

Disruptions of Intentionality and Suicidal Behavior

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Abstract

Suicide is one of the top ten causes of death in all ages. Entrapment plays a key role, characterizing the tunnel vision of individuals experiencing suicidal distress, with suicide becoming the only perceived escape route. Failure or loss of intentionality, precipitating entrapment, maybe is another key factor in the transition from suicidal ideation to enactment. There is not a typical suicide victim and the fluctuating pattern of suicidality can be better explained with non-linear models. The cusp catastrophe model can demonstrate the distal and proximal risks of suicide, including the spectrum of disturbed emotional intentionality. For those with high distal risk, a small increase in a proximal risk may push an individual to jump in a suicidal episode, in a non-linear way. On the other hand, a permanent loss of intentionality object dominates in patients with poor insight, who have feelings that do not represent anything outside of themselves. Emotional intentionality theories can enrich our understanding of the transition phase from suicidal ideation to suicidal action.

Key words

suicide, intentionality, emotion, feelings, emotional intentionality, intentionality failure, cusp catastrophe model, non-linear models, entrapment, suicide risk

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Suicidality

Suicide constitutes one of the major public health problems worldwide and account for almost 1 million deaths per year, which means a mortality rate of 16 per 100,000 [1]. Suicidal behavior is a complex phenomenon and the result of the interplay of various factors. These factors may be neurobiological, psychological, societal or individual, genetic, or environmental, chronic, distal, acute, and proximal [2, 3]. Research showed that suicide ideation fluctuates considerably over time, as do common risk factors such as hopelessness and perceived burdensomeness [4]. A suicidal gesture may serve as a dramatic way of alerting others, although those with a history of suicide attempts are about 20 times more likely to eventually end their own lives than those without [5]. Suicide, and also homicide, peaks in spring and summer may be explained partially by biological factors, like serotonergic alterations [6, 7]. There is a close interaction between serotonin, norepinephrine and dopamine; the hypothalamic-pituitary-adrenal axis; as well as with aggression/impulsivity, pessimism, neuroticism, and hopelessness endophenotypes [8]. The biological findings in depression seem to be similar with that in suicidal patients: decreased noradrenaline and serotonin neurotransmission, low BDNF concentrations, raised cytokines, dysregulation of the HPA axis and cortical and subcortical functional and structural brain changes [9].

The Acute Suicidal Affective Disturbance, as a clinical entity consisting of acute suicide risk and several related features, appears to be a unified clinical entity that may assist clinicians in determining a client's potential for death by suicide [10]. More than 3000 constructs/factors have been proposed and tested as possible risk factors for suicidal ideations and behaviors over the past 50 years [11]. Franklin et al (2017) [12] have summarized them in the following five categories: (1) internal psychopathology, like anxiety disorders; mood disorders; hopelessness; emotion dysregulation; sleep disturbances, (2) demographic factors, like age; education; employment; ethnicity; gender; marital status; religion; socioeconomic status, (3) prior suicidal thoughts and behaviors, like prior deliberate self-harm, non-suicidal self-injury, suicide attempt, suicide ideation, (4) external psychopathology, like aggressive behaviors; impulsivity; incarceration history; antisocial behaviors; substance abuse, (5) social factors, like abuse history; family problems; isolation; peer problems; stressful life events.

Intentionality

The Aristotelian philosophy of Descartes' days held that the universe was inherently purposeful or teleological. Spinoza asserts that every individual thing strives to persevere in its existence. He calls such striving *conatus* (a Latin term meaning *will* or *appetite*), and he argues that this *conatus* is nothing but the actual essence of the thing. *Intentionality* was introduced by St. Thomas Aquinas in the 13th century to Christianize the biological doctrine of Aristotle, using the concept to describe the process by which human beings and animals thrust their bodies into the world. For Franz

Brentano (1838-1917) [13] intentionality is seen as "directedness" of mental phenomena towards an object. For Martin Heidegger the subject is structured intentionally within itself, while for Walter Freeman (2000) [14], intentionality is neither objective nor subjective, it is certainly both. Among materialists, surgeons use intentionality to denote the biological process of healing by which a wound closes and the body re-establishes its integrity. Daniel Dennett (1987) [15] proposes *intentional stance* as so powerful that it can be developed into a valid *intentional theory*.

Intentionality seems to have essential connections with both consciousness and evolutionary selected functions, comprising the endogenous initiation, construction, and direction of behavior into the world [16]. Immanuel Kant used the term "self-organizing" in his 1790 "Critique of Judgment", where he argued that *teleology* is a meaningful concept only if such an entity whose parts or "organs" are simultaneously ends and means, exist. Millikan's (2017) [17] theory explains intentionality in terms that are broadly biological or teleological, 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 John Searle, (1990, 1995) [18, 19], intentionality is that feature of the mind by which mental states are *directed at*, or are *about* or *of*, or *refer to*, or *aim at*, states of affairs in the world. His theory of intentionality is opening also the problem of *intentionality of perception* using the expression of *experience of*. As he realizes the "of" of "experience of" is in short the "of" of Intentionality. Furthermore, Searle believes that *collective intentionality* is a biologically primitive phenomenon that we humans share with other social animals. Without collective intentionality there could not have been social reality and without a *pre-intentional sense of community* there could not have been collective intentionality. Taken together this implies that social reality would not have been possible without a *pre-intentional sense of community*.

