

**RESEARCH ARTICLE****A Model of Post-Traumatic Stress Disorders and Dissociative Identity Disorder from the perspective of Social Emotions****Author**

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Email: [fontalex00@gmail.com](mailto:fontalex00@gmail.com)DOI: <https://doi.org/10.18103/mra.v10i3.2743>**Abstract**

Post-Traumatic Stress Disorder, Complex Post-Traumatic Stress Disorder and Dissociative Identity Disorder are conditions caused by exposure to one or more stressful events of extraordinary magnitude and/or repeated over many years. The traumatic experience(s) may have different outcomes in different persons: some people fully recover within a short time, while others go on to develop one of these three disorders, whose interdependencies are still poorly understood. The present work utilises an updated version of a model of mental functioning, that has been previously applied to schizophrenia, to provide an interpretation of the aforementioned conditions. The model, built through the method of Artificial Life and with the toolset of Artificial Intelligence, foresees that the mind is subject to two forces: trauma, which represents the attack on the mind, and dissociation, which embodies the mind defence in both physiological and pathological conditions. The balance between these forces determines the pathological outcome.

## 1. Introduction

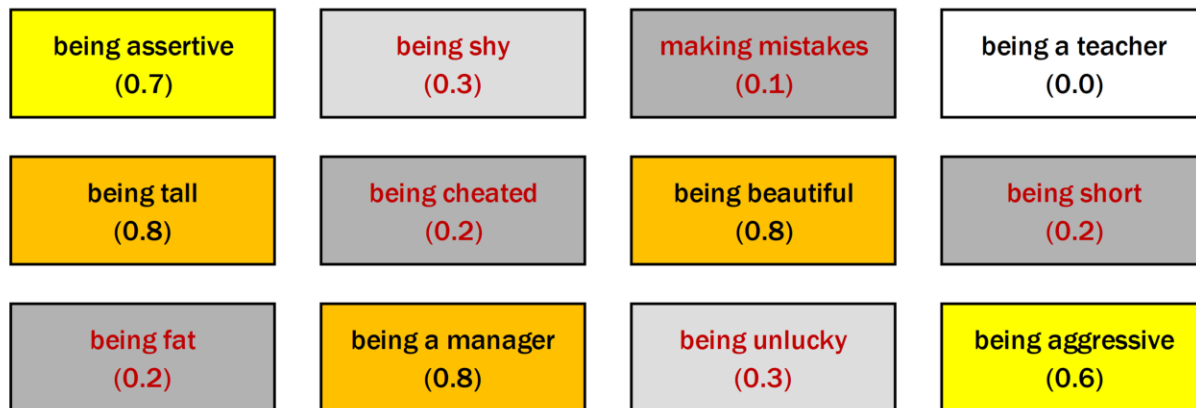
PTSD (Post-Traumatic Stress Disorder) is a condition caused by exposure to one or more stressful events of extraordinary magnitude or catastrophic nature; C-PTSD (Complex PTSD) is a disorder caused by severe, persistent and/or repeated psychological traumas; a third pathology induced by traumatic experiences is DID (Dissociative Identity Disorder), characterised by the presence of different personality states. All these conditions have their origin in traumatic experiences, but their degree of severity and course can be quite variable.

The differential diagnosis between PTSD and C-PTSD has been object of debate. It has been speculated that C-PTSD may be equivalent to PTSD, when the latter is comorbid with BPD (Borderline Personality Disorder), although this hypothesis seems to have lost its appeal [1]. Also, C-PTSD and DID display a significant degree of comorbidity [2] and, overall, there seems to be quite a significant overlap between the three

conditions, but their exact interdependencies remain poorly understood.

The field of psychology is characterised by a steady proliferation of diagnostic models and subcategories (541 in DSM-5, described in 947 pages, versus 128 in DSM-1, described in 132 pages). This approach, which seems to be inspired by the principle of “divide and inflate”, contrasts with experimental evidence, which suggests that symptoms, environmental and genetic risk factors are shared among many disorders. Our approach is aimed at reversing this trend.

The objective of this work is twofold. On the one hand, we wish to propose an updated version of a model of mental functioning, inspired by the method of Artificial Life [3] and built with the toolset of Artificial Intelligence, which has already been used to explain and interpret schizophrenia [4]. Secondly, we will utilise the model to provide an interpretation for the shared and unique features of PTSD, C-PTSD and DID.



**Figure 1:** Features and social value. The mind uses a large set of ideas or features to make sense of the world. Features have an associated property called social value, which can be high (highlighted in yellow) or (highlighted in grey), to various degrees.

