

CATHOLIC UNIVERSITY OF RWANDA
FACULTY OF SOCIAL WORK
DEPARTMENTS OF WSD AND CFS

MENTAL HEALTH ISSUES AND POST TRAUMATIC STRESS DISORDER

Course Notes for Students

Compiled by Emmanuel HAKIZIMANA

Bachelor of Clinical Psychology

Master of Social Work and Community Development

PhD Candidate

2015

Course Description

Class: level II

Departments: Welfare and Social Development & Child and Family Studies

Subdivisions:

Part I. Part I. Introduction to Global Mental Health: Effects of Mental Health on Individuals and Populations

Part II. Genocide

Part III. Post-traumatic stress disorder

Assignment Pattern:

60% CAT (Written and oral, Individual and Group Works)

40% Final exam

Additional Notes:

List of mental health illnesses

20th Century atrocious genocide

Course weight: 10 credits

Face-to-face: 50 hours

Table of Contents

Course Description	2
Table of Contents	3
Part I. Introduction to Global Mental Health: Effects of Mental Health on Individuals and Populations.....	5
1.1. Effects of Psychological Disorders on the Patient.....	5
1.2. Effects of Psychological Disorders on Families/Caregivers	6
1.3. Effects of Psychological Disorders on Society.....	7
1.4. Mental health.....	8
1.4.1. History	9
1.4.2. Significance	10
1.4.3. Perspectives	10
1.4.3.4. Emotional mental health issues around the world	12
1.4.3.7. Emotional mental health improvement.....	17
1.5. Mental Health Illnesses	18
Part II. Genocide.....	20
2.1. Etymology	20
2.2. Genocide as a crime.....	21
2.2.1. International law	21
2.2.2. Specific provisions	22
2.2.3. CPPCG coming into force.....	23
2.2.4. UN Security Council on genocide	23
2.2.5. Municipal law	24
2.3. Criticisms of the CPPCG and other definitions of genocide	24
2.4. By ad hoc tribunals.....	25
2.5. Nuremberg Tribunal (1945–1946).....	26
2.6. International Criminal Tribunal for the Former Yugoslavia (1993 to present)	26
2.7. International Criminal Tribunal for Rwanda	27
2.8. Extraordinary Chambers in the Courts of Cambodia	28
2.9. By the International Criminal Court.....	29
2.10. Darfur, Sudan	29
2.11. Stages of genocide, influences leading to genocide, and efforts to prevent it.....	30
Part III. Posttraumatic Stress Disorder (PTSD).....	34

3.1.	Terminology	34
3.2.	Classification	34
3.3.	Causes.....	34
3.3.1.	Domestic violence	35
3.3.2.	Evolutionary psychology	35
3.4.	Risk factors	36
3.4.1.	Military experience.....	36
3.4.2.	Drug misuse.....	37
3.4.3.	Foster care	38
3.5.	Pathophysiology	38
3.5.1.	Neuroendocrinology	38
3.5.2.	Neuroanatomy	39
3.6.	Diagnosis	41
3.6.1.	Diagnostic and Statistical Manual	41
3.6.2.	International Classification of Diseases.....	43
3.6.3.	Differential Diagnosis.....	44
3.7.	Prevention.....	44
3.7.1.	Early detection.....	44
3.7.2.	Psychological debriefing	45
3.7.3.	Risk-targeted interventions.....	45
3.7.4.	Medications	46
3.8.	Management	46
3.8.1.	Psychological.....	46
3.8.2.	Cognitive behavioral therapy.....	47
3.8.3.	Eye movement desensitization and reprocessing	47
3.8.4.	Interpersonal psychotherapy.....	48
3.8.5.	Medication.....	48
3.8.6.	Symptom management	48
3.8.7.	Medications by symptom group affected	50
	REFERENCES	54
	References for Mental health and PTSD	54
	References for Genocide	68

Part I. Introduction to Global Mental Health: Effects of Mental Health on Individuals and Populations

The World Health Organization (WHO) recognizes the importance of psychological well-being, defining health as “*a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity (WHO, 2003)*”. In 2002, of the estimated 450 million people worldwide living with mental or behavioral disorders, 90 million were drug or alcohol dependent, 25 million suffered from schizophrenia, and 150 million had depression.