Pre-intentional feelings

Moods seem to be a *pre-intentional state*, constituting the background in the context of which intentionally directed emotions target their objects. Moods are mechanisms whose function is to monitor the balance between demands and resources. They are characterized as *background feelings* or as styles of *anticipation* of experience. Moods appear to lack intentional object; but this lack does not deprive them of intentionality, since moods are states with a content which represents an unbound affective property [20]. For Angela Mendelovici (2013) [21], while some moods might in fact be directed at represented objects of some sort, other moods lack represented objects altogether. For example, some cases of anxiety really don't seem to be plausibly directed at anything. One just feels «anxious». Similarly, some cases of sudden elation really don't seem to be directed at the world as a whole, an unspecified object, or anything else.

Emotions are taken as forming an *anticipatory structure* of experiencing the world, a structure which makes intentional,

mental and bodily, acts possible. They have been characterized as *background feelings* or as styles of *anticipation of experience* [22]. In order to solve the philosophers' frame problem, Laura Candiotti (2019) [23] noticed that although *emotion rationality* is independent of the rationality of judgments, perceptions, and functional desires, emotions work in conjunction with judgments, perceptions, and functional desires. For de Sousa (1987) [24] emotions do "control" the salience of features of perception and reasoning. For Jaak Panksepp (1998) [25] emotion is the "stretching forth" of intentionality, which is seen in primitive animals preparing to attack in order to gain food, territory, or resources to reproduce. It seems that emotions have a *formal object*, which is the type of object at which their contents are directed, e.g., fear's formal object is what is *fearable* or *frightening* and anger's formal object is what is *worthy of irritation* [26]. The primitive form of emotion is called "motivation" or "drive" by behaviorists. Aristotle has described *orexis* as a kind of motion toward the *agathon*, i.e., something very close to what we recognized as *value*. In phenomenology, this *orectic striving* is what actually stretches the *temporal horizontality* or, better, what makes actual the 'traversing' of it [27].

Matthew Ratcliffe (2005) [28] offers an analysis of what 'existential feelings' consist of, showing how they can be both feelings and part of the structure of intentionality. In his paper "Feeling of Being" he reported that everyday talk of 'feeling' is not restricted to emotional feeling and the current emphasis on emotions has led to a neglect of other kinds of feeling, like feelings of homeliness, belonging, separation, unfamiliarity, power or control, which are ways of 'finding ourselves in the world'. He defines *existential feelings*, (as kinds of background feelings) as «pre-intentional» (rather than intentional themselves), i.e. as conditions of possibility of other intentional states: "We can still consider existential feelings intentional at least in the sense that they are always related to the world, that we experience as a whole. They make a considerable contribution to the structure of experience, thought and action, giving the examples of feeling 'complete', 'flawed and diminished', 'unworthy', 'humble', 'separate and in limitation', 'at home', 'a fraud', 'slightly lost', 'overwhelmed', 'abandoned', 'stared at', 'torn', 'disconnected from the world', 'invulnerable', 'unloved', 'watched', 'empty', 'in control', 'powerful', 'completely helpless', 'part of the real world again', 'trapped and weighed down', 'part of a larger machine', 'at one with life', 'at one with nature', 'there', 'familiar', 'real'".

In the neurophysiological level, the initial undifferentiated emotional states in the limbic system, through the mediation of feelings, are transformed into *nuanced emotions*. Porges's *polyvagal theory* [29], is the neurological basis of social engagement, which is evolutionarily linked to the autonomic nervous system and it relays emotional experience. The *interoceptive information* appears to help to generate a heightened physiological awareness of salient stimuli and appropriate behavioral responses [30]. On the other hand, the *insula* appears to interact as a hub, integrating "salient" stimuli and events with *visceral* and *autonomic nervous system* activity [9]. *Predictive processing* is a theoretical framework that posits that the brain's overall function is to minimize long-term average prediction error, maintaining the organism in its expected homeostatic states [31]. However,

prediction error minimization is not sufficient to explain all mental phenomena, and *affectively salient stimuli* can capture our attention even when precision expectations are low [32]. Anterior insular cortex provides a natural locus for comparator mechanisms underlying *interoceptive predictive coding*, a model suggested as a new view of emotional feelings as *interoceptive inference* [33]. Chronic anxiety has been suggested to result from heightened interoceptive prediction error signals, while disrupted interoceptive predictive coding may causally account for many other psychiatric disorders [34].