This paper is divided into six parts: this first section is the introduction; the second section is dedicated to the model of emotions; the third section incorporates the model of emotions into a model of mental functioning in normal conditions; the fourth section introduce a reinterpretation of the concept of dissociation; the fifth section uses the model to provide an interpretation of PTSD, C-PTSD and DID; the last section draws the conclusions and outlines future research directions.

## 2. A model for social emotions

Many theories on the role and origin of emotions exist [5], as well as computational models of the process of emotion generation [6]. Our

hypothesis is that emotions were evolved to serve three basic needs. The first need is survival (task involving one individual) and *fear* is probably the most primitive emotion evolved to serve this need: avoiding physical damages and escaping death. Besides favouring the avoidance of physical damages, fear (or its close relative, anxiety) also plays another role: it serves to avoid other “bad” emotions: we can have, e.g., fear of shame, fear of guilt, fear of emotional pain.

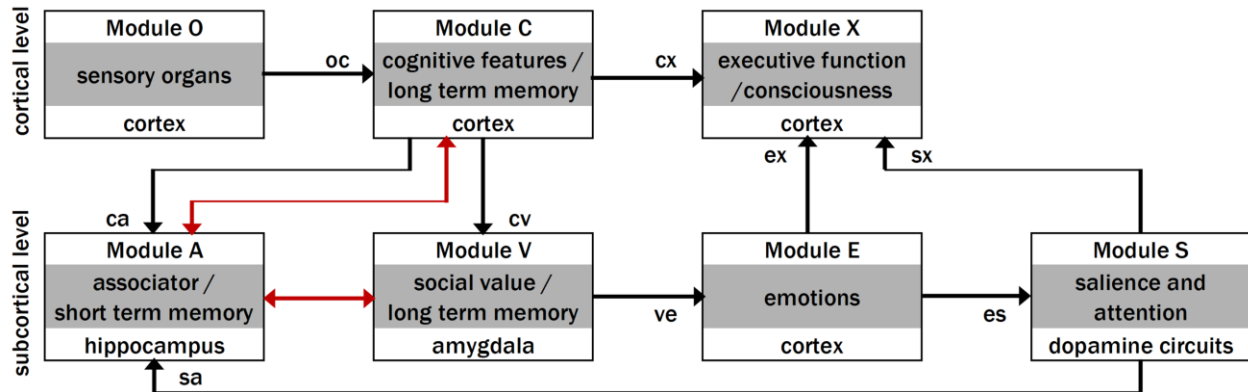
The second need is reproduction (task involving two individuals). *Love, jealousy and romantic pain* (the pain caused by the loss of a romantic /sexual partner, by either abandonment or death) serve this need: finding and keeping a



associations. Value is a long-term property, expected to change on a slow time scale.

We postulate the existence of a module in the human mind which encodes the social value of features (Fig. 2, left). This module receives in input a set of active features, characterising the situation perceived by the mind, and “loads” them with value. Based on these value-loaded features, it computes two variables, called *sval* (self value) and *oval* (object value), determined by the combined effect of the values of all *active* features associated

to self and object, respectively. Since the set of active features depends on the situation, so does the self value. A person, for example, can have an important role at work and, as a result, his/her self value will be high at work, where work-related features (office, colleagues, meeting rooms) are active. The same person may have been belittled by his/her parents and, as a result, his/her self value will be low at home, where home-related features (bedroom, parents, old photographs) are active.



**Figure 3:** Process of emotion generation. Cognitive features are activated and sent to consciousness and to the associator, where their co-activations are recorded. The next processing step is carried out by the social value module, where cognitive features are loaded with value. Based on this information, the emotions module produces an emotion, which is presented to consciousness. Emotions generate salience, which is also presented to consciousness.

The social value module then computes other variables, which are used to determine the variation of *sval* and *oval* and who is responsible for this variation: *svalc* (self value change), *ovalc* (object value change), *sconvalc* (self contribution to changes), *oconvalc* (object contribution to changes). Based on these variables’ values, the emotional module generates an emotion: Fig. 2, right, reports a scheme of the emotions generated. Some emotions are only based on the self value, and not on its variations: *high mood*, for instance, is produced when *sval* is high; *low mood* is produced when *sval* is low. Emotions can be grouped into three poles.

**Pole HP:** emotions characterised by high *sval* and positive *svalc*. *Happiness* is generated when *svalc* is positive. *Satisfaction* is produced when *svalc* is positive and *sconvalc* is positive (in other words: when the self value increases thanks to an action performed by the self). *Gratitude*, on the other hand, is generated when *svalc* is positive and *oconvalc* is positive (in other words: when the self value increases thanks to an action done by the object).

