared intentionality, norm compliance, deference to authority, social identity processes, religiosity, and morality, can be understood as manifestations of the same dynamics that create *superorganism-like* social structures. For Watsuji,¹⁰ intentionality is a relational property of the whole person engaging with the things, spaces, and other people in a common world. The quality of our intentional acts is strongly regulated by *betweenness*. For example, in the *betweenness* of *seeing* another person, one's activity of seeing, is a seeing determined by its being seen by the other. The characterization of the social character of intentionality is useful for illuminating subtle experiential anomalies typical of schizophrenia.^{11,12}

Intentionality guides all voluntary thought and behavior, and is also implicated in every meaning and value.¹³ Bolton^{14,15} considered whether mental disorders might be specified by a class of "radical failures" of intentionality. He suggested that the mind is in good working order to the extent that its intentional objects and connections are appropriate. Any failure of intentionality, because of inappropriateness of an intentional object or connection, or absence of an intentional object altogether, may result in a mental disorder.

Naturalizing intentionality

Naturalizing intentionality is of the most important items in philosophy of mind. Spinoza¹⁶ asserts that every individual thing strives to persevere its existence, adding that when this striving (*conatus*) is related only to the mind, it is called *will* and when it is related to the mind and body simultaneously, it is called *appetite*. The concept of the *conatus* was first developed by the Stoics who used the word *ὁρμή* (*hormê*, translated in Latin by *impetus*) to describe the movement of the soul towards an object, and from which a physical act results. Nowadays, the archaic concept of *conatus* is being reconciled with modern biology and neuroscience and is explained in terms of chemistry and neurology,¹⁷ whereas the Spinozistic conception of a *conatus* is a historical precursor to modern theories of *autopoiesis*.¹⁸ Conscious *desire* presupposes a kind of *primordial orectic* orientedness of the organism, whereas *orectic* states can finally give rise to conscious *conations* or *desires*.¹⁹ *Orexis* is the Aristotelian term for *appetite*, sometimes signifying appetite in general and at other times the power of the will. Additionally, *instinct* contains *impulse* or *appetite*, for realizing what is targeted by the *conative* element. Conation, urge and drive are terms that are used almost interchangeably

to indicate the forceful or impulsive aspect of appetites, while feeling and affectivity are generally used to indicate the felt quality connected with appetitive activity.²⁰

However, is there a common ground, conceptual or physiological, for both intentionality and conatus? Is there an *intentional system* running in parallel with specific brain networks and functions, linked to will, appetite or reward? Moreover, is that intentional system a higher order system, having a monitoring or regulatory role on brain or behaviour?

For Millikan,^{21,22} intentionality has been related to purposiveness and such purposes help us to understand intentionality in a naturalistic way. She explains *intentionality* using the explanatory resources of natural selection: what thoughts and sentences and desires are 'about' is ultimately elucidated by reference to *what* has been selected and what it has been selected *for*, i.e., what advantage it conferred on ancestors who possessed it. For biologists, intentionality of all sorts is ultimately the result of evolution via natural selection. Fitch²³ suggested *intrinsic intentionality* as a thoroughly materialistic and derived through evolution, pointing out that the ion flux in the human nervous system has a form of intrinsic intentionality, a type of "aboutness". Moreover, he proposed *nano-intentionality*, a microscopic form of *aboutness*, inherent in individual eukaryotic cells, that includes a goal-directed capacity to respond in an adaptive manner to novel circumstances. The *nano-intentional* ability of cells to rearrange their structure in response to their circumstances represents a basic, primitive type of *goal-directed aboutness* that predated neurons, brains and minds. Fitch²³ noticed that "without nano-intentionality intentionality proper can never emerge; without such a capacity, all of the information processing in the world will not make a *system intentional*".