Though most efforts to improve global mental health focus on improving care for individuals living with psychological disorders, the WHO stresses that a comprehensive definition of mental health should extend beyond the absence or presence of diagnosable psychological disorders to include “subjective well-being, perceived self-efficacy, autonomy, competence, intergenerational dependence and recognition of the ability to realize one’s intellectual and emotional potential” (WHO, 2003). Although the following modules will focus on the public health implications of psychological disorders, mental health delivery programs should utilize this more inclusive definition of mental health.

While it is often overlooked as a public health issue due to a historical focus on communicable and more immediately life-threatening diseases (such as HIV/AIDS and malaria), mental health has profound effects on an individual’s quality of life, physical and social well-being, and economic productivity. Because psychological disorders also affect families and communities of the mentally ill, understanding the effects of mental illness on individual patients and social systems is necessary for the improvement of mental health care systems and the development of effective mental health care delivery programs.

1.1. Effects of Psychological Disorders on the Patient

Individuals with psychological disorders are at greater risk for decreased quality of life, educational difficulties, lowered productivity and poverty, social problems, vulnerability to abuse, and additional health problems. Education is often compromised when early-onset mental disorders prevent individuals from completing their education or successfully pursuing a career. Kessler et al. (1995) found that individuals with a psychological disorder were significantly less likely to complete high school, enter college, or receive a college degree, compared to their peers without mental illness (Kessler, R.C.,

Foster, C.L., Saunders, W.B., Stang, P.E., 1995). In addition, psychological disorders result in lowered individual productivity due to unemployment, missed work, and reduced productivity at work. A 2001 study found that five to six million U.S. workers aged 16 to 54 years “lose, fail to seek, or cannot find employment” due to mental illness. Of mentally ill individuals who were employed, mental illness was estimated to reduce their annual income by \$3,500 to \$6,000 (Marcotte, D.E., Wilcox-Gok, V., 2001). Reduced earnings and decreased employment potential put mentally ill individuals at an increased risk of poverty. As Lund et al. (2011) explain, mental illness and poverty “interact in a negative cycle”, in which poverty acts as a risk factor for mental illness, and mental illness increases the risk that individuals will “drift into or remain in poverty” (Lund, C., DeSilva, M., Plagerson, S., Cooper, S., Chisholm, D., Das, Jishnu, Knapp, M., Patel, V., 2011). This negative cycle may also contribute to high rates of homelessness among individuals with mental illness; the Substance Abuse and Mental Health Services Administration estimates that 20 to 25 % of the U.S. homeless population suffers from severe mental illness, while only 6% of the general U.S. population is severely mentally ill.

Psychological disorders can also contribute to other health problems and stressors. For instance, patients with comorbid depression (depression co-occurring with another health condition) are three times less likely to adhere to medical treatment regimens than are non-depressed patients. Furthermore, mentally ill individuals are vulnerable to low-quality care, abuse, and human rights violations, particularly in low-income areas with limited mental health care resources. Mentally ill individuals and their families may also experience significant social stigma and discrimination (WHO, 2003)

1.2. Effects of Psychological Disorders on Families/Caregivers

The burden of caring for a mentally ill individual often falls on the patient’s immediate family or relatives. Families and caregivers of individuals with psychological disorders are often unable to work at full capacity due to the demands of caring for a mentally ill individual, leading to decreased economic output and a reduction in household income. Loss of income and the financial costs of caring for a mentally ill person put these households at an increased risk of poverty. Family members may also experience significant and chronic stress due to the emotional and physical challenges of caring for a mentally ill family member. Although the experience of caring for mentally ill relatives varies among families and cultures, a 1999 review article reported that family caregivers’ largest challenges were providing assistance with daily activities (e.g. providing transportation, offering financial assistance,

helping with housework, cleaning, and money management) and stress associated with care (e.g. concerns about possible violence, embarrassing behaviors, and intra-family conflict) (Baronet, A-M.,1999).