Failures of intentionality

For Peter Goldie (2002) [35] there are two types of emotional feelings. (a) *bodily feelings*, characterized by consciousness awareness of bodily changes that occur during emotions (muscle activity, autonomic nervous system responses, and hormonal changes), (b) "*feeling-towards*", characterized by an unreflective emotional engagement with the world beyond the body. One can unreflectively feel an emotion without being reflectively aware that one is feeling an emotion. Feelings towards, have an *intentional object* outside the body, whereas bodily feelings have intentional objects within the body. As a *grief-pang*, a bodily feeling specifically associated with the emotion of grief, it borrows the intentional object of grief, and is consciously apprehended as a bodily-feeling-towards the loss. But, is it a feeling of the body, or a *feeling toward the loss*? Benjamin Sheredos (2009) [36] suggested that we can have it both ways, but not at once: A conscious experience of a single, current bodily state can be consciously apprehended as being directed at the body at one moment, and can be apprehended as being directed at the world the next. When the latter occurs, we experience states of our bodies as properties of the environment. For Sheredos: "Our bodily feelings become a phenomenological lens, through which we experience an object in the world as emotionally salient. When involved in an emotional feeling towards, bodily feelings don't just borrow intentionality, they loan out phenomenology. The phocal point is out there, with the loss, not in your innervated body. It is the loss that is felt as grievous, via the pang in one's chest"

On the other hand, Derek Bolton (2001) [37] has been reported: "Contemporary theories of mind commonly take intentionality to be fundamental, and I consider to what extent mental disorder can be defined in terms of *radical failures of intentionality*. Dennett has suggested that *breakdown of intentional systems* is to be explained from the physical stance, but explanations of breakdown from the design stance and even from the *intentional stance* are possible. Evolutionary theory emphasizes the intentionality of mind and behaviour, and is increasingly applied in models of psychopathology". For Edmund Husserl [38], the temporal microstructure of consciousness—as intentional—consists of a dynamic self-organizing process comprised of both a retention of what I have just seen, heard, or thought, as well as an anticipatory *protention* of what I expect to continue seeing, hearing, or thinking. This temporal synthesis ("inner time consciousness") is a tacit background process organizing

our experiences into sequences of coherent units. For Thomas Fuchs (2007) [39], this temporal microstructure of intentional consciousness can become fragmented in psychotic patients.

Focusing on *disruptions of intentionality* can deepen and enrich our understanding of core disturbances involved in different psychopathologies. For Bolton (2008) [40], the intentional connections between delusions and behavior are appropriate, given their experiences. He has considered whether mental disorders might be specified by a class of “radical failures” of intentionality, exhibited in the patient’s mental life, noticing: “The mind is in good working order to the extent that its intentional objects and connections are appropriate [...] failure of intentionality, whether inappropriateness of an intentional object or connection, or absence of an intentional object altogether, suggests disorder”. Bolton uses the example with patients suffering from *Capgras* and *Cotard* delusions who lose conscious access to normal intentional objects of affective experience, and thus explaining the delusion in the absence of an intentional object. These patients have not lost mental states whose intentional objects are familiar people, or themselves, reporting that their alleged spouse feels unfamiliar, or that their body is not real, respectively. It is only the patient’s affective experience that is restricted to a subset (or, null-set) of appropriate intentional objects [37, 40].

Entrapment and suicidality

There is no “typical” suicide victim. It is also important to reassess suicidal ideation frequently due to its fluctuating pattern. *Neuroticism* is a personality trait that has been linked to negative affect, while *hopelessness* maybe is a clinical endophenotype associated with suicidal behavior. Pollock and Williams (2004) [41] propose that suicidal behavior is associated more with hopelessness than with the severity of depression, while a negative correlation between prefrontal 5-HT_{2A} receptor binding and hopelessness has been reported [42]. Moreover, *impulsivity*, characterized by disinhibition and a tendency to act quickly on urges or in response to stimuli, increases the risk of suicidal behavior and it has been defined as a personality trait or emotional style [9].

In the integrated motivational–volitional (IMV) model, *entrapment* is hypothesized to play a key role. This characterizes the ‘tunnel vision’ of individuals experiencing suicidal distress and suicide becomes the only perceived escape route. Within the “motivational phase” of this model, suicide ideation is posited to result from feelings of defeat and entrapment, which are, in turn, moderated by feelings of thwarted belongingness and a lack of social support [43]. The “volitional phase”, is hypothesized to govern behavioral enaction, such that a suicide attempt is argued to be the result of the interaction between additional risk factors such as impulsivity or fearlessness about death and suicidal thoughts. There have been proposed reinforcing feedback loops like: entrapment > suicide ideation > defeat > entrapment, or increases in feelings of defeat > stronger feelings of entrapment > more suicide ideation > stronger feelings of defeat [44]. O’Connor & Kirtley (2018) [43] have been proposed some key premises of the IMV model of suicidal behavior: (a) The presence of pre-motivational vulnerability factors (b) Vulnera-

bility factors combined with stressful life events, including early life adversity (c) Defeat/humiliation and entrapment as the key drivers for the emergence of suicidal ideation (d) Entrapment as the bridge between defeat and suicidal ideation (e) Volitional-phase factors govern the transition from ideation/intent to suicidal behaviour. We may suggest that adding emotional intentionality disruptions close to the entrapment could offer better explanations in our understanding of this suicidal process.