The brain contains an exploration *seeking system* that is responsible for having an instinct-like behavior *towards* something or *towards wanting* it.²⁴ In parallel with its homeostatic function, eating can also be a pleasurable experience, which is related to the brain's core *reward circuits*, implicated also in drug use and sexuality. Reward circuits include the dopaminergic ventral tegmental area, signaling motivation and reward seeking, the amygdala, associated with emotional learning; the nucleus accumbens, involved in reward learning, and the lateral hypothalamus, which integrates motivation signals and links homeostatic and hedonic system.²⁵

Appetite and feeding are controlled by the *homeostatic system*, which ensures that a person gets enough calories to survive, and the *hedonic system*, which regulates the pleasure and reward aspects of eating.²⁵ *Orexigenic* and *anorexigenic neurons* (from Latin *orexis* [appetite] and Greek *órexis* [desire]), stimulate and suppress food-seeking behaviors, respectively, are located in the *arcuate nucleus* of the hypothalamus, and project to the paraventricular nucleus, promoting catabolism, to the ventromedial hypothalamus, suppressing feeding behavior, and to the lateral hypothalamus, promoting calorically dense food and locomotor activity, through melanin-concentrating hormone and orexin.²⁵

The Lateral Hypothalamic Area (LHA) plays a role in arousal, feeding, motivation, and reward. LHA lesions reduce food intake and cause weight loss, while electrical stimulation of the LHA increases feeding.²⁶ In addition, the hypothalamic agouti-related peptide (AgRP) neurons control neuronal pathways that regulate higher-order brain functions during development and in adulthood.²⁷ Finally, the comorbidity between diabetes and psychotic disorders is well-known, but not fully understood. There is some, but limited and inconsistent, evidence that non-CNS alterations are associated with CNS changes and symptoms in first episode psychosis,²⁸ whereas a high prevalence of impaired fasting glucose and metabolic syndrome in patients with psychosis.²⁹

Linear and non-linear modelling of psychopathology

Non-linear dynamics describe the complex behavior of chaotic or fuzzy systems. Looking at history through mathematics, Rashevsky³⁰ developed a theory of *bifurcation*, which he applied to the complexity and divergence of human behavior. He stated: “A change in the behavior of a single individual may precipitate in an unstable social configuration, a process that leads to a finite, sometimes radical change”. *Catastrophe theory*, as described by Thom³¹ in 1972, is derived from topology, a field of mathematics that studies the properties of surfaces in numerous dimensions. *Catastrophe theory* has been applied to situations where gradual changes in the environment correspond to abrupt changes in the expressed behavior. The type of *cuspl catastrophe model* describes a sudden behavioral change observed once a predictor variable crosses the *cuspl* threshold. The slightest disruption can precipitate a fall from which there is no return.

The *cuspl catastrophe model* contains the main qualities of *bimodality*, and the behavioral outcome is partitioned into three modes, (a) *divergence*, where small changes in the input parameters leads to dramatic changes in behavior, (b), *quick transitions or jumps*, where the transition from one mode of behavior to the other may happen rapidly and (c) *hysteresis*, where the transition from one mode to the other mode does not occur at the same place on the surface.³² For those with high distal risk, for example, the suicidal ideation pathways follow path B (see [Figure 1](#)), where even a small increase in proximal risk may push an individual “over the edge,” leading to suicidal episode.³³ After that, a *prolapse* requires a substantially larger reduction in proximal risk to help the individual regain a healthier level.³⁴ When a certain threshold level of stress is reached, a “catastrophic” transition from low to high risk occurs, while the system is slowly getting less and less resilient. The *sudden jump* can be better explained by an already fragile system (distal factors) rather than a novel stressor (proximal factor).³⁵

In order to get back from a high to a low-risk phase, it is not sufficient to restore stress levels to the level prior to the collapse. This dependence of the current state of the system on the previous state is called *hysteresis*, a term derived from the Greek word ὑστέρησις, meaning “deficiency” or “lagging behind”, and coined in 1881 by Sir James Alfred Ewing, after showing that magnetization of a sample will remain magnetically polarized even when the external field is removed. *Hysteresis* explains how a network can stay active long after the events in the external field that triggered its activation have waned. *Hysteretic systems* are regarded to have “memory” of previous events or a dependence on its history. The dynamic character of the voltage dependence ion channels seems to be rooted in its *hysteretic behavior* and has important consequences on the physiology and pharmacology. For example, hysteresis in ion channels behavior can make the deactivation of K_V-related conductance more resilient to closing at resting and to developing hyperpolarized potentials during repolarization.³⁶

Moreover, mental disorders may arise due to the presence of hysteresis in strongly connected symptom networks, which implies that symptoms continue to activate one another, even after the triggering cause of the disorder has disappeared. According to Borsboom,³⁷ “*hysteresis* is a very plausible feature of psychopathology networks, because – in many cases of psychopathology – triggering events can cause pervasive problems long after triggers themselves have disappeared”. Important examples would be the etiology of post-traumatic stress disorder, the development of major depression, or the effects of childhood abuse.