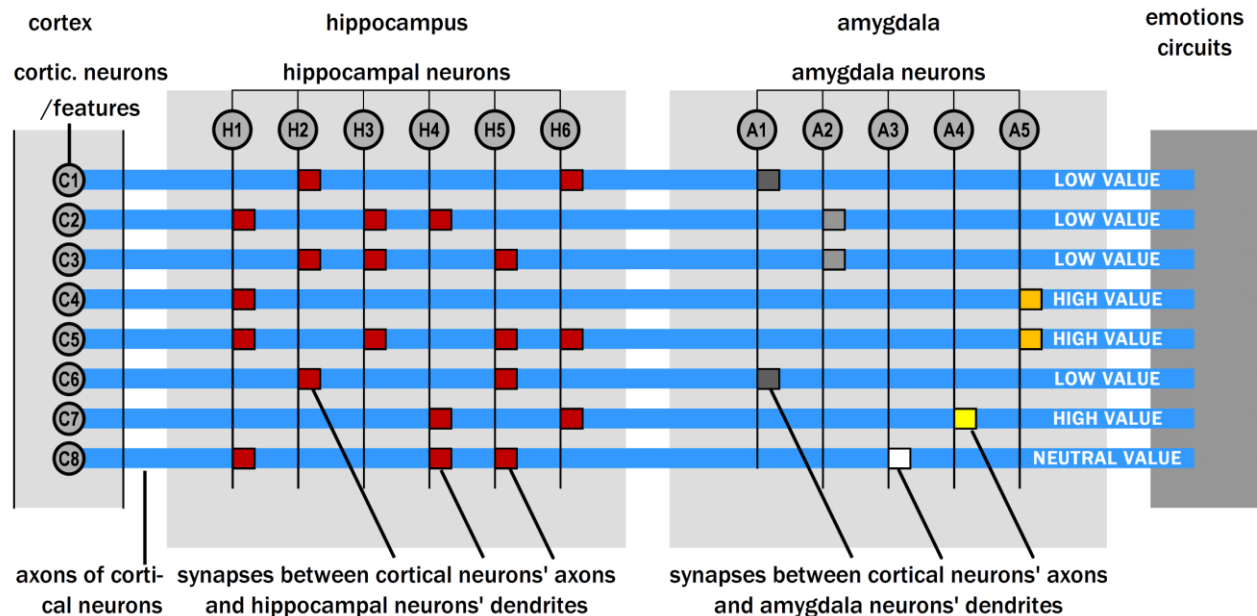
**Pole HN:** emotions characterised by high *sval* and negative *svalc*. *Disappointment* is generated

when *svalc* (self value change) is negative. *Anger* is produced when *svalc* is negative, and *oconvalc* is high (in other words: when the object’s action causes a decrease in self value). Example: the object reveals an embarrassing detail about the self in public, the self value registers a negative variation, for which the object is responsible. The association of anger to a higher social status seems to be well established [7]. *Envy* is generated when *ovalc* is positive (in other words: if the object value increases). It seems that the elicitation of envy requires that *sval* and *oval* be not too dissimilar [8].

**Pole LN:** emotions characterised by low *sval* and negative *svalc*. *Social pain* is generated when *svalc* is negative (in contrast to disappointment, *sval* is low in this case). *Shame* occurs when *svalc* is negative and *sconvalc* is high (in other words: when the self value decreases due to an action performed by the self). Example: the self forgets a line during a public speech, the self value registers a negative variation, for which the self is responsible. A high *oval* may be needed for the elicitation of shame: we do not feel ashamed if our mistakes are witnessed by a person who has a low hierarchical value (e.g. a small child). *Guilt* is generated when *svalc* and *ovalc* are negative and

sconvalc is high (in other words: when the actions of the self-cause a decrease in object value). The reason why also svalc is negative is that harming another person is an action with low social value:

once it is associated with the self, sval decreases. The association of shame, guilt and sadness to a lower social status is confirmed by numerous studies [9].



**Figure 4:** Cortical neurons representing cognitive features send their axons to the hippocampus, where their co-activations are recorded in the synaptic weights between cortical neurons' axons and hippocampal neurons' dendrites. After leaving the hippocampus, the axons reach the amygdala, where the social values of features are encoded in the synaptic weights between cortical neurons' axons and amygdala neurons' dendrites.

### 3. Modules of the mind, neurobiological correlates

The process of emotion generation can be represented through a set of interacting modules (Fig. 3). Cognitive features are activated (through sensory input or memory recall) and sent to consciousness and to the associator where, according to our hypothesis [10], their co-activations, or associations, are recorded. The next processing step is carried out by the social value module, where cognitive features are loaded with value. Based on this information, the emotions module produces an emotion which is presented to consciousness. Emotions generate salience, which is also presented to consciousness.