Entrapment is a potentially modifiable predictor of suicide attempts over time and the challenge is that there are not yet any evidence-based treatments to reduce entrapment. Possibly, entrapment is the result of an emotional intentionality failure. Entrapment was found to be directly related to suicidal ideation in adolescents, but it also acts as a mediator of the relationship between anger suppression and suicidal ideation [45]. Elevated defeat, entrapment and suicidal behaviour have also been found in individuals with emotional trauma and a diagnosis of post-traumatic stress disorder (PTSD), while defeat and entrapment mediate the relationship between PTSD symptoms and suicidal behavior [46]. Entrapment, also, mediates the relationship between ruminative flooding, panic-dissociation and fear of dying with suicidal ideation [47]. Wetherall et al (2019) [48], have found that entrapment was a mediator of the relationship between defeat and suicidal ideation cross-sectionally, supporting the IMV model’s prediction. Furthermore, when entrapment was high, resilience also moderated the relationship between defeat and suicidal ideation.

Recent studies introduced the *suicide crisis syndrome* (SCS), a condition associated with imminent suicidal behavior and characterized by (a) a pervasive feeling of entrapment in which the escape from an unbearable life situation is perceived as both urgent and impossible (Criterion A) and (b) affective disturbance, loss of cognitive control, hyperarousal, and social withdrawal (Criterion B). Bloch-Elkouby et al (2020) [49] findings support both the suggested symptoms of the SCS and the central role of entrapment in the proposed criteria for the syndrome. *Emotional pain* appears to be closely linked to *entrapment* and may belong in Criterion A. The SCS is an evidence-based pre-suicidal cognitive and affective state predictive of short-term suicide risk. The recent formulation SCI-2, proposed as a suicide-specific DSM diagnosis, features a feeling of *entrapment* accompanied by four additional symptom clusters: *affective disturbance*; *loss of cognitive control*; *hyperarousal*; and *social withdrawal*. According to the authors: “The SCI-2 is a valid and reliable tool to assess the presence and intensity of the Suicide Crisis Syndrome and to predict short-term prospective suicidal behaviors and attempts among psychiatric outpatients and inpatients, regardless of patients’ readiness to disclose suicidal ideation” [50].

The Cusp Catastrophe Model in Suicidal behavior

The *ideation-to-action framework* is the basis for several theories, although they share the belief that suicidal desires do not automatically lead to suicidal actions. In all theoretical models there are distinct stages with its own risk factors, processes, and

explanations for the progression from suicidal ideation to lethal suicide attempts [11]. However, in the real world changes in many outcomes are rarely linear. Historically, the *cusp catastrophe model* has been applied to the prediction of health behaviors [51]. It can explain why relatively small changes in a parameter can result in catastrophic changes in the state of a system. Rene Thom (1975) [52] originally proposed the *catastrophe theory* to understand complicated phenomena that included both gradual, continuous change and sudden, discontinuous or catastrophic change. Robert Gilmore (1993) [53] pointed the following five elements for the presence of *catastrophe*: 1. *bimodality* (i.e., existence of two distinctly different behavioral modes); 2. *sudden jump* (i.e., abrupt changes in outcomes between the two modes even with slight changes in the predictors); 3. *inaccessibility* (i.e., an outcome unlikely to be in the area between the two modes); 4. *hysteresis* (i.e., the change of an outcome from one mode to the other cannot be determined by control factors of the same value); and 5. *divergence* (i.e., a slight change in the control factors can lead to substantial change in the outcome and deviation from the linear model).

The unpredictability of suicide is one of the mental health challenges. The *cusp catastrophe model* is capable of quantifying such a quantum mechanism to simultaneously consider linear and nonlinear relationships along with sudden jumps in outcome measures. In summary, the cusp catastrophe model would be a particularly appropriate statistical approach when an outcome measure has the properties of bimodal distribution (bimodality) with spurts (sudden jumps), an inaccessible middle region between these two modes (inaccessibility), a delay between these transitions (hysteresis), and deviation from a linear relationship between the response outcome measure and the predictors (divergence) [54]. Kira et al (2019) [55], estimating cumulative stressors and traumas as bifurcation control factor, have found that the explained variance by cusp catastrophe non-linear model was highly superior to the linear model in predicting suicidality. *Distal risks* may be conceived as background or relatively stable characteristics, e.g., previous history of suicidal behaviour. For those with high distal risk, the associated suicidal ideation pathways may resemble paths B and C in Figure 1, where a small increase in proximal risk may push an individual “over the edge,” leading to suicidal episode. Similarly, Randolph Nesse (2004) [56] proposes the *cliff-edged fitness function*, where, as fitness increases the system fails catastrophically in some individuals. A *prolapse* then requires a substantially larger reduction in proximal risk to help the individual regain a healthier stage [57]. When a certain threshold level of stress is reached a “catastrophic” transition from low to high risk occurs, although the system is slowly getting less and less resilient, and the sudden collapse can be explained by an already fragile system rather than a novel stressor [58].