Historically, the *cuspl catastrophe model* has been used to predict health behaviors.³⁸ The ultimate goal is to provide a better understanding of relapse and this model can help individuals remove themselves from a *hysteresis cycle*. Furthermore, *critically slowing down* indicators can help predict attacks in chronic diseases such as asthma, heart arrhythmias,

migraine, epilepsy, depression and suicidality. When a system approaches a critical transition, it returns more slowly to its stable attractor under small perturbations. The return time to the stable state can thus be used as an index, that shows whether a critical change is about to happen or not. On the other hand, when a patient suddenly becomes disordered, we tend to look for major changes that caused the transition, but for some patients, it might be that the system was slowly getting less and less *resilient*, and that the sudden collapse can be explained by an already fragile system rather than a novel stressor (Table 1, Figure 1).³⁹

Non-linear disruptions of intentionality in psychosis

In the *cusp catastrophe model*, *distal risk factors* are defined as background predisposing factors that create an increased statistical risk of relapse. *Proximal risk factors* actualize the statistical risk, indicating the time of relapse and may include situational threats to self-efficacy, affective states, stressful life events, emotional traumas, or the rapid deterioration of social support. The increasing level of distal risk will create a *bifurcation*, whereby the potential for relapse is greatly increased if the level of proximal risk is also high. Depression, drug use and suicidality have been studied according to this model, as complex systems where a small change in mood can have a large effect on the situation someone is in.^{33,40,41} The cumulative and proliferation dynamics of *trauma* are more powerful bifurcation control factors in these non-linear dynamics.³³ Focusing on *disruptions of intentionality* can deepen and enrich our understanding of core disturbances involved in different psychopathologies, including psychosis.^{42,43} As an intentional and self-organizing process, the temporal microstructure of consciousness, comprised of both a *retention* and a *protention*, can become fragmented in psychotic patients.^{44,45} Patients suffering from *Capgras* and *Cotard* delusions lose conscious access to normal intentional objects of affective experience, since the patient's affective experience is restricted to a subset, or null-set, of appropriate intentional objects.^{14,15,46}

Delusions represent a cognitive effort by the patient to comprehend aberrantly salient experiences, whereas hallucinations reflect a direct experience of the *aberrant salience* of internal representations.^{47,48,49} In the case of the *Muller-Lyer illusion lines*,⁵⁰ the *intentional content* of the visual experience is in conflict with and is overridden by the intentional content of our beliefs. In the case of hallucinations, the perceiver has the same experience, but no *intentional object* is present. Maybe, the capacity to *misrepresent* is often thought to be essential for representing: no possibility of misrepresentation, no representing.⁵¹ A capacity to misrepresent may require a basic capacity to represent non-existent objects⁵² and in psychosis there is a loss of both the intentional object and the capacity to misrepresent. In psychosis a permanent loss or lack of intentionality dominates, including an absence of intentional object, from the beginning, or even from the primordial phase of the disorder. This might be the reason for their difficulty in recovering from the *inaccessibility region* (see Figure 1).^{42,43,53,54}

In the Sino-Japanese study of *ningen*, the English term for *human being*, the character *nin* signifies two men supporting each other, whereas *gen* implies 'between' or 'among'.¹² We not only engage with the world; the world, in turn, engages with us. Schizophrenia is more than just a brain disorder; it is a self-disorder, a multi-level disturbance of an individual's relationship with their world. Self-disturbances in schizophrenia are thus equally relational disturbances or disturbances of *betweenness*.^{10,11,12} The individual intentionality is derived from collective intentionality, which is a *shared intentionality* that shares mental states like emotions, intentions, and beliefs with others.^{6,7} Two forms of shared intentionality have been proposed: *joint intentionality* and *we-intentionality*. Unlike joint intentionality, *we-intentionality* relies on the agents' capacity to understand themselves as group members and

to adopt the group's perspective. In psychotic patients we-intentionality has been found to be impaired, as opposed to joint intentionality which remains unaffected.⁵⁵

