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This module and its multiple choice questions are available online on the website of the Irish Journal of Psychological Medicine (www.ijpm.org). The CPD policy of the College of Psychiatry of Ireland indicates that psychiatrists who participate in suitable online learning which fits the criteria for CPD may claim CPD points under the Personal CPD category (up to a maximum of 5 points per year).

We are confident that this CPD Section of the Irish Journal of Psychological Medicine will prove to be a valuable resource for consultant psychiatrists, psychiatric trainees and all journal readers. We welcome feedback from readers and, especially, any suggestions for topics to be covered in future CPD modules. Suggestions should be emailed to: psychological@medmedia.ie

Post-traumatic stress disorder: present and future

Niall Crumlish

Post-traumatic stress disorder (PTSD) and acute stress disorder (ASD) differ from almost every other psychiatric diagnosis in that they may only be diagnosed with reference to an aetiological event – an external traumatic stressor. ASD occurs immediately after the stressor and is comparatively short-lived, while PTSD is a prolonged abnormal response that may take months to develop. The types of stressor leading to ASD and PTSD are identical and were intended to be tightly defined, involving a perceived threat of death, serious injury or loss of physical integrity.[1]

It is useful initially to distinguish ASD and PTSD from adjustment disorders, which are also diagnosed only after an observable life event. An adjustment disorder may be thought of as a gradual and prolonged response to stressful changes in a person's life.[2] The range of stressors precipitating an adjustment disorder is potentially much broader than that precipitating ASD or PTSD, as a threat of death or injury is not needed.

Indeed, a 'threat' as such is not needed, as the event may be a loss. Events such as job loss or the breakup of a relationship may lead to an adjustment disorder, as well as threats such as accidents or assaults. The diagnostic criteria for adjustment disorder do not specify what the immediate response, if any, to the precipitating stressor must be.[1]

PTSD criterion A1 – the traumatic stressor

According to the DSM-IV, the stressor leading to PTSD or ASD may be (1) experienced directly, (2) witnessed, or (3) experienced by others and subsequently learned about. The stressor (criterion A1) must be responded to with intense fear, helplessness or horror (A2).

Events that are experienced directly include military combat, violent personal assault, being kidnapped, being taken hostage, or being tortured. Witnessed events include the serious injury or unnatural death of another person, or unexpectedly seeing a dead body or body parts. Events that are learned about include violent assault, serious accident, or serious injury experienced by a family member or a close friend, or the sudden, unexpected death of a family member or a close friend.[1]

The diagnostic criteria for ASD and PTSD are in *Tables 1* and *2*. Recently, the chair of the task force revising PTSD for the DSM-V noted that the most controversial aspect of the DSM-IV traumatic stressor criterion was that learning about a trauma rather than experiencing it at first hand could itself count as a criterion A1.[3] Many clinicians and researchers felt that including vicariously experienced events as stressors overly broadened the range of experiences potentially resulting in PTSD: "to qualify as a trauma survivor, one need only respond with fright to learning about the misfortunes of others".[4] Nonetheless, there are no plans to remove the 'learned about' element of the stressor criterion from the DSM-V.[3,5]

Origins and inclusion in DSM-III

Syndromes similar to PTSD, occurring in the context of combat, have been recognised over the last two centuries[6] and it is often argued that PTSD was included in 1980 because of pressure by veterans' associations to acknowledge the experiences of American soldiers in the Vietnam War.[4,7] PTSD was

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added to the Diagnostic and Statistical Manual of Mental Disorders in its third edition, the DSM-III, so that it has existed as a discrete disorder for just 30 years.[8] Prior to this, PTSD was included in the ninth addition of the International Classification of Diseases (ICD-9), but it was included as a subtype of adjustment disorder rather than as a separate diagnostic entity.[9] In the DSM-III conceptualisation of PTSD, the precipitating trauma did not have to be war-related, but had to be "outside the range of usual human experience".[8]

Neurobiology of PTSD

There are several replicated neuroimaging findings in PTSD. Probably most consistently, bilateral hippocampal volume in adults, but not children, is reduced. This suggests an initially normal hippocampus, which develops abnormally only after trauma exposure.[10-12] Other brain areas implicated include the amygdala and the anterior cingulate cortex.[10,12,13] There is evidence that stress-induced glucocorticoid release can cause structural and functional damage to the hippocampus.[14]