The scheme proposed contains the implicit assumption that memory records are exclusively composed of cognitive features. Emotions are not recorded in memory, but generated “on-the-fly” based on the set of active features, once such features are activated, through either external input or memory recall. This explains how the emotions associated with an event can change with time (e.g.,

painful memories become less painful as a result of therapy) even though the cognitive trace of the event persists (although even cognitive memories, both traumatic and non-traumatic, can change over time).

Based on current neurobiological knowledge, the processing of perceptual and abstract features occurs in the cortex (in e.g. occipital cortex for visual features), while the executive function is implemented in the prefrontal cortex. The associator corresponds to the hippocampus, a structure essential for the formation of new memories. Social values could be encoded in the amygdala which, according to recent studies [11], would encode “good” and “bad” signals and would be indispensable for understanding social hierarchy. The emotional module could be located in the striatum, the salience module could be implemented by dopamine circuits.

Fig. 4 shows a possible physical implementation of associator and social value module in hippocampus and amygdala respectively, based on the assumption that cognitive features are implemented by single neurons in the cortex



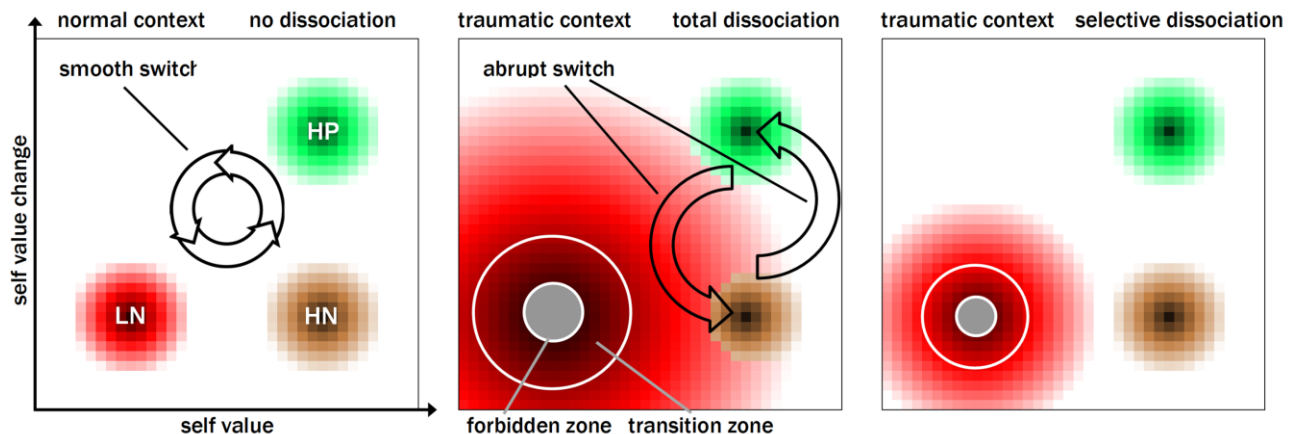
(consistent with experiments that show how neurons exhibit a selective response to very complex features [12]). Cortical neurons send their axons (through the entorhinal cortex, which acts as a connection “hub” [13]) to the hippocampus, where the co-activations (associations) of features are recorded in the synaptic weights between cortical neurons’ axons and hippocampal neurons’ dendrites. After leaving the hippocampus, the paths of these axons continue into the amygdala (located near the hippocampus), where the social values of features are encoded in the synaptic weights between cortical neurons’ axons and amygdala neurons’ dendrites.

In our model, the social emotions elicited are completely determined by the social value of active features. The plasticity of the emotional response resides in the social value module, where the social values of features are stored. These values can be modified through a learning process which, in our model, is mediated by the associator (red arrows in Fig. 3), where features co-activations are stored: from these associations, the social value of existing features can be modified (we recall that social value changes by association). The proposed scheme mainly refers to social emotions: non social emotions may be implemented differently (although even non-social emotions, such as fear, could have a social component).

#### 4. Trauma and dissociation

A *situation* is defined as a set of features simultaneously active. Examples of situations are: “piano lesson with teacher” (co-active features: image of hands on the keyboard, sound of teacher’s voice, sound of piano); “badminton match with a friend” (co-active features: image of racquet, image of opponent, sound of racquet hitting the ball). Situations can be grouped in different contexts, such as, e.g., “parental relation”, “romantic relation”, “relation with schoolmates”.

A context can be conveniently represented on a plane, where the x axis represents the self value and the y axis represents the self value change: the two most important variables for the determination of social emotions. Each point on the plane corresponds to a situation (Fig. 5, left) and belongs to the “zone of influence” of one of the emotional poles defined in section 2. Each pole originates from an “epicentre” representing the most prototypical situation associated with the pole, and extends towards points representing less prototypical situations. The epicentre of the anger pole, for instance, may correspond to a situation-point characterised by an object behaving very dishonestly, eliciting a very intense anger, while points further away may be characterised by a better behaviour of the object.