For instance, a 2006 study in Botswana investigated the experiences of families caring for a mentally ill family member. The study was conducted using in-depth interviews, focus group discussions, and field observations in Gaborone, the capital city, and Molepolole, a rural village. Although the extended family structure common in Botswana allowed for distribution of caregiver responsibilities, most families reported that lack of financial and medical resources at the family and community levels made it difficult and stressful to provide adequate care. In South Africa, in-depth interviews with eight family caregivers in Limpopo revealed that many caregivers felt that their own physical and mental well-being was at risk, particularly when caring for a violent or destructive family member. Caregivers also reported social isolation due to their family member's mental illness, as caregiving duties prevented them from attending social events such as funerals and church services. Particularly in rural areas lacking community resources for the mentally ill, the degree of satisfaction with family functioning (perception of "family burden") and the size of a caregiver's support network may significantly influence patient functioning, with increased support improving patient outcomes even in cases with high reported family burden (Kohn-Wood, L.P., Wilson, M.N., 2005).

1.3. Effects of Psychological Disorders on Society

Although the specific societal impact of mental illness varies among cultures and nations, untreated mental illness has significant costs to society. In 2001, the WHO estimated that mental health problems cost developed nations between three and four % of their GNP (gross national product). When mental illness expenditures and loss of productivity are both taken into account, the WHO estimated that mental disorders cost national economies several billion dollars annually. In 1997, a Harvard Medical School study estimated that the United States lost more than 4 million workdays and experienced 20 million "work cutback days" (days of impaired workplace performance) due to mental illness (Kessler, R.C., Frank, R.G., 1997).

In addition, psychological disorders can exacerbate other public health issues, increasing the burden on national economies and impeding international public health efforts. In 2001, at least five to ten million

people worldwide used intravenous drugs, and five to ten % of new HIV infections were due to transmission via intravenous drug use. Mental illnesses are also associated with increased risk of non-adherence to medical regimens for other health conditions. For infectious diseases, improper or incomplete use of medication can lead to drug resistance, which may have “profound public health implications” for the global community. Furthermore, maternal depression may put infants at increased risk of low birth weight, childhood health problems, and “incomplete immunization”, all of which are risk factors for childhood mortality (Patek, V., 2007).

Although the majority of individuals with mental illness do not exhibit dangerous behaviors, violence and incarceration among mentally ill individuals can place a significant financial and social burden on communities and nations. Worldwide, approximately 10 million people are incarcerated, and the WHO reports that the prevalence of mental health problems is “very high”, especially among female inmates. In the U.S. in the late 2000s, nearly one million adults with serious psychological disorders were incarcerated annually. A study in the Pinellas County, Florida jail found that not having outpatient mental health treatment was significantly associated with increased risk of misdemeanor arrests and days incarcerated, and having a substance abuse disorder was associated with more days in jail, which is consistent with national incarceration statistics. National data from the 2002 Survey of Inmates in Local Jails revealed that homelessness was significantly more prevalent among the inmate population as compared to the general U.S. adult population, and inmates who had been homeless were significantly more likely than were other inmates to have mental health and substance abuse problems. The authors posit that the relationship between homelessness and mental illness “may reflect limited access to mental health services, particularly inpatient services”, due to deinstitutionalization in the United States, which has resulted in limited availability of psychiatric hospital beds, and strict criteria for hospitalization. The WHO recommends that developing and developed nations adopt more comprehensive preventative and interventional mental health programs to reduce the negative effects of mental illness on patients and their local and global communities (Greenberg, G.A., Rosenheck, R.A., 2008).

1.4. Mental health

Mental health describes a level of psychological well-being, or an absence of a mental disorder. From the perspective of 'positive psychology' or 'holism', mental health may include an individual's ability to enjoy life, and create a balance between life activities and efforts to achieve psychological resilience.

Mental health can also be defined as an expression of emotions, and as signifying a successful adaptation to a range of demands.

The World Health Organization defines mental health as "a state of well-being in which the individual realizes his or her own abilities, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to his or her community".^[3] It was previously stated that there was no "official" definition of mental health. Cultural differences, subjective assessments, and competing professional theories all affect how "mental health" is defined. There are different types of mental health problems, some of which are common, such as depression and anxiety disorders, and some not so common, such as schizophrenia and bipolar disorder. (Kitchener, BA & Jorm, AF, 2002)

Most recently, the field of global mental health has emerged, which has been defined as 'the area of study, research and practice that places a priority on improving mental health and achieving equity in mental health for all people worldwide (Patel, V., Prince, M., 2000).