PTSD can be considered a disorder of memory and emotion processing, so that disabling levels of fear are associated with activation of a memory;[15] the brain areas implicated in the aetiology of PTSD, particularly the amygdala and hippocampus, are consistent with this theory. Increased activity in the amygdala in PTSD indicates that it is hyper-responsive to traumatic memories.[16]

A potential mechanism for this is that noradrenergic overactivity in the amygdala causes overconsolidation of fear memories.[17] The hippocampus is involved in memory and stress regulation,[10] and it has been proposed that a hippocampus damaged by glucocorticoid release[14] cannot dampen the amygdala's exaggerated response to traumatic memory in PTSD.[18]

Epidemiology and comorbidity

In the United States, the National Comorbidity Survey (NCS) reported a lifetime prevalence of 7.8% for DSM-III-R PTSD.[19] Perhaps surprisingly, the NCS replication (NCS-R) reported a lower lifetime prevalence of 6.8%,[20] despite the broadening of diagnostic criteria that occurred in the DSM-IV.[21] In the NCS-R, one-year prevalence was 3.5%.

The European Study on the Epidemiology of Mental Disorders (ESEMeD) reported a lower lifetime prevalence of 1.9%; 0.9% among men and 2.9% among women.[22] The authors considered only briefly the reasons for the low prevalence in Europe relative to the US, speculating that their use of a new version of the Composite International Diagnostic Interview (CIDI) may have led to a lower false positive rate than in the NCS-R. This seems an inadequate explanation for a difference of five percentage points and a 3.5-fold disparity in prevalences.[20,22]

Internationally, a systematic review reported lifetime prevalences ranging from 0.18% in Italy to 11.2% in Mexico. The authors reported a 'best estimate' of an international prevalence of PTSD of 2.1%,[23] which was closer to the ESEMeD than to the NCS-R. An epidemiological survey in Iraq reported a lifetime prevalence in the general population of 2.5%.[24] The lifetime prevalence among resident Iraqis contrasts with a reported prevalence of 43.9% among Iraqi refugees who migrated to the United States after the first Gulf War.[25]

Refugees are known to experience high levels of PTSD and other mental disorders.[26] A Lancet meta-analysis reported a lifetime PTSD prevalence among refugees worldwide of 9%, or 10 times that of the general population in the refugees' destination countries.[27] PTSD may be even more common among those seeking refugee status than among those who have acquired it.[28]

Table 1: DSM-IV diagnostic criteria for ASD (APA 1994)

Criterion	Description
A	The person has been exposed to a traumatic event in which both of the following were present: (1) The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others; (2) The person's response involved intense fear, helplessness, or horror
B	Either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following dissociative symptoms: (1) A subjective sense of numbing, detachment, or absence of emotional responsiveness; (2) A reduction in awareness of his or her surroundings (eg. 'being in a daze'); (3) Derealization; (4) Depersonalization; (5) Dissociative amnesia (ie. inability to recall an important aspect of the trauma)
C	The traumatic event is persistently reexperienced in at least one of the following ways: recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience; or distress on exposure to reminders of the traumatic event
D	Marked avoidance of stimuli that arouse recollections of the trauma (eg. thoughts, feelings, conversations, activities, places, people)
E	Marked symptoms of anxiety or increased arousal (eg. difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness)
F	The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or impairs the individual's ability to pursue some necessary task, such as obtaining necessary assistance or mobilizing personal resources by telling family members about the traumatic experience
G	The disturbance lasts for a minimum of two days and a maximum of four weeks and occurs within four weeks of the traumatic event
H	The disturbance is not due to the direct physiological effects of a substance (eg. a drug of abuse, a medication) or a general medical condition, is not better accounted for by Brief Psychotic Disorder, and is not merely an exacerbation of a preexisting Axis I or Axis II disorder