**Figure 5: Left: normal context.** Each point in a context plane represents a situation and is associated with an emotion (whose intensity is represented by the colour shade). The mind can switch smoothly between emotional poles, no pole requires dissociation and the repertoire of emotions is fully accessible. **Middle: traumatic context.** In a traumatic context, emotional pole LN is characterised by too intense emotional levels and is inhibited. When the mind happens to be around this pole (forbidden zone), disso-ciation intervenes. To avoid dissociation, the mind oscillates between prototypical zones of the permitted poles (HP, HN), staying in each pole as long as the situation remains prototypical. When the mind nears a traumatic pole (transition zone), splitting symptoms appear, and the mind switches to the prototypical zone of another permitted pole. **Right: traumatic context with selective dissociation.** The effect of selective dissociation is a restructuring of the space around the traumatic pole, with an overall reduction of emotional levels.

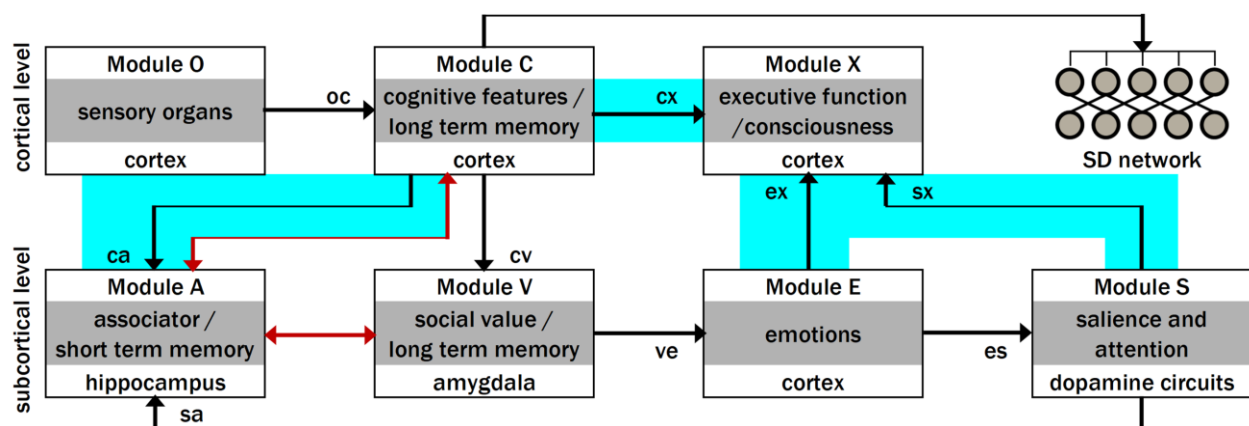
We assume that the only emotional pole that can be involved in a trauma is LN, characterised by low sval and negative svalc, which includes emotions such as shame, guilt and social pain. Emotional poles characterised by high self value, such as happiness, but even anger, cannot become traumatic. We define a context “normal” if the highest emotional levels are not too high (Fig. 5, left). In this condition the mind can switch smoothly between all poles, experiencing different levels of the emotions associated to each pole.

In our definition, a *social trauma* occurs when the intensity of the elicited social emotion is too high and exceeds the tolerance threshold of the mind (this definition is somewhat broader than the one customarily accepted and includes events that would be normally characterised as humiliating experiences). Since social emotions depend on the social value of the active self-associated features, the occurrence of trauma requires that such features have a low value. If a person with big ears, crooked teeth and thin lips thinks these features are bad and gets criticised or mocked for this, a trauma may occur.

The standard response to trauma is represented by the phenomenon of *dissociation*, defined as the

distortion, limitation or loss of the normal associative links between perceptions, emotions, thoughts and behaviour. Dissociation can take the form of mental “black-out”, depersonalisation (feeling of separation from one’s body), derealisation (feeling of being detached from the world), selective amnesia and emotional detachment [14, 15, 16].

In our model, the space around a traumatic pole can be divided into three zones (Fig. 5, right): 1) the “forbidden zone”, an area around the centre in which strong, black-out-like dissociation takes place; 2) the “transition zone”, a “safety belt” around the forbidden zone; 3) the rest of space, where emotional levels are below the mind’s tolerance threshold. In case of trauma, the adoption of dissociation makes it possible for the mind to stay near a traumatic pole, excluding the awareness of intolerable thoughts and emotions from consciousness. However, the disconnection of aspects of reality may entail a high cost, as potential dangers may go undetected. Therefore, the mind will try to avoid traumatic poles and orbit around non-traumatic ones, where the perception of reality is not restricted.