In order to get back from a high to a low-risk phase, it is not sufficient to restore stress levels to the level before the collapse. This dependence of the current state of the system on the previous state is called *hysteresis* (an Ancient Greek word meaning “deficiency”). Hysteresis is the dependence of the state of a system on its history, e.g, magnetic hysteresis loops are mainly *rate-independent*, which makes a durable

memory possible. Hysteresis in cell biology often follows bistable systems where the same input state can lead to two different, stable outputs. T cells exhibit hysteresis, taking a lower signal threshold to activate T cells that have been previously activated. Also, some neurons do not return to their basal conditions from a stimulated condition immediately after removal of the stimulus is an example of hysteresis [59]. In a parallel way of thinking, the *Kuramoto model* was motivated by the phenomenon of *collective synchronization*, when a huge system of oscillators spontaneously locks to a common frequency, despite differences in the individual frequencies of the oscillators [60]. Synchronization phenomena are manifestations of the maximization of information exchange, resulting in a widespread redistribution of energy/matter [61]. The *Kuramoto model* highly simplified computational model of coupled oscillators help us understand brain dynamics, which might bear some relevance to clinical psychiatry [62].

Indeed, several neurobiological studies can explain this described process in suicidal patients. In the case of panic disorder, the “anticipatory anxiety” for the impending panic attack appears to be located in the limbic system, and the ability of panic attacks to produce generalized anxiety may be viewed as an example of the *kindling phenomenon* that occurs in the limbic lobe with stimuli coming from the brainstem [9]. Also, dysregulated hypothalamic-pituitary-adrenal (HPA) axis function was proposed decades ago within the stress-diathesis model. However, cortisol levels were not found to be related to either the duration or the severity of suicidal ideation [63] and individuals who reported suicidal ideation had lower cortisol levels compared to those without suicidal ideation [64].

Moreover, *microglia* is regarded to play crucial roles in brain homeostasis and inflammation. Post-mortem examinations of suicide victims’ brains show an increased density of microglia in the anterior cingulate cortex, the dorsolateral prefrontal cortex, and the mediodorsal thalamus regions [65], while in living subjects, PET scans show increased microglial activation in the presence of suicidal ideation [66]. *Translocator protein* (TSPO) is upregulated in activated microglial cells and is an indication of *neuroinflammation*. TSPO was shown to be significantly increased in those with suicidal ideation, most robustly in the regions of the anterior cingulate cortex and the insula [66]. Also, lithium’s anti-aggressive and suicide-preventive capacity in clinical practice is well established, while numerous ecological studies indicate similar effects of nutritional lithium [67, 68]. Recently, Schoepfer et al, (2021) [69] applied a new technique (a neutron-induced coincidence method) utilizing the ${}^6\text{Li}(n,\alpha){}^3\text{H}$ reaction for the position sensitive, 3D spatially resolved detection of lithium traces in post-mortem human brain tissue in suicide versus control. Controls had a significantly higher endogenous lithium concentrations in white compared to gray matter, while suicide people a lower lithium concentrations in emotion-modulating regions.

An emotional intentionality failure theory for suicide

We know that emotions can have the following aspects: They are often private and subjective *feelings*; they can take the form of *actions* which are considered as “emotional”, such as defense, attack or the response to a threat; they are states of bodily stimulation, expressions or demonstrations of distinct responses, physical or of the autonomic nervous system; they are *motivational programs* that coordinate responses in order for specific adaptation problems to be resolved. We also know that when individuals are in a *negative affective state*: (a) In order to process the information, they strongly use their cognitive sources, (b) they carefully process incoming information, paying attention to details, (c) They thoroughly examine the new information and doubt the usefulness or accuracy of their knowledge or their performance, (d) The sad mood colors the information that “the environment is not safe”. Into this phase, depressive or psychotic patients follow non-adaptive cognitive-emotional strategies: *rumination* (a constant thinking about negative emotions); *catastrophizing* (the particular emphasis on the negativity of the situation); *self-blame* (blaming oneself for the situation); *other-blame* (blaming others for the situation); *avoidance*, *inhibition*, *suppression* and *holding back negative emotions* which exerts adverse effects on physical health [9].