Most people who experience a traumatic stressor do not develop PTSD. A Dutch population-based study found the lifetime prevalence of traumatic stressors to be as high as 80.7%, while the lifetime prevalence of PTSD was 7.4%. In this study, predictors of PTSD after trauma were younger age, female sex, divorced or widowed marital status, non-Dutch cultural background, and unemployment.[29]

Other vulnerability factors, according to a widely cited meta-analysis[30] include younger age, low socioeconomic status, lack of education, childhood abuse or other previous trauma, and a family psychiatric history; specifically, pretrauma depression, according to Resnick and colleagues.[31]

A study of firefighters found that personality factors – high hostility and low self-efficacy – predicted almost half the variance in PTSD symptoms after a stressor.[32] That study found that biological parameters did not predict severity of PTSD, but others have found differently. For instance, the emergence

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of psychopathology after trauma is associated with smaller hippocampal volume[33] and possession of the short variant of the serotonin transporter gene (5-HTT).[34]

PTSD is characterised by frequent comorbidity. In the NCS-R,[20] over 80% of respondents with PTSD had at least one other Axis I disorder. The most frequently reported comorbidity in the NCS was with major depressive disorder (48%), while dysthymia, alcohol and sub-stance abuse disorders and other anxiety disorders were also common, present in 10-40% of cases.[19]

The relationship between ASD and PTSD

ASD was introduced to the DSM classification system only with the publication of the DSM-IV.[1] The purpose of including ASD was so that post-traumatic symptoms would be recognised before the one-month duration of disturbance required for PTSD, and so that people at high risk of developing PTSD would be identified early.[35] Essentially, after one month of persistent characteristic post-traumatic symptoms, an individual's diagnosis changes from ASD to PTSD in a manner analogous to the evolution from schizophreniform disorder to schizophrenia after six months of symptoms. The symptoms of ASD and PTSD are not identical, but they are divided into the same three groups – hyperarousal, re-experiencing, and avoidance – see *Tables 1* and *2*.[1]

ASD is sometimes described as a precursor of PTSD.[36] However, PTSD may be diagnosed in the absence of a pre-existing ASD, as ASD requires that symptoms emerge within a month of the stressor; if symptoms emerge more than a month afterwards, the diagnosis is PTSD.[1] A study by a leading Australian PTSD group assessed 597 victims of major trauma for ASD, finding a point prevalence of 6% (33 individuals), and reassessed 507 of the cohort at three months, identifying 49 cases of PTSD (7%). Of the 49 PTSD cases, 34 had not been diagnosed with ASD at the first assessment.

Equally, ASD need not necessarily evolve into PTSD as it may remit. In the same study, only 15 (45%) of those diagnosed with ASD population (n = 33) met criteria for PTSD at three months. The positive predictive value of ASD for PTSD was low.[37] The same group reported evolution to PTSD after two years among 63% of patients with full ASD and 70% of people with subsyndromal ASD, ie. those who missed on an ASD diagnosis by one criterion.[38] Studies among children have found subsyndromal ASD to predict PTSD as well as full ASD.[39]

The consensus appears to be that the dissociative symptom cluster in ASD has high specificity for predicting PTSD, but the false negative rate is high, resulting in low sensitivity and suggesting that ASD is an inadequate screen for PTSD.[40]

A review of early predictors of PTSD was unable to be definitive about helpful strategies for early detection, reporting that the relationship between early responses to trauma and later PTSD was not linear and that more accurate prediction would require a focus on "the interaction between symptoms, biological responses and cognitive factors".[37] This review underlined the complexity of the task of predicting PTSD, and indeed the complexity of the PTSD construct itself.

Of note, one prolific author on PTSD has noted that the effect size for almost every risk factor for PTSD, including ASD, is small. This makes it difficult to predict progression to PTSD after trauma based on any pretrauma characteristic, trauma severity, or reactions at the time of the trauma. This author recommended predicting later PTSD by evaluating early symptoms three to four weeks after the trauma.[41]

Table 2: DSM-IV diagnostic criteria for PTSD (APA 1994)