**Figure 6:** Selective dissociation network. This figure shows the modules of Fig. 3 with the addition of the SD (selective dissociation) network. The SD network takes in input cognitive features and sends modulatory signals to cognitive and emotional links (shown in light blue). The effect of modulation can be perceived as a restriction of consciousness.

Let us assume that, in a traumatic context, the mind is initially near the happiness pole (HP, Fig. 5, middle panel). The mind will stay around this pole as long as the conditions are prototypical, i.e. as long as the object relation is “perfect”. As the situation departs from the happiness pole’s prototypical scenario and nears the transition zone of the pain pole (LN), the mind switches abruptly to the prototypical zone of the anger pole (HN). When the situation deviates from the anger pole’s

prototypical scenario, the mind returns to the happiness pole and the cycle repeats itself. This corresponds to the defence mechanism of *splitting*, defined as the inability to integrate positive and negative aspects of self and others, which results in a view of the world in “black and white” [17].

The transition zone is a safety belt built around the forbidden zone, characterised by high emotional levels. This causes an increase of the emotional salience of the features involved in the trauma (link

sx in Fig. 3), which may represent the person's defects criticised. As a result, these features become the focus of attention and appear magnified and distorted: if the feature criticised is "big ears", when looking at the mirror the person will see his/her ears magnified and more protruded, like in a caricature. The magnification of defects serves the purpose of warning the executive function that the current situation is close to a traumatic point, and gives an indication of the features that need to be closely monitored.

This phenomenon is reminiscent of symptoms relevant to a wide range of mental disorders, starting from the hallucinations, the delusions and, more in general, the "delusional atmosphere" that characterise schizophrenia [15]. Transitory perceptual distortions or "pseudo-hallucinations" are not uncommon also in personality disorders [18]. Perceptual distortions are present in body dysmorphic disorder, which can either appear stand-alone [19] or be responsible for the dysmorphic body image associated with eating disorders (among many other conditions) [20].

We hypothesise that, upon the first occurrence of a trauma, dissociation is total (akin to the "freezing" behaviour observed in "disorganised attached" children [21]). In case of repeated traumas, the mind would resort to a *selective dissociation* which, near a traumatic pole, excludes from consciousness only *some* emotional or cognitive channels, to preserve a higher level of functioning. The process that leads to selective dissociation is a form of learning: the mind selects the smallest subset of reality that needs to be excluded from consciousness to avoid unpleasant emotions, without losing touch with the "here and now". In its simplest form, it is obtained by turning down the "volume" of painful emotions, but it can also involve cognitive distortions.

The learning of selective dissociation is obtained through a modulation of the connections between the mind modules. We hypothesise that this is achieved through a dedicated *selective dissociation (SD) network* that takes in input the set of cognitive features and send modulatory signals to emotional and cognitive links (Fig. 6). For each situation, the SD network decides the degree of attenuation of each channel: as a result, different classes of situations are selectively mapped to different attenuation patterns. The effect of modulation translates to a restriction of reality perception.

The effect of selective dissociation is a "restructuring" of the space around traumatic poles, with a reduction of emotional levels and a

corresponding shrinking of the transition and forbidden zones (Fig. 5, right). With selective dissociation, the mind can once again navigate through all emotional poles. The difference between a normal condition and a pathological one lies in the extent of the transition and forbidden zones (in which total dissociation must be used). If these are large, emotion switching can take place but is not fluid: in this case, we still speak of (mitigated) splitting.

## 5. Interpretation of PTSD, C-PTSD and DID PTSD

As recalled in section 1, PTSD is a condition caused by exposure to one or more traumatic events of extraordinary magnitude. Typical PTSD symptoms, which usually appear within days or weeks after the event, include hyperarousal, re-experiencing of traumatic memories (the so-called flashbacks), emotional numbing, helplessness, and a shaking of one's self and perception of the world. The effect of trauma is variable: some people fully recover within a relatively short time, while others go on to develop full-blown PTSD.

Our model foresees that a PTSD-inducing trauma is caused by a too strong "bad" emotion, which catches the mind by surprise, so to speak (it happens for the first time and the mind has no defences in place). Without loss of generality, we can hypothesize that the set of trauma-inducing emotions can be broken down into a non-social and a social component. The non-social part may include emotions such as fear of dying or pain associated with sexual rejection. Fear, in particular, may represent the primary driver of PTSD triggered by war experiences, the first kind of this condition to catch the scientific community's attention.

The social component, on the other hand, includes the emotions belonging to pole C: guilt, shame, pain caused by social rejection (Fig. 2). These emotions are in turn caused by low-value self-associated cognitive features: in the case of simple PTSD, the traumatic landscape is characterised, at least initially, by few very low-value features in few contexts (most contexts are unaffected). Selective dissociation is deployed in traumatic contexts, but is unable to patch up the trauma(s) entirely.