1.4.1. History

In the mid-19th century, William Sweetzer was the first to clearly define the term "mental hygiene", which can be seen as the precursor to contemporary approaches to work on promoting positive mental health. Isaac Ray, one of the thirteen founders of the American Psychiatric Association, further defined mental hygiene as an art to preserve the mind against incidents and influences which would inhibit or destroy its energy, quality or development (Johns Hopkins University, 2007).

Dorothea Dix (1802–1887) was an important figure in the development of "mental hygiene" movement. Dix was a school teacher who endeavored throughout her life to help those suffering from mental illness, and to bring to light the deplorable conditions into which they were put. This was known as the "mental hygiene movement". Before this movement, it was not uncommon that people affected by mental illness in the 19th century would be considerably neglected, often left alone in deplorable conditions, barely even having sufficient clothing. Dix's efforts were so great that there was a rise in the number of patients in mental health facilities, which sadly resulted in these patients receiving less attention and care, as these institutions were largely understaffed (Barlow, D.H., Durand, V.M., Steward, S.H. 2009).

At the beginning of the 20th century, Clifford Beers founded the National Committee for Mental Hygiene and opened the first outpatient mental health clinic in the United States of America.^{[7][9]}

The mental hygiene movement, related to the social hygiene movement, had at times been associated with advocating eugenics and sterilization of those considered too mentally deficient to be assisted into productive work and contented family life.

After 1945, references to mental hygiene were gradually replaced by the term mental health.

1.4.2. Significance

Evidence from the World Health Organization suggests that nearly half the world's population are affected by mental illness with an impact on their self-esteem, relationships and ability to function in everyday life. An individual's emotional health can also impact physical health and poor mental health can lead to problems such as substance abuse (Richards, K.C.; Campania, C. Muse-Burke J.L, 2010).

Maintaining good mental health is crucial to living a long and healthy life. Good mental health can enhance one's life, while poor mental health can prevent someone from living an enriching life. According to Richards, Campania, & Muse-Burke (2010) "There is growing evidence that is showing emotional abilities are associated with prosocial behaviors such as stress management and physical health" (2010). It was also concluded in their research that people who lack emotional expression are inclined to anti-social behaviors. These behaviors are a direct reflection of their mental health. Self-destructive acts may take place to suppress emotions. Some of these acts include drug and alcohol abuse, physical fights or vandalism.

1.4.3. Perspectives

1.4.3.1. Mental well-being

Mental health can be seen as an unstable continuum, where an individual's mental health may have many different possible values. Mental wellness is generally viewed as a positive attribute, such that a person can reach enhanced levels of mental health, even if the person does not have any diagnosed mental health condition. This definition of mental health highlights emotional well-being, the capacity to live a full and creative life, and the flexibility to deal with life's inevitable challenges. Many therapeutic

systems and self-help books offer methods and philosophies espousing strategies and techniques vaunted as effective for further improving the mental wellness of otherwise healthy people. Positive psychology is increasingly prominent in mental health.

A holistic model of mental health generally includes concepts based upon anthropological, educational, psychological, religious and sociological perspectives, as well as theoretical perspectives from personality, social, clinical, health and developmental psychology (Hattie, J.A.; Myers, J.E.; Sweeney, T.J., 2004).

An example of a wellness model includes one developed by Myers, Sweeney and Witmer. It includes five life tasks—essence or spirituality, work and leisure, friendship, love and self-direction—and twelve sub tasks—sense of worth, sense of control, realistic beliefs, emotional awareness and coping, problem solving and creativity, sense of humor, nutrition, exercise, self care, stress management, gender identity, and cultural identity—which are identified as characteristics of healthy functioning and a major component of wellness. The components provide a means of responding to the circumstances of life in a manner that promotes healthy functioning. The population of the USA in its majority is considered to be mostly uneducated on the subjects of mental health.