Criterion	Description
A	The person has been exposed to a traumatic event in which both of the following were present: (1) The person experienced, witnessed or was confronted by an event that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others; (2) The person's response involved intense fear, helplessness or horror
B	The traumatic event is persistently re-experienced in one (or more) of the following ways: (1) Recurrent or intrusive recollections; (2) Recurrent distressing dreams; (3) Acting or feeling as if the traumatic event were recurring, through reliving, illusions, hallucinations, or dissociative flashback episodes; (4) Intense psychological distress at exposure to cues that symbolise or resemble an aspect of the traumatic event; (5) Physiological reactivity on exposure to such internal or external cues
C	Persistence avoidance of stimuli associated with the trauma and numbing of general responsiveness, as indicated by three (or more) of the following: (1) Efforts to avoid thoughts, feelings, or conversations associated with the trauma; (2) Efforts to avoid activities, places or people that arouse recollections of the trauma; (3) Inability to recall an important aspect of the trauma; (4) Markedly diminished interest or participation in significant activities; (5) Feeling of detachment or estrangement from significant others; (6) Restricted range of affect; (7) Sense of a foreshortened future
D	Persistent symptoms of increased arousal, as indicated by two (or more) of the following: (1) difficulty falling or staying asleep; (2) Irritability or outbursts of anger; (3) Difficulty concentrating; (4) Hypervigilance; (5) Exaggerated startle response
E	Duration of the disturbance (criteria B, C, D) is more than one month
F	The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning

Treatment of PTSD

The first line treatment of PTSD, according to the National Institute for Clinical Excellence (NICE), is psychological therapy. Trauma-focused CBT (TF-CBT) and eye movement desensitization and reprocessing (EMDR) were the treatments of choice in these guidelines.[42]

Bisson and Andrew[43] conducted a Cochrane review of psychological treatments, and found sufficient evidence to support the use of individual or group TF-CBT, EMDR, and stress management over wait-list/treatment as usual. A meta-analysis of psychological treatments for 'chronic' PTSD (symptoms present for longer than three months) supported the use of TF-CBT, EMDR, stress management and group CBT.[44]

A Cochrane review of pharmacotherapy in PTSD found evidence for efficacy for antidepressants.[45]

Evidence for individual therapies

Trauma-focused CBT (TF-CBT)

An influential paper from a decade ago described a cognitive model of PTSD.[46] A key insight in this paper was that people with PTSD after an event were unable to see the trauma as a discrete, time-limited event that by now was in the past. Those

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with PTSD appraised the traumatic event so as to create a sense of 'serious current threat', and may overgeneralise from the traumatic event to arrive at core beliefs such as 'nowhere is safe', or 'I attract disaster'.[46]

CBT for PTSD should incorporate these concepts and include an element of cognitive restructuring as well as exposure to traumatic memories. The patient should identify cognitive distortions relating to the trauma and challenge negative automatic thoughts or assumptions using evidence.[44] Evidence for TF-CBT comes principally from the two meta-analyses mentioned above.[43,44] Age-appropriate TF-CBT may be offered to children, including those who have experienced sexual abuse.[42]

Three Irish trials of CBT for PTSD symptoms exist. The highest quality trial is an RCT comparing immediate cognitive therapy to a wait-list control group for trauma related to terrorism and other conflict. The trial (n = 58) was conducted by the Northern Ireland Centre for Trauma and Transformation in Omagh, and found significant advantages for cognitive therapy over wait-list with respect to PTSD symptoms, depression, and functioning.[47] The same group previously published an uncontrolled trial of cognitive therapy for survivors of the Omagh bombing.[48] A novel open trial (n = 14) from Cork reported preliminary evidence for the use of driving simulators as a form of exposure therapy in driving phobia, with or without PTSD, after serious RTAs.[49]

Eye movement desensitization and reprocessing (EMDR)

EMDR is among the least understood effective treatments in psychiatry. According to Shapiro, who devised it,[50] EMDR involves the therapist eliciting from the patient sequences of rhythmic saccadic eye movements while the patient concentrates on their traumatic memory. Shapiro hypothesised that the eye movements of EMDR mimicked REM sleep and thus aided processing of the traumatic memory, which was impaired in PTSD.[50]

This mooted mechanism of action was supported by a recent paper, which proposed that EMDR induces a state similar to REM sleep that facilitates the integration of traumatic memories into semantic memory, resulting in a reduction in amygdala-mediated negative affect associated with the memories. The authors of this paper noted that EMDR may aid memory processing more effectively than REM sleep, because thought content and affect are more controlled in EMDR than in REM sleep.[51]

Empirically, EMDR was supported in the Cochrane review of psychological treatments, although there were fewer and smaller trials of EMDR than of CBT.[43] A meta-analytic comparison of EMDR and TF-CBT showed that each was equally effective.[52] NICE recommends that all people with PTSD should be offered individual, outpatient TF-CBT or EMDR.[42] No Irish trials of EMDR exist.