A typical anatomical characteristic of PTSD is represented by structural alterations to the hippocampus [22]. According to our model, selective dissociation is physically implemented through a negative modulation of the connections between brain modules: this also includes the information fed to the hippocampus and presented



to consciousness, through the links *ca* and *cx* shown in Fig. 3 which, based on our model, are the carriers of cognitive features. This leads to a shrinkage of their target areas, which determines a reduction of hippocampal volume.

Another typical PTSD symptom is represented by flashbacks, elicited in situations similar to those in which the trauma occurred. One of the problems, from both a therapeutic and a conceptual viewpoint, is that the degree of similarity is usually very low (e.g. a firecracker may trigger the flashback of a combat scene). To explain why this happens, we introduce a measure of dissimilarity *D* between two situations, defined as the number of features the two situations do not share. For instance, a visual scene with a lemon and one with a shoe are dissimilar, because they have few features in common: one contains a yellow round fruit, the other portrays a non-yellow non-round non-fruit.

The next ingredient we need to factor in is (selective) dissociation, which emerges to protect from painful emotions. If some cognitive features are dissociated, they will not be counted in the *D* formula: as a result, the dissimilarity score between two situations will be lower (the two situations will appear more similar). Therefore, dissociation may contribute to render flashbacks less specific and hence more pervasive. If the awareness of war is dissociated, the sound of bombs is indistinguishable from the sound of firecrackers or fireworks; if the malevolent intention in the abuser's mind is dissociated, the abuser will look like any other person. As a result, a larger set of situations will be feared and avoided.

As we said, the severity and course of PTSD can be very different: this is something that can be accounted for in our model. Let us make an example: a woman is raped. Upon the event's occurrence, a new low value feature (i.e. "being a raped woman") is linked to the person's self, causing a sudden drop in the person's self-esteem. In the weeks and months afterwards, this feature enters in contact with the other self-associated features (all these features are self-associated, hence they also become associated with each other).

As shown in the previous sections, the value propagates between features through association. If the old self-associated features are mostly of high value (e.g. "being a good mother", "being an esteemed university professor"), the value flows from these features to the newly acquired feature, whose value increases, until the trauma is solved. On the other hand, if the old features are mostly of low value (as a result of the person's past experiences), this healing process cannot take place,

low-value features reinforce each other, and the trauma persists.

Since value is relative, some events could be traumatic for some people and not for others. With a thought experiment, we can imagine that, on another planet, being raped is a normal process, a rite of passage marking a woman's transition to adulthood. In this imaginary world, the feature "being raped" would have a high value and the event would not be traumatic (we recall that this analysis is done from the perspective of social emotions: non-social emotions, such as fear, or sensations, such as physical pain, might still cause problems).

### Complex PTSD

Complex PTSD is a mental condition caused by severe and persistent and/or repeated trauma (e.g.: maltreatment, sexual abuse, physical or emotional neglect in childhood, dysfunctional relationships in adulthood, etc.). In contrast to classical PTSD, in C-PTSD traumas usually take place over many years. The outcome is characterised by a broad spectrum of cognitive, affective and psychosocial impairments, which usually have a long-lasting impact on the individual's life.

While in PTSD, according to our model, there are usually few low-value features in the patient's mind, there will be many more in C-PTSD, for two reasons. On the one hand, the low-value ideas directly originating from the traumas (e.g., "being sexually abused by father", "being neglected by mother", "being bullied by schoolmates") get reinforced through repeated exposure, over a long period. This leads to a crystallisation of such ideas, which become part of the person's self-image.

On the other hand, once the original low-value ideas have become steadily associated with the self, they transfer low value to other neutral self-associated ideas such as, e.g., "being a baseball player". This happens when low-value and neutral ideas co-occur in the individual's mind (e.g., when the person remembers being bullied at school while playing baseball). In the long run, this mechanism generates a large set of low-value self-associated ideas, which keep re-occurring, thereby reinforcing each other.

The SD network intervenes and deploys customised dissociation patterns in each context, and this contributes to alleviate the emotional stress, making the situation more tolerable. However, given the breadth of contamination of low-value features in the individual's mind, selective dissociation is unable to patch up the

traumas fully. For all these reasons, complex PTSD is a condition much more stable and difficult to treat, compared to standard PTSD.

### **Dissociative Identity Disorder**

Dissociative Identity Disorder (DID) is characterised by the presence of different personality states (dissociative identities), which take turns in controlling the thinking, feeling and acting of a person. These different personalities have their own characteristics, behaviours, abilities, patterns of perception and thinking. A key DID symptom is the evidence of memory gaps relevant to events or personal information that cannot be explained by ordinary forgetfulness.