This manuscript focuses on a rather neglected issue, that concerns both aspects of philosophy and neurobiology, relating to the phenomenon of intentionality, and its specific role in psychosis. Methodologically designed studies of intentionality in psychosis have revealed a variety of findings: The subjective experience of psychotic patients with body-affecting first-rank psychotic symptoms is rooted in the disturbance of intentionality and diminished sense of agency.⁵⁶ Moreover, psychotic patients exhibited a striking bias to over attribute intentionality, and especially an inability to inhibit the automatic attribution of intentionality.⁵⁷ In a philosophical analysis of intention, tested through fMRI experiments, a hyper-intentionality state was prominent in patients with paranoid schizophrenia and a hypo-intentionality state in autistic spectrum disorders.⁵⁸ Moreover, it seems that the flow of information evoking spontaneous attributions of intentionality is disrupted in schizophrenia, with flow-on detrimental effects on accurate *theory of mind* reasoning. This finding may indicate that referential and persecutory ideation motivates inappropriate mentalising when objective cues of intentionality are absent.⁵⁹ Research findings indicate that intentionality is a complex phenomenon that requires a complex theory. Therefore, we may think of an *intentional system*, as a higher order system, which has a main regulatory and monitoring role in brain and behavior.⁶⁰

Conclusion

Our intentionality is profoundly originating from genes. In order for them to be able to survive, people composed of these genes have to produce intentional states. Intentional stance is a strategy that allows prediction and explanation of the observed behaviour of a system. It compares predictive strategies, (i.e., physical vs design stance), whereas *theory of mind* compares different mental states (e.g., true vs false beliefs).^{1,61} On the other hand, in the real-world changes in many outcomes are rarely linear.^{62,63} The *cusp catastrophe model* can explain why relatively small changes in a parameter can result in catastrophic changes in the state of a system. If distal factors are low, the *intentional system* will be heading in a fluctuating and linear way, depending on the proximal factors' severity), and characterized by inappropriateness of an intentional object or connection. If distal factors are severe, the intentional system will be heading towards a disruption or collapse, in a non-linear way, also depending on the proximal factors' severity, and characterized by an absence of intentional object and/or loss of conscious access to normal intentional objects. This sudden collapse can be explained by an already fragile system rather than by a novel stressor. Focusing on *disruptions of intentionality* theories can deepen and enrich our understanding of core disturbances involved in different psychopathologies, including psychosis, while strategies for sustainable management of such cases should focus on maintaining resilience.³⁵ We proposed an *intentionality failure theory* for psychosis, which occurs through a multifactor and usually non-linear fluctuating pattern of intentionality. This *what-matters model* would be useful, employing a combination of the principles of intentionality and causality, and opening new directions for research, treatment and prevention.^{13,41,60}

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Table 1. The biological, psychological, and socio-cultural (distal and proximal) factors interacting with the intentional system, in psychosis. If distal factors are not serious (low risk), the intentional system will be usually headed (depending on the proximal factors' severity) in a fluctuating and linear way, accompanied by inappropriateness of an intentional object or connection (A). If distal factors are severe (high risk) the intentional system will be headed in disruption or collapse (also depending on the proximal factors' severity), in a non-linear way, accompanied by an absence of intentional object and/or loss of conscious access to normal intentional objects (B) (as visualized in Figure 1).