Other psychotherapies

Evidence exists for stress management[43] and variants of behavioural therapy, including pro-longed exposure therapy,[53] stress inoculation training,[54] narrative cognitive therapy[55] and narrative exposure therapy (NET).[15] NET was developed for PTSD among victims of organised violence, and is the best supported treatment for PTSD among refugees.[56] A search for psychosocial treatments revealed an enticing Cochrane review of sports and games (including computer and card games) for PTSD; unfortunately, no trials meeting the review's inclusion criteria actually existed, leading to the inevitable conclusion that more research was needed.[57] There is little empirical support for psychodynamic psychotherapies.[43]

Psychological treatment of ASD

Although not mentioning ASD specifically, the NICE guidelines for treatment of PTSD recommend that those with severe post-traumatic symptoms should be offered TF-CBT in the month after the traumatic event; at this point, the diagnosis remains ASD.[42] CBT has been shown to be more effective than supportive counselling (SC) for ASD in the first two weeks after trauma, with 8% (n = 2) of the CBT group going on to develop PTSD compared to 83% (n = 8) of the SC group.[58] A recent meta-analysis supported TF-CBT in the reduction of acute symptoms and prevention of PTSD, with the caveats that few high quality studies exist, and follow-up is too brief.[59]

The correct course of action when an individual has experienced a trauma but has no more than mild symptoms is to initiate watchful waiting and arrange follow-up within one month.[42] The WHO recommends psychological first-aid, which includes non-intrusive emotional support, provision of basic necessities, protection from further trauma and organisation of ongoing support networks.[60] Once-off debriefing should not be routinely offered, and certainly not insisted upon, as it is not only unhelpful but may increase trauma among susceptible individuals.[61]

Pharmacotherapy

The NICE guidelines recommend that medication be considered not as a first line but only when a patient has expressed a preference for drug over psychological treatments. NICE noted that noted that the evidence base for drug therapy was 'very limited' and on that basis recommended only paroxetine and mirtazapine for general use, with phenelzine and amitryptyline reserved for prescription by specialists.[42]

The Cochrane review of drug treatment in PTSD aggregated the results of 35 trials (n = 4,597), reporting a medication response rate of 59.1% and a placebo response rate of 38.5%, resulting in a number needed to treat (NNT) of 4.9 (95% CI = 3.9-6.3). The authors suggested that evidence was most convincing for SSRIs, largely because more trials had been conducted. Medication was effective in ameliorating comorbid depression as well as core PTSD symptoms.[45] The review found no evidence of antipsychotic efficacy, though other authors have found in support of atypical antipsychotics as second line agents.[62] Quetiapine and olanzapine may be of use in the management of sleep disturbance, including nightmares, in PTSD.[63,64] Drugs that dampen noradrenergic overactivity, such as clonidine, prazosin and propranolol, have been the subject of considerable speculation, if less empirical evaluation, as potential treatments.[65] No high quality RCTs of anti-adrenergic drugs exist, but a direct retrospective comparison of quetiapine and prazosin among veterans favoured prazosin over quetiapine.[66] Lastly, some antiepileptic or mood stabilising drugs, including lamotrigine, have limited trial evidence as second line agents.[67]

Novel and experimental treatments

This topic was comprehensively reviewed last year, and the interested reader is directed to Cukor and colleagues.[68] One noteworthy treatment modality under investigation is psycho-therapy assisted by MDMA, which theoretically assists patients in processing memories of traumatic events without the associated negative affect.[69]