DID has many aspects in common with complex PTSD. Both disorders are usually the outcome of prolonged exposure to traumatic events. As a result, many low-value features are present in the person's mind, and many contexts are involved. In both cases the broad presence of low-value ideas is determined by the mechanism of propagation by association: once the set of low-value ideas reaches a critical mass, the contamination becomes difficult to stop. This leads to a point of mental functioning

which is very stable, and whose attraction is difficult to resist.

The SD network reacts by deploying customised dissociation patterns, in a large number of contexts. The distinguishing mark of DID is the peculiar relationship between these patterns. While in complex PTSD the various dissociation patterns have much in common (the subset of non-dissociated cognitive features is large), this set is much smaller in DID. As a result, the continuity of the self is lost, and the different dissociation patterns behave as independent personalities.

### **6. Conclusions**

PTSD, C-PTSD and DID are three conditions caused by exposure to traumatic experiences, which share many characteristics, but whose interdependencies are still poorly understood. The present work described a model which interprets PTSD, C-PTSD and DID with a unique framework, whose distinguishing mark is the interplay between trauma and the defence against trauma, represented by dissociation. Future works will be aimed at deepening the analysis and at integrating further conditions into the interpretation framework.

**References**

1. Cloitre M. et al.. Distinguishing PTSD, Complex PTSD, and Borderline Personality Disorder: a latent class analysis. *European Journal of Psychotraumatology*. 2014.
2. Bozkurt H. et al.. High psychiatric comorbidity in adolescents with dissociative disorders. *Psychiatry and Clinical Neurosciences*. 2015;69:369–374.
3. Langton C.. *Artificial Life*. Addison-Wesley 1989.
4. Fontana A.. Is psychosis caused by defective dissociation? An artificial life model for schizophrenia. *European Journal of Trauma and Dissociation*. 2017;2(1):11–19.
5. Izard C.E.. Emotion theory and research: highlights, unanswered questions, and emerging issues. *Annual Reviews Psychology*. 2009;60:1–25.
6. Marsella S. et al.. Computational models of emotion. in *Blueprint for affective computing*:21–41 2010.
7. Tiedens L.Z.. Anger and advancement versus sadness and subjugation: the effect of negative emotion expressions on social status conferral. *Journal of Personality and Social Psychology*. 2011;80(1):86–94.
8. Noller J.U.. “Ich beneide dich”: das Paradox des Neides. *Academia.edu*. 2020.
9. Stevens A., Price J.. *Evolutionary psychiatry: a new beginning*. New York: Routledge 1996.
10. Fontana A.. A deep learning-inspired model of the hippocampus as storage device of the brain extended dataset. *Arxiv*. 2017;1706.05932.
11. Bzdok D. et al.. An investigation of the structural, connectional and functional subspecialization in the human amygdala. *Human Brain Mapping*. 2013;34(12):3247–3266.
12. Quiroga R.Q. et al.. Invariant visual representation by single neurons in the human brain. *Nature*. 2005;435(7045):1102–1107.
13. Canto C.B. et al.. What does the anatomical organization of the entorhinal cortex tell us? *Neural Plasticity*. 2008;381243:635–704.
14. Lanius R.A.. Trauma-related dissociation and altered states of consciousness: a call for clinical, treatment, and neuroscience research. *European Journal of Psychotraumatology*. 2015;6.
15. Moskowitz A., Corstens D.. Auditory hallucinations: psychotic symptom or dissociative experience? *Journal of Psychological Trauma*. 2008;6:35–63.
16. Nijenhuis E.R., Hart O.. Defining dissociation in trauma. *Journal of Trauma and Dissociation*. 2011;12(4):464–473.
17. Perry J.C. et al.. Defense mechanisms in schizotypal, borderline, antisocial, and narcissistic personality disorders. *Psychiatry Interpersonal and Biological Processes*. 2013;76(1):32–52.
18. Gras A. et al.. Hallucinations and borderline personality disorder: a review. *Encephale*. 2014;40(6):431–438.
19. Phillips K.A.. Body dysmorphic disorder: recognizing and treating imagined ugliness. *World Psychiatry*. 2004;3(1):12–17.
20. Ruffolo J. et al.. Comorbidity of body dysmorphic disorder and eating disorders: severity of psychopathology and body image disturbance. *The International Journal of Eating Disorders*. 2006;39(1):11–19.
21. Main M., Solomon J.. Discovery of an insecure disoriented attachment pattern: procedures, findings and implications for the classification of behavior. *Affective development in infancy*. 1986.
22. Bremner J.D.. Traumatic stress: effects on the brain. *Dialogues in Clinical Neuroscience*. 2006;8(4):445–461.