Distal Risks – Predisposing Factors			
Intentional System	Biological	Psychological	Socio-cultural
Low pre-intentional sense of community Inappropriateness of an intentional object or connection	Genes Autoimmune disorders Aberrant salience	Early life adversity Anticipatory anxiety Delusional mood	Low sense of community Neglect Social alienation
Proximal Risks – Accelerating Factors			
Absence of intentional object Loss of conscious access to intentional objects	Viruses Psychoactive substances Homeostatic imbalance	Late life events Threat Entrapment	Lack of social support Lose Defeat

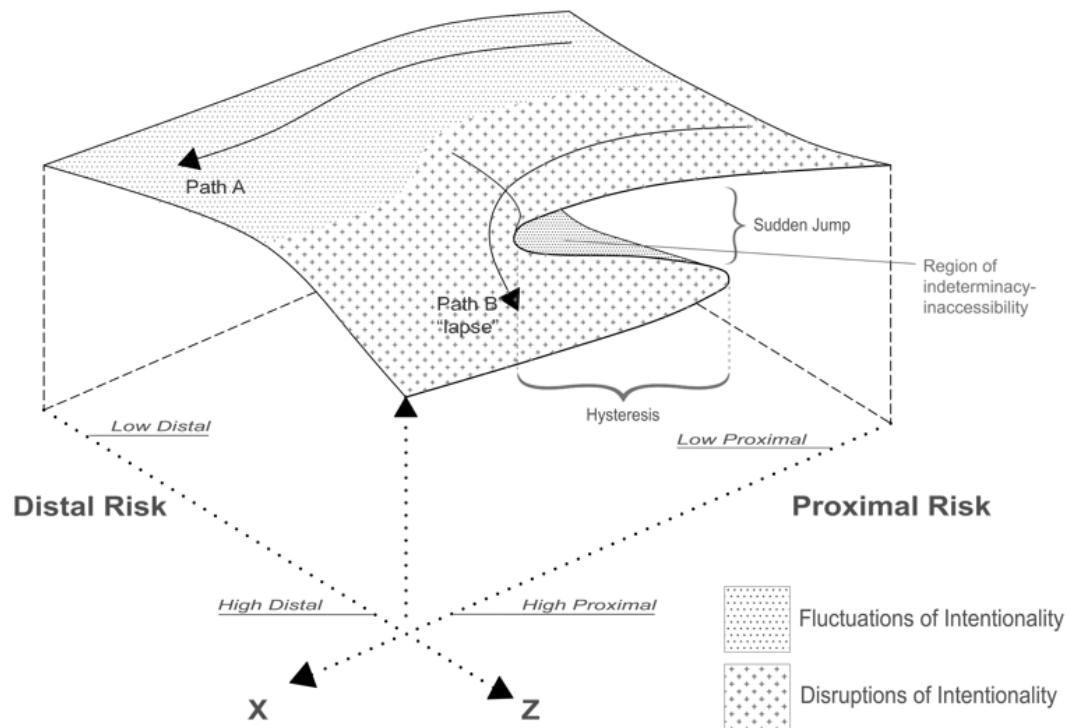


Figure 1. A visualization of the intentional system in psychosis, according to the cusp catastrophe model. Distal risk was defined as a predisposing factor that increases the probability of relapse. Proximal risk included any accelerating factor that immediately precipitated relapse, actualizing the statistical risk, and indicating the timing of relapse. If distal risk is low, then proximal risk will be linearly related to psychopathology (shown as Path A). If distal risk is high, then proximal risk is non-linearly related to psychopathology, and small changes in proximal risks may predict sudden relapse (Path B). The sudden collapse can be explained by an already fragile intentional system rather than a novel stressor. Most of the psychotic patients may be entrapped in the inaccessibility-indeterminacy region.^{30-33,1}

