[Encephale.](https://www.ncbi.nlm.nih.gov/pubmed/23062450%22%20%5Co%20%22L%27Encephale.) 2012 Oct;38(5):373-80. doi: 10.1016/j.encep.2011.12.003. Epub 2012 Jan 24.

**[Posttraumatic stress disorder (PTSD) as a consequence of the interaction between an individual genetic susceptibility, a traumatogenic event and a social context].**

[Article in French]

[Auxéméry Y](https://www.ncbi.nlm.nih.gov/pubmed/?term=Aux%C3%A9m%C3%A9ry%20Y%5BAuthor%5D&cauthor=true&cauthor_uid=23062450)1.

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**Abstract**

**INTRODUCTION:**

Why are some individuals more likely than others to develop a posttraumatic stress disorder (PTSD) in the face of similar levels of trauma exposure? Monitoring the traumatic process combining the antecedents, the determinants of the psychic trauma and the acute symptoms can clarify the causes of the final onset of a chronic repetition syndrome. Epidemiologic research has clarified risk factors that increase the likelihood of PTSD after exposure to a potentially traumatic event. PTSD is an interaction between a subject, a traumatogenic factor and a social context. With each epidemiological, psychopathological and more particularly neurogenetic study, we will expand on the impact of these interactions on the therapeutic treatment of psycho-traumatised persons.

**LITERATURE FINDINGS:**

Most studies have shown that unrelated to the traumatic event, additional risk factors for developing PTSD include younger age at the time of the trauma, female gender, lower social economic statuts, lack of social support, premorbid personality characteristics and preexisting anxiety or depressive disorders increase the risk of PTSD. The psychic trauma is firmly attached to the repetition and the previous traumas are as many risks of developing a subsequent PTSD in the wake of a new trauma: PTSD in adults may represent a prolonged symptomatic reaction to prior traumatic assault, child abuse and childhood adversities. Related to the traumatic event, the organic pain, the traumatic brain injury, but also the sight of blood can lead to a trauma being considered as more serious or more harmful to life. It is useful to recognize the acute reactions of exhaustion stress as they can guide both the pharmacotherapeutic and the psychotherapeutic treatment thanks to debriefings. Even though the majority of people with acute stress disorder subsequently develop PTSD, the current data indicate that too many people can develop PTSD without initially displaying acute stress disorder. Though peritraumatic dissociation and peritraumatic distress have emerged as the strongest predictors for PTSD and have to be treated as soon as possible with the debriefing or the pharmacology; initial evidence suggests the potential benefits of early intervention, shortly after the trauma, and psychological debriefing has received increasing interest from the scientific community. However the Anglo-Saxon techniques (such as Critical Incident Stress Debriefing also known as the Mitchell model) are in total contrast with the French approach. In the first case the emotional response is controlled to ensure the pursuit of the group action, whilst in the second case the debriefing concerns patients with acute symptoms in order to prevent the development of a PTSD structuring of the latter. The facts, emotions and thoughts are not partitioned but inter-linked, thus enabling a fragmentation of the traumatic experience. In the face of the annihilation experienced, speech production by the subject is restored linking the person to the human community, once abandoned. However, debate continues on the efficacy of single session debriefing in the prevention of PTSD. At the time of the acute stress reactions, benzodiazepines are contraindicated at this stage as they promote dissociation and ulterior revivals. On the other hand, treatment with propranolol could be proposed: a two or three week course of propranolol begun in the aftermath of a traumatic event can reduce subsequent PTSD symptoms.