1.4.3.2. Prevention

Mental health can also be defined as an absence of a mental disorder. Focus is increasing on preventing mental disorders. Prevention is beginning to appear in mental health strategies, including the 2004 WHO report "Prevention of Mental Disorders", the 2008 EU "Pact for Mental Health" and the 2011 US National Prevention Strategy. Prevention of a disorder at a young age may significantly decrease the chances that a child will suffer from a disorder later in life (National Research Council & Institute of Medicine, 2009).

1.4.3.3. Cultural and religious considerations

Mental health is a socially constructed and socially defined concept; that is, different societies, groups, cultures, institutions and professions have very different ways of conceptualizing its nature and causes, determining what is mentally healthy, and deciding what interventions, if any, are appropriate. Thus,

different professionals will have different cultural, class, political and religious backgrounds, which will impact the methodology applied during treatment.

Research has shown that there is stigma attached to mental illness. In the United Kingdom, the Royal College of Psychiatrists organized the campaign *Changing Minds* (1998–2003) to help reduce stigma.

Many mental health professionals are beginning to, or already understand, the importance of competency in religious diversity and spirituality. The American Psychological Association explicitly states that religion must be respected. Education in spiritual and religious matters is also required by the American Psychiatric Association (Richards, P.S.; Bergin, A.E., 2000)

1.4.3.4. Emotional mental health issues around the world

Emotional mental disorders are a leading cause of disabilities worldwide. Investigating the degree and severity of untreated emotional mental disorders throughout the world is a top priority of the World Mental Health (WMH) survey initiative, which was created in 1998 by the World Health Organization (WHO). “Neuropsychiatric disorders are the leading causes of disability worldwide, accounting for 37% of all healthy life years lost through disease. These disorders are most destructive to low and middle-income countries due to their inability to provide their citizens with proper aid. Despite modern treatment and rehabilitation for emotional mental health disorders, “even economically advantaged societies have competing priorities and budgetary constraints”.

The World Mental Health survey initiative has suggested a plan for countries to redesign their mental health care systems to best allocate resources. “A first step is documentation of services being used and the extent and nature of unmet needs for treatment. A second step could be to do a cross-national comparison of service use and unmet needs in countries with different mental health care systems. Such comparisons can help to uncover optimum financing, national policies, and delivery systems for mental health care.”

Knowledge of how to provide effective emotional mental health care has become imperative worldwide. Unfortunately, most countries have insufficient data to guide decisions, absent or competing visions for resources, and near constant pressures to cut insurance and entitlements. WMH surveys were done in Africa (Nigeria, South Africa), the Americas (Colombia, Mexico, U.S.A), Asia and the Pacific (Japan,

New Zealand, Beijing and Shanghai in the Peoples Republic of China), Europe (Belgium, France, Germany, Italy, Netherlands, Spain, Ukraine), and the middle east (Israel, Lebanon). Countries were classified with World Bank criteria as low-income (Nigeria), lower middle-income (China, Columbia, South Africa, Ukraine), higher middle-income (Lebanon, Mexico), and high-income.

The coordinated surveys on emotional mental health disorders, their severity, and treatments were implemented in the aforementioned countries. These surveys assessed the frequency, types, and adequacy of mental health service use in 17 countries in which WMH surveys are complete. The WMH also examined unmet needs for treatment in strata defined by the seriousness of mental disorders. Their research showed that “the number of respondents using any 12-month mental health service was generally lower in developing than in developed countries, and the proportion receiving services tended to correspond to countries’ percentages of gross domestic product spent on health care”. “High levels of unmet need worldwide are not surprising, since WHO Project ATLAS’ findings of much lower mental health expenditures than was suggested by the magnitude of burdens from mental illnesses. Generally, unmet needs in low-income and middle-income countries might be attributable to these nations spending reduced amounts (usually <1%) of already diminished health budgets on mental health care, and they rely heavily on out-of-pocket spending by citizens who are ill equipped for it”.