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D-cycloserine, an antibiotic with a partial NMDA receptor agonist effect, has therapeutic potential in conjunction with exposure therapy, as NMDA receptor antagonism can facilitate extinction of learned fear; preliminary trials in other anxiety disorders have been encouraging.[70]

Psychotherapies that are being developed or adapted for the treatment of PTSD include mindfulness-based therapy,[71] interpersonal psychotherapy[72] and imagery rescripting (IR).[73] IR is a technique aimed specifically at child abuse survivors with roots in prolonged exposure therapy. A key difference between IR and imaginal exposure is that in IR, patients are encouraged to remember their childhood traumas and also to imagine themselves as an adult entering the scene of the trauma and rescuing the child.[68] IR has been used for PTSD following other traumas, such as industrial accidents,[74] but evidence for efficacy over exposure alone is awaited.

Current controversies: PTSD at a crossroads

PTSD has existed as a separate diagnostic entity for just 30 years.[8] There were some who opposed the inclusion of PTSD in the DSM-III, arguing that the distress experienced by traumatised people was already covered by existing diagnoses, so that PTSD simply combined a variety of symptoms from multiple comorbid disorders and attributed them, possibly wrongly, to a specific event.[4]

Objections to the diagnosis of PTSD have not gone away, with recent authors continuing to challenge the assumptions underlying the diagnosis – eg. that the stressor criteria are directly related to the symptom criteria, and that characteristics of the stressful events are the key determinants of morbidity, not individual vulnerability.[75] Despite the volume of research in the last three decades on the neurobiology, phenomenology and treatment of PTSD, there are ongoing, vehement and even growing challenges to the validity of the PTSD construct itself.[7,76]

It should be noted the proposed revision of diagnostic criteria for the DSM-V does not indicate any fundamental alterations to the diagnosis, other, arguably, than the removal of criterion A2, removing the requirement for "fear, helplessness or horror".[5] This apparent further broadening of the diagnosis will likely provoke intense debate, so plus ça change: PTSD has incited scrutiny, research and controversy for more than 30 years, and no doubt will do so well into the future.

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Multiple Choice Questions

1. Regarding PTSD:

PTSD may only be diagnosed with reference to an external traumatic stressor	T	F
PTSD is usually very short lived	T	F
Once off debriefing after a traumatic event should be routinely offered	T	F
PTSD has existed as a discrete disorder for more than 50 years	T	F
To qualify as PTSD the stressor must be responded to with intense fear, helplessness or horror	T	F

2. Neurobiology of PTSD:

Bilateral hippocampal volume is reduced in children but not in adults	T	F
Other areas of the brain implicated include the amygdala and anterior singulate cortex	T	F
PTSD associated hippocampal volume suggests a normal hippocampus which develops normally after trauma exposure	T	F
PTSD can be considered a disorder of memory and emotion processing	T	F
A hippocampus damaged by glucorticoid release cannot dampen the amygdala's exaggerated response to traumatic memory	T	F

3. Epidemiology and comorbidity of PTSD:

Studies have shown equal incidence rates in Europe and the US	T	F
Acute stress disorder is an excellent predictor of PTSD	T	F
Age has been shown to have no effect on incidence rates	T	F
Most people who experience a traumatic stressor develop PTSD	T	F
The most frequently reported comorbidity has been major depressive disorder	T	F

4. Treatment for PTSD:

First line treatment for PTSD is psychological therapy	T	F
Trauma-focused CBT is the only therapy recommended for PTSD	T	F
Age appropriate trauma-focused CBT may be offered to children, including those who have experienced sexual abuse	T	F
Eye movement desensitisation and reprocessing works by inducing a state similar to REM sleep which aids memory processing	T	F
Narrative exposure therapy is particularly suited to refugees with PTSD	T	F

5. Pharmacotherapy and novel treatments:

Medication should only be considered when a patient expresses a preference for it over psychological treatments	T	F
There is evidence for the efficacy of antidepressants in PTSD	T	F
Psychotherapy assisted by MDMA has been proposed as a treatment enabling memory processing without associated negative effect	T	F
Exposure therapy combined with D-cycloserine is proposed to facilitate extinction of learned fear	T	F
Imagery rescripting (IR) has a proven track record for traumas such as industrial accidents.	T	F

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