**DISCUSSION:**

A genetic polymorphism is evidently at work in the development of a PTSD via the regulation of the expression of genes of interest to the serotoninergic system and the adrenocorticotropic axis. The 5-HTTLPR (promoter region of SLC6A4 witch encodes the serotonin transporter) constitutes a genetic candidate region that may modulate emotional responses to traumatic events. The interaction between variation at the 5HTTLPR and stressful life events could predict depression and PTSD. Considering the dopaminergic pathway, the A1 allele coding the type 2 dopaminergic receptor is associated with a severe comorbidity of PTSD with the presence of somatic disorders, anxiety, social change and depression. For noradrenergic neuromodulation, an interaction between the polymorphism of gene GABRA2 and the occurrence of PTSD is described whereas an interaction between the number of traumatic events and Val(158)Met polymorphism of the gene coding for catecholamine-o-methyltransferase has also been found. The role of polymorphisms in FKBP5 (a co-chaperone of hsp 90 which binds to the glucocorticoid receptor) in predicting PTSD too, with a gene-by-environment point of view. These gene-by-environment studies are needed to focus more on distinct endophenotypes and influences from environmental factors. If several candidate genes are involved, a weighting of susceptibility to such and such a neurological regulation system will imply various endophenotypes. According to the monoamine predominantly incriminated, PTSD can take on a more hyper-vegetative clinical expression linked with noradrenergic overuse. Differently, avoidance behaviour and the depressive aspect invoke more a modification of the serotoninergic modulation whilst posttraumatic psychotic reactions question the role of dopaminergic pathways. Neuroscientific discoveries interesting the biological support of PTSD can thus modify our view of the conception of the disorder in relation to different therapeutic prospects.

**CONCLUSION:**

Chronic PTSD can manifest itself in different clinical forms. The repetition syndrome can appear a long time after the traumatic event, following a paucisymptomatic latency period, which can last several years or even decades. The absence of complaints from the patient is common, the latter suffering in silence. Often other comorbid disorders and other complaints arise sooner than the clinical picture. Thus a depressive episode characterised as drug-seeking behaviour is frequently encountered. The therapeutic accompaniment traditionally combines a pharmacological and a psychotherapeutic treatment even if recommendations are rare. A posttraumatic stress disorder is never just a coincidence. The different stages of the evolution and the establishment of a PTSD are the expression of an interaction between the outside and the inner self. Despite a known progression of the posttraumatic stress disorder, this deleterious evolution is far from being a foregone conclusion. On the contrary, several levels of prevention are possible at each stage of its structuration to propose treatments to subjects who are vulnerable and/or present symptoms. No neurobiological study has yet found a biological marker, which would apparently and inevitably destine a subject to structure, a posttraumatic stress disorder in reaction to a stress. Conversely, the psychopathological study finds afterwards that a particular subject has necessarily built a traumatic repetition syndrome according to the concordance of significant data relative to his/her history. The event strikes a repression or an anterior biographical deadlock and of which the thematic questions the fundamentals of human culture in its emancipation with nature, like the question of death and its consequences: bereavement, parentality, transgenerational transmission and organicity often linked to the illness. A therapeutic proposal constitutes an environmental factor par excellence which can be either protective or deleterious. If the traumatic repetition syndrome has been known since Antiquity, the birth of PTSD has followed the chronology of the DSM according to the sociopolitical contexts encountered. A PTSD does not occur by chance: the conditions of possibility of the trauma are established by genetic and psychological determinants interactively integrated at the heart of a social context. After the increase in a psychotraumatic interest in international publications since the 1980s, a fight against over-victimisation seems to be setting in. The advances in genetic and neuroimaging techniques are in the process of superseding psychometric studies in terms of reliability and validity; maybe we should see in this social evolution the changes of tomorrow concerning the clinical of PTSD and its treatment. The healing of the psycho-traumatised subject cannot just be established on the passive status of victim, which would be detrimental to reflection and ultimately reconstruction: the rebirth of the subject will require active commitment, which could distract from the deadly repetition. Whilst the confrontation with death resembled nonsense, the subject will question the psychotraumatic determinants of his/her life history to reinstate this tragic event within a search for meaning. Such restructuring is built on the intersubjectivity of the clinical relationship, which occurs within a social context. PTSD is a pathology which interacts with the societal context: on the one hand the trauma is established on the brutal reconsideration of social values which seem immutable and on the other hand, the clinical and nosographical concept of PTSD is changing with the evolution of society.

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