1.4.3.5. Emotional mental health in the United States

According to the World Health Organization in 2004, depression is the leading cause of disability in the United States of America for individuals ages 15 to 44 (Thomson Healthcare, 2007) Absence from work in the U.S. due to depression is estimated to be in excess of \$31 billion per year. Depression frequently co-occurs with a variety of medical illnesses such as heart disease, cancer, and chronic pain and is associated with poorer health status and prognosis. Each year, roughly 30,000 Americans take their lives, while hundreds of thousands make suicide attempts (Centers for Disease Control and Prevention). In 2004, suicide was the 11th leading cause of death in the United States of America (Centers for Disease Control and Prevention), third among individuals ages 15–24. Despite the increasingly availability of effectual depression treatment, the level of unmet need for treatment remains high (Munce, SE; Stansfeld SA, Blackmore ER, Stewart DE, 2007).

There are many factors that influence mental health including:

- Mental illness, disability, and suicide are ultimately the result of a combination of biology, environment, and access to and utilization of mental health treatment.
- Public health policies can influence access and utilization, which subsequently may improve mental health and help to progress the negative consequences of depression and its associated disability.
- Research conducted by Mental Health America found the following factors to be considerably allied with improved depression status and lower suicide rates:

Mental health resources	On average, the higher the number of psychiatrists, psychologists, and social workers per capita in a state, the lower the suicide rate.
Barriers to treatment	The lower the percentage of the population reporting that they could not obtain healthcare because of costs, the lower the suicide rate and the better the state's depression status.
Mental health treatment utilization	The lower the percentage of the population that reported unmet mental healthcare needs, the better the state's depression status. When mental health treatment is utilized more, while holding the baseline level of depression in the state constant, the higher the number of antidepressant prescriptions per capita in the state, and the lower the suicide rate.
Socioeconomic characteristics	The more educated the population and the greater the percentage with health insurance, the lower the suicide rate. The more educated the population, the better the state's depression status.
Mental health policy	The more generous a state's mental health parity coverage, the greater the number of people in the population that receive mental health services.

The Spirit Level: Why More Equal Societies Almost Always Do Better argues about 50% of the reason why Americans have the highest rates of mental illness including depression, anxiety and addiction is due to America having the highest income inequality in the world (Pickett, Richard Wilkinson, Kate, 2009).

Emotional mental illnesses should be a particular concern in the United States of America since the U.S.A has the highest annual prevalence rates (26 percent) for mental illnesses among a comparison of 14 developing and developed countries. While approximately 80 percent of all people in the United States with a mental disorder eventually receive some form of treatment, on the average persons do not access care until nearly a decade following the development of their illness, and less than one-third of people who seek help receive minimally adequate care.

1.4.3.6. Mental health policies in the United States

The mental health policies in the United States have experienced four major reforms: the American asylum movement led by Dorothea Dix in 1843; the “mental hygiene” movement inspired by Clifford Beers in 1908; the deinstitutionalization started by Action for Mental Health in 1961; and the community support movement called for by The CMCH Act Amendments of 1975.

In 1843, Dorothea Dix submitted a Memorial to the Legislature of Massachusetts, describing the abusive treatment and horrible conditions received by the mentally ill patients in jails, cages, and almshouses. She revealed in her Memorial: “I proceed, gentlemen, briefly to call your attention to the present state of insane persons confined within this Commonwealth, in cages, closets, cellars, stalls, pens! Chained, naked, beaten with rods, and lashed into obedience. . . .” (Dix, D., 2006) Many asylums were built in that period, with high fences or walls separating the patients from other community members and strict rules regarding the entrance and exit. In those asylums, traditional treatments were well implemented: drugs were not used as a cure for a disease, but a way to reset equilibrium in a person’s body, along with other essential elements such as healthy diets, fresh air, middle class culture, and the visits by their neighboring residents. In 1866, a recommendation came to the New York State Legislature to establish a separate asylum for chronic mentally ill patients. Some hospitals placed the chronic patients into separate wings or wards, or different buildings (Luchins, A. S., 2001).