There are three main *ideation-to-action frameworks* of psychological theories for suicide: (1) The *Interpersonal-Psychological Theory of Suicide* (IPTS) [70] predicts that the combination of feeling like a *burden to others* (“perceived burdensomeness”) and being *socially alienated* (“thwarted belonging”) will produce death desires or suicidal ideation. (2) The *Three Step Theory* [71], proposes that the two most frequently reported factors that preceded suicide attempts and deaths were the presence of *pain* (emotional misery or pain) and feelings of *hopelessness* about the future. (3) The *Integrated Motivational-Volition Theory* (IMV) model [72] differs from the IPTS in several ways, including that the pathways to suicidal ideation are *defeat* and *entrapment* instead of belongingness and burdensomeness. In brief, the IMV model is a tri-partite model that describes the biopsychosocial context in which suicidal ideation and behaviour may emerge (*pre-motivational phase*), the factors that lead to the emergence of suicidal ideation (*motivational phase*) and the factors that govern the transition from suicidal ideation to suicide attempts/death by suicide (*volitional phase*).

Having in mind the phenomenology of intentionality, together with the theoretical and research findings in the area of affective or emotional intentionality, and findings in neuroscience, we may suggest that the key feature of the second phase (motivational phase - IMV model) is the *failure of intentionality*. This failure seems to be strongly tied with, and precipitate, entrapment. In this phase the patient’s affective experience is restricted to a subset (or, null-set) of appropriate intentional objects [40]. At this point, *emotional pain* leads to *entrapment*, and through a tunnel vision the suicidal thoughts are a very possible solution [49]. Consistent with the *differential activation hypothesis* [73], we would expect that the process between ideation and enactment shortens with

repeated engagement in suicidal behaviour, such that over time the transition between intention and behaviour becomes increasingly rapid. Thus, we can suggest that suicidal ideation becomes an encoded phenomenon, like the property of panic attacks to produce generalized anxiety, as a process of a *kindling phenomenon* occurring mainly in the limbic lobe. A recent study showed that an inter-network gamma connectivity with fronto-parietal-control-network and default-mode-network might be the key neuropathological interactions underling the progression from suicidal ideation to suicidal attempt [74].

The application of cusp catastrophe modeling within the field of suicide prevention has been demonstrated by Bryan and Rudd (2018) [75]. Within a sample of 76 active duty U.S. army soldiers, they showed that over time, those with a multiple attempt history displayed two stable states, corresponding to a high-risk state and a low-risk state for suicidal behavior. Also, searching for psychoactive substance abuse in the army, and using the Antonovsky’s sense of coherence questionnaire in 1,098 young male conscripts, Giotakos (2003) [76] have been found that those with suicidal ideation had a significantly lower sense of coherence questionnaire score, as compared with the whole sample. Also, subjects with past or current substance use had a significantly higher incidence of past or current, respectively, suicidal ideation or behavior, as compared with those without a history of substance use.

Intentional acts do not require awareness, whereas voluntary acts require self-awareness. It seems that in the third phase (volitional phase) of the IMV model, a loss of intentionality occurs. In this *loss of intentionality phase* there is an absence of intentional object and an absolute loss of conscious access to normal intentional objects. We may also suggest that the third phase of the IMV model is not a “volitional” phase. In this final phase the suicidal person is yet entrapped and hopeless. He has no choice; he acts by chance, according impulsive choices that have been encoded in limbic system. In general, in this transition from suicidal ideation to suicidal action, the suicidal person behaves, more or less, in an unconscious, but not in a volitional state. In this phase the person has not the ability to organize or to plan something in a volitional state of clear consciousness. This transition from suicidal ideation to suicidal act is not a result derived from a clear and volitional planning, as IMV model assumes. The suicidal action in this *catastrophe point* is the result of despair, loss of emotional intentionality, and entrapment, in a psychological environment with vague feelings. (Figure 1).

Loss of Intentionality and Suicide in Psychotic patients

Research has long noted higher prevalence rates of suicidal thoughts and behaviors among individuals with psychotic symptoms. But what is happening with suicidality in the *negative syndrome of schizophrenia*? In a meta-analysis of longitudinal studies Huang et al (2018) [77] have found that positive symptoms consistently conferred risk across outcomes, while negative symptoms were not significantly associated with suicidal ideation, and were “protective” against death. Massons et

al (2017) [78] have found that those subjects aware of having a mental disease (higher insight) had higher scores on suicidality than those with poor insight, although no insight dimensions survived the multivariable regression model. They have found also that depression and previous suicidal behaviour to predict suicidality, while the Markova and Berrios Insight Scale total score and two specific domains (awareness of “*disturbed thinking and loss of control over the situation*” and “*having a vague feeling that something is wrong*”) were related to suicidality.