ΕΙΔΙΚΟ ΑΡΘΡΟ

Μοντελοποιώντας τις διαταραχές της προθετικότητας στην ψύχωση

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ΠΕΡΙΛΗΨΗ

Στη φιλοσοφία, η *προθετικότητα* σχετίζεται με την *κατευθυντικότητα* τη *σχετικότητα* ή την *αναφορικότητα* των ψυχικών φαινομένων. Φαίνεται να έχει στενή σχέση με την ψυχική αναπαράσταση, τη συνειδητότητα, καθώς και με εξελικτικά επιλεγμένες λειτουργίες. Η *φυσικοποίηση της προθετικότητας* με περιγραφικούς ή λειτουργικούς όρους αποτελεί έναν από τους σημαντικότερους σκοπούς της φιλοσοφίας του νου. Τέτοια αιτιακά μοντέλα είναι χρήσιμα, συνδέοντας τις αρχές της προθετικότητας και της αιτιότητας. Για παράδειγμα, ο εγκέφαλος περιέχει ένα *σύστημα αναζήτησης* που είναι υπεύθυνο για την ικανότητα για εγγενή ορμή προς κάτι ή προς το να θέλουμε κάτι. Αυτό συνδέεται με τη συναισθηματική μάθηση, τη συμπεριφορά αναζήτησης, την αναζήτηση μάθησης, αλλά και με τα συστήματα ομοιόστασης και ηδονής. Μπορούμε να υποθέσουμε ότι τέτοια συστήματα του εγκεφάλου αναπαριστούν κομμάτια ενός ευρέως *συστήματος προθετικότητας*, ενώ μη-γραμμικές δυναμικές μπορούν να εξηγήσουν την πολύπλοκη συμπεριφορά αυτών των χαοτικών και ασαφών συστημάτων. Ιστορικά, το *μη-γραμμικό μοντέλο καταστροφής* έχει χρησιμοποιηθεί στην πρόβλεψη συμπεριφορών στο χώρο της υγείας. Το μοντέλο αυτό μπορεί να εξηγήσει γιατί σχετικά μικρές αλλαγές μιας παραμέτρου μπορεί να καταλήξουν σε καταστροφικές αλλαγές στην κατάσταση του συστήματος. Εάν η *απώτερη επικινδυνότητα* είναι χαμηλή, τότε η *εγγύς επικινδυνότητα* θα συνδέεται γραμμικά με την ψυχοπαθολογία. Εάν όμως η *απώτερη επικινδυνότητα* είναι υψηλή, τότε η *εγγύς επικινδυνότητα* θα συνδέεται μη-γραμμικά με την ψυχοπαθολογία. Μικρές αλλαγές της *εγγύς επικινδυνότητας* θα προδικάζουν μια ξαφνική υποτροπή και μια ελάχιστη διατάραξη θα προδιαθέτει σε κατάρριψη χωρίς επιστροφή. Η φάση της *υστερήσης* εξηγεί επίσης γιατί το δίκτυο παραμένει ενεργές, αν και τα γεγονότα που οδήγησαν στην ενεργοποίηση έχουν εκλείψει. Φαίνεται ότι στους ψυχωτικούς ασθενείς υπάρχει διαταραχή της προθετικότητας εξαιτίας ακαταλληλότητας του *προθετικού αντικειμένου* ή σύνδεσης, ή παντελής απουσία του προθετικού αντικειμένου. Στην ψύχωση, η διαταραχή αυτή φαίνεται να συμβαίνει μέσω ενός πολυπαραγοντικού, μη-γραμμικού και κυμαινόμενου προτύπου προθετικότητας. Απώτερος σκοπός είναι η καλύτερη κατανόηση της υποτροπής. Η αιφνίδια κατέρριψη στο μοντέλο αυτό μπορεί να εξηγηθεί από την ύπαρξη ενός ευάλωτου *προθετικού συστήματος*, παρά από την εμφάνιση ενός στρεσογόνου γεγονότος. Το *μοντέλο καταστροφής* μπορεί να βοηθήσει τα άτομα να μετακινηθούν πέρα από τον *υστερετικό κύκλο*, και οι στρατηγικές για βιώσιμο χειρισμό αυτών των καταστάσεων θα πρέπει να βασίζονται στη διατήρηση της ανθεκτικότητας. Εστάζοντας στις *διαταραχές της προθετικότητας* μπορούμε να εμβαθύνουμε και να εμπλουτίσουμε την κατανόησή μας σχετικά με τις πυρηνικές διαταραχές που ενέχονται σε ποικίλες ψυχικές διαταραχές, περιλαμβανομένης της ψύχωσης.

ΛΕΞΕΙΣ ΕΥΡΕΤΗΡΙΟΥ: Προθετικότητα, ψύχωση, σχετικότητα, κατευθυντικότητα, μοντέλο καταστροφής, μη-γραμμική μοντελοποίηση.

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