In *A Mind That Found Itself* (1908) Clifford Whittingham Beers described the humiliating treatment he received and the deplorable conditions in the mental hospital. One year later, the National Committee for Mental Hygiene (NCMH) was founded by a small group of reform-minded scholars and scientists – including Beer himself – which marked the beginning of the “mental hygiene” movement. The movement emphasized the importance of childhood prevention. World War I catalyzed this idea with an

additional emphasis on the impact of maladjustment, which convinced the hygienists that prevention was the only practical approach to handle mental health issues. However, prevention was not successful, especially for chronic illness; the condemnable conditions in the hospitals were even more prevalent, especially under the pressure of the increasing number of chronically ill and the influence of the Depression.

In 1961, the Joint Commission on Mental Health published a report called Action for Mental Health, whose goal was for community clinic care to take on the burden of prevention and early intervention of the mental illness, therefore to leave space in the hospitals for severe and chronic patients. The court started to rule in favor of the patients' will on whether they should be forced to treatment. By 1977, 650 community mental health centers were built to cover 43 percent of the population and serve 1.9 million individuals a year, and the lengths of treatment decreased from 6 months to only 23 days. However, issues still existed. Due to inflation, especially in the 1970s, the community nursing homes received less money to support the care and treatment provided. Fewer than half of the planned centers were created, and new methods did not fully replace the old approaches to carry out its full capacity of treating power. Besides, the community helping system was not fully established to support the patients' housing, vocational opportunities, income supports, and other benefits. Many patients returned to welfare and criminal justice institutions, and more became homeless. The movement of deinstitutionalization was facing great challenges.

After realizing that simply changing the location of mental health care from the state hospitals to nursing houses was insufficient to implement the idea of deinstitutionalization, the National Institute of Mental Health in 1975 created the Community Support Program (CSP) to provide funds for communities to set up a comprehensive mental health service and supports to help the mentally ill patients integrate successfully in the society. The program stressed the importance of other supports in addition to medical care, including housing, living expenses, employment, transportation, and education; and set up new national priority for people with serious mental disorders. In addition, the Congress enacted the Mental Health Systems Act to prioritize the service to the mentally ill and emphasize the expansion of services beyond just clinical care alone. Later in the 1980s, under the influence from the Congress and the Supreme Court, many programs started to help the patients regain their benefits. A new Medicaid service was also established to serve people who were suffering from a "chronic mental illness." People who were temporally hospitalized were also provided aid and care and a pre-release program was

created to enable people to apply for reinstatement prior to discharge. Not until 1990, around 35 years after the start of the deinstitutionalization, did the first state hospital begin to close. The number of hospitals dropped from around 300 by over 40 in the 1990s, and finally a Report on Mental Health showed the efficacy of mental health treatment, giving a range of treatments available for patients to choose (Koyanagi, C., 2007).

The 2011 National Prevention Strategy included mental and emotional well-being, with recommendations including better parenting and early intervention programs, which increase the likelihood of prevention programs being included in future US mental health policies. The NIMH is researching only suicide and HIV/AIDS prevention, but the National Prevention Strategy could lead to it focusing more broadly on longitudinal prevention studies.

1.4.3.7. Emotional mental health improvement

Being mentally and emotionally healthy does not preclude the experiences of life which we cannot control. As humans we are going to face emotions and events that are a part of life. According to Smith and Segal, “People who are emotionally and mentally healthy have the tools for coping with difficult situations and maintaining a positive outlook in which they also remain focused, flexible, and creative in bad times as well as good” (Smith, M; Segal, R. Segal, J., 2011) (2011). In order to improve your emotional mental health, the root of the issue has to be resolved. “Prevention emphasizes the avoidance of risk factors; promotion aims to enhance an individual’s ability to achieve a positive sense of self-esteem, mastery, well-being, and social inclusion” (Power, A., 2010) (Power, 2010). It is very important to improve your emotional mental health by surrounding yourself with positive relationships. We as humans, feed off companionships and interaction with other people. Another way to improve your emotional mental health is participating in activities that can allow you to relax and take time for yourself. Yoga is a great example of an activity that calms your entire body and nerves. According to a study on well-being by Richards, Campania and Muse-Burke, “mindfulness is considered to be a purposeful state, it may be that those who practice it believe in its importance and value being mindful, so that valuing of self-care activities may influence the intentional component of mindfulness” (Richards, K.C.; Campania, C. Muse-Burke, J.L., 2010).