Except psychosis, *poor insight* arises as a common mechanism in many other mental disorders, or even it would be an independent and trans-diagnostic factor into the human personality. A variety of phenomena might be considered as reflecting *impaired insight* in psychosis, like failure to recognize signs, symptoms or disease, failure to derive appropriate cognitive representations, despite recognition of the disease, and misattribution of the source or cause of the disease. Intentional acts, also, do not require awareness, but voluntary acts require self-awareness. The theoretical approaches regarding impaired insight include (a) the disturbed perceptual input, (b) the impaired linkage between thought and emotion and (c) the breakdown of the process of self-monitoring and error checking. On the other hand, the *salience network* is an intrinsic brain circuit that plays a key role in stimulus processing, attention, and transition/switch between mental states. The main structures involved are the *anterior insula* and the *anterior cingulate cortex*. This system is responsible for switching the brain's *default network* from default mode to task-related activity mode, and it is thought to be involved in the development of psychotic symptoms. Delusions are a cognitive effort by the patient to make sense of these aberrantly salient experiences, whereas hallucinations reflect a direct experience of the *aberrant salience* of internal representations [79-81].

We remain confounded by the so-called “hard problem”: an inability to explain in scientific terms the phenomenal “feel” of conscious experience [82]. For Edelman et al (2011) [83], the hard problem does not require a solution, but rather, a cure. For this experience we may consider *information transfer, or exchange, as energy processing exchange, resulting from any type of interaction, basically, the flow of energy* [61]. In line with the experiments of Libet et al. (1983) [84], our ability to make choices might arise from random fluctuations in the brain's background electrical noise, and this suggests that individuals might well behave independently of cause and effect [85]. In the example of the *Müller-Lyer illusion lines*, 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. The distinction between correct and incorrect representation is often regarded as a central normative distinction and a capacity to *misrepresent* is often thought to be essential for representing: no possibility of misrepresentation, no representing. This connects with Roderick Chisholm's (1957) [86] concern with non-existent objects, since a capacity to misrepresent amounts to a basic capacity to represent non-existent objects. We may suggest that in the negative syndrome of schizophrenia there exist a loss of both: the intentional object and the capacity to misrepresent.

For Watsuji (1996) [87], some of the causes of schizophrenia do not reside solely within the individual but also within the dynamics of their broader relation to a shared world. The author introduced the concept of *betweenness*, which can take many forms: from the bodily intimacy of newborn-caregiver interactions or sexual intercourse, to more encompassing forms of betweenness like emotional contagion within large groups. For Searle [18], “the crucial element in collective intentionality is a sense of doing, wanting, or believing something together, and the individual intentionality that each person has is derived from the collective intentionality that they share”. *Shared intentionality*, as a synonymous of collective intentionality, have been described as the power of the mind to share mental states like emotions, intentions, and beliefs with others. There are two forms of shared intentionality: *joint intentionality* and *we-intentionality*. By contrast to joint intentionality, *we-intentionality* relies on the agents' capacity to understand themselves as group members and to adopt the group's perspective. Salice & Henriksen (2021) [88] propose that in schizophrenia spectrum disorders *we-intentionality* is impaired, in contrast to joint intentionality which remains unaffected. As an example from the field of suicide, efforts by Aboriginal groups to preserve and promote their culture and identity were found to be associated with dramatic reductions in rates of youth suicide [89].

Raw feelings do not represent anything outside of themselves; they have no connection with anything outside of the mind itself. For example, anxiety has no intentional object while fear has such an object. So, it is possible that in patients with the negative syndrome of schizophrenia there is a permanent loss of intentionality, in combination with feelings that do not represent anything outside of themselves. Having in mind the above theories and findings, we may also suggest that in the negative syndrome of schizophrenia dominates a permanent loss or lack of intentionality, including an absence of intentional object, from the beginning, or even from the primordial phase of the disorder. For this reason, it is very difficult for them to recover from the *indeterminacy region* (see Figure 1).

Epilogue

In this paper I propose an *intentionality failure theory* for suicidality. Visualization may offer a more intuitive way to understand the described findings. Figure 1, taking the idea from the cusp catastrophe model, demonstrates a multifactor fluctuating pattern of suicidality. Here, intentionality failure is strongly tied with entrapment. If distal risk is low, then proximal risk will be linearly related to suicidality (shown as Path A). If both distal and proximal risks are high the individual is in high risk of suicidality, but if distal risk is high and proximal risk is low, an individual is in low risk for suicidality. If distal risk is high, then proximal risk is nonlinearly related to suicidality and small changes in proximal risk predict either sudden lapse (Path B) or prolapse (Path C). Strategies for sustainable management of such cases should focus on maintaining resilience [58].

Our intentionality is derived from genes. In order for them to be able to survive, the people composed of these genes

have to produce intentional states. Moreover, affective intentionality is an embodied and enactive process that connect us to a shared world and guide our dealings with it. This *what-matters model* [90], would be useful, employing a combination of the principles of intentionality and causality. Having

in mind the rich philosophical and neuroscientific research, we can suggest that the field of emotional intentionality is a prominent scientific area, and can help us better understand of emotion-cognition interplay, emotion (dys)regulation, psychopathology, or even suicidality [91-95].

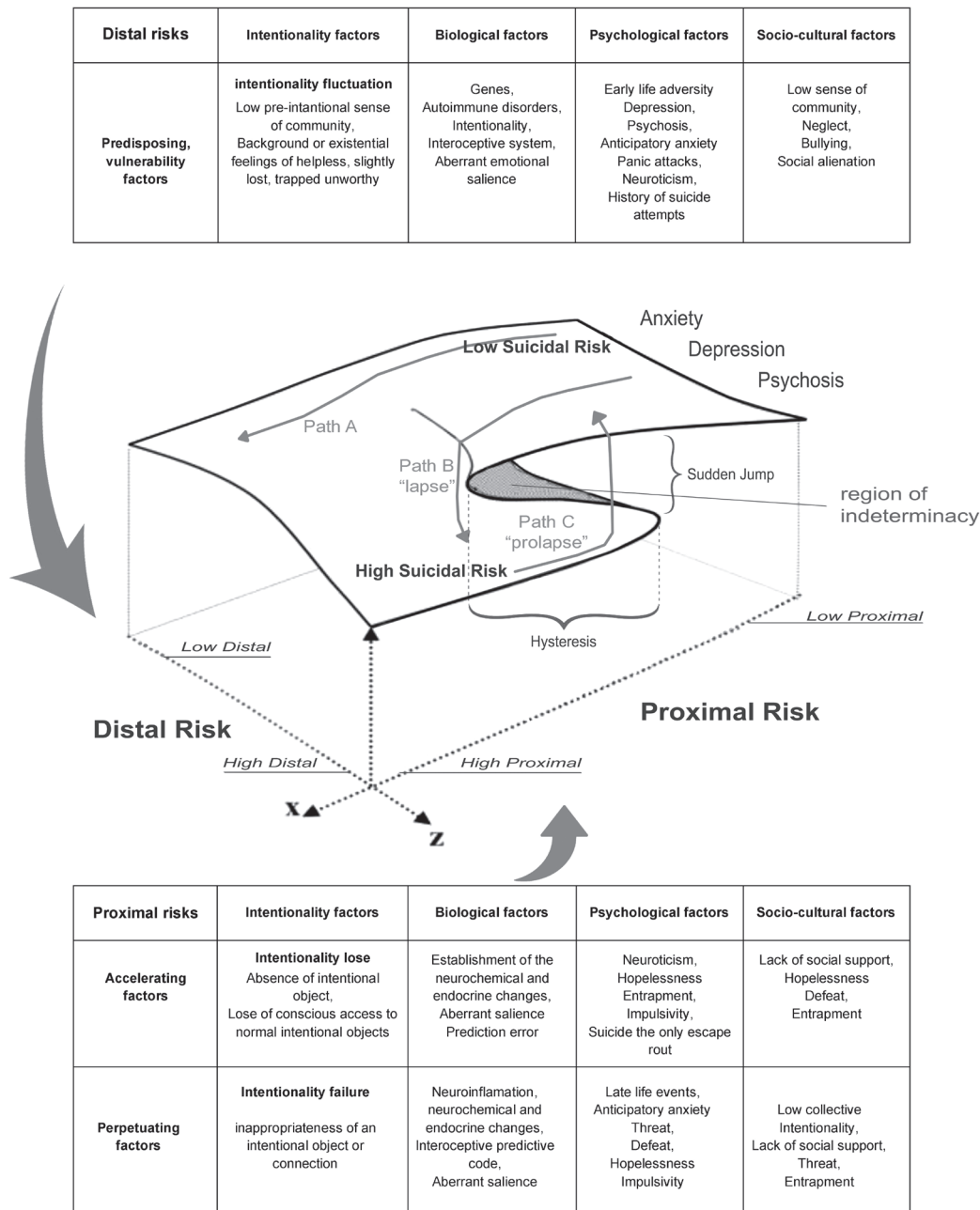


Figure 1. A pattern of suicidal ideation to suicidal action process, according to the cusp catastrophe model. The diagnosis spectrum from anxiety to psychotic disorder, represents a spectrum from low to high suicidality. Distal risk was defined as the splitting parameter, which follows the z-axis and proximal on the x-axis and predicts the direction of the change in suicidal behavior. Distal risk was defined as a predisposing factor that increases the probability of relapse. Proximal risk included any accelerating factor that immediately precipitated suicidal thoughts. Perpetuating factors can be a mixture of both, in long standing cases. If distal risk is low, then proximal risk will be linearly related to suicidality (shown as Path A). If both distal and proximal risks are high the individual is in high risk of suicide, but if distal risk is high and proximal risk is low, an individual is in low risk for suicide. If distal risk is high, then proximal risk is nonlinearly related to suicidality and small changes in proximal risks predict either sudden lapse (Path B) or prolapse (Path C). Suicidal patients with poor insight, usually suffering from psychosis or psychotic depression, could be represented in the indeterminacy region [44, 51-55].

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