1.5. Mental Health Illnesses

There are many different conditions that are recognized as mental illnesses. The more common types include:

- **Anxiety disorders:** People with anxiety disorders respond to certain objects or situations with fear and dread, as well as with physical signs of anxiety or nervousness, such as a rapid heartbeat and sweating. An anxiety disorder is diagnosed if the person's response is not appropriate for the situation, if the person cannot control the response, or if the anxiety interferes with normal functioning. Anxiety disorders include generalized anxiety disorder, post-traumatic stress disorder (PTSD), obsessive-compulsive disorder (OCD), panic disorder, social anxiety disorder, and specific phobias.
- **Mood disorders:** These disorders, also called affective disorders, involve persistent feelings of sadness or periods of feeling overly happy, or fluctuations from extreme happiness to extreme sadness. The most common mood disorders are depression, mania, and bipolar disorder.
- **Psychotic disorders:** Psychotic disorders involve distorted awareness and thinking. Two of the most common symptoms of psychotic disorders are hallucinations -- the experience of images or sounds that are not real, such as hearing voices -- and delusions, which are false beliefs that the ill person accepts as true, despite evidence to the contrary. Schizophrenia is an example of a psychotic disorder.
- **Eating disorders:** Eating disorders involve extreme emotions, attitudes, and behaviours involving weight and food. Anorexia nervosa, bulimia nervosa and binge eating disorder are the most common eating disorders.
- **Impulse control and addiction disorders:** People with impulse control disorders are unable to resist urges, or impulses, to perform acts that could be harmful to themselves or others. Pyromania (starting fires), kleptomania (stealing), and compulsive gambling are examples of impulse control disorders. Alcohol and drugs are common objects of addictions. Often, people with these disorders become so involved with the objects of their addiction that they begin to ignore responsibilities and relationships.
- **Personality disorders:** People with personality disorders have extreme and inflexible personality traits that are distressing to the person and/or cause problems in work, school, or social relationships. In addition, the person's patterns of thinking and behaviour significantly

differ from the expectations of society and are so rigid that they interfere with the person's normal functioning. Examples include antisocial personality disorder, obsessive-compulsive personality disorder, and paranoid personality disorder.

Other, less common types of mental illnesses include:

- **Adjustment disorder:** Adjustment disorder occurs when a person develops emotional or behavioural symptoms in response to a stressful event or situation. The stressors may include natural disasters, such as an earthquake or tornado; events or crises, such as a car accident or the diagnosis of a major illness; or interpersonal problems, such as a divorce, death of a loved one, loss of a job, or a problem with substance abuse. Adjustment disorder usually begins within three months of the event or situation and ends within six months after the stressor stops or is eliminated.
- **Dissociative disorders:** People with these disorders suffer severe disturbances or changes in memory, consciousness, identity, and general awareness of themselves and their surroundings. These disorders usually are associated with overwhelming stress, which may be the result of traumatic events, accidents, or disasters that may be experienced or witnessed by the individual. Dissociative identity disorder, formerly called multiple personality disorder, or "split personality," and depersonalization disorder are examples of dissociative disorders.
- **Factitious disorders:** Factitious disorders are conditions in which physical and/or emotional symptoms are created in order to place the individual in the role of a patient or a person in need of help.
- **Sexual and gender disorders:** These include disorders that affect sexual desire, performance, and behaviour. Sexual dysfunction, gender identity disorder, and the paraphilias are examples of sexual and gender disorders.
- **Somatoform disorders:** A person with a somatoform disorder, formerly known as psychosomatic disorder, experiences physical symptoms of an illness, even though a doctor can find no medical cause for the symptoms.
- **Tic disorders:** People with tic disorders make sounds or display body movements that are repeated, quick, sudden, and/or uncontrollable. (Sounds that are made involuntarily are called vocal tics.) Tourette's syndrome is an example of a tic disorder.

Part II. Genocide

Genocide is the systematic destruction of all or a significant part of a [racial](#), [ethnic](#), [religious](#) or national group. Well-known examples of genocide include the [Holocaust](#), the [Armenian genocide](#), and more recently the [Rwandan genocide](#).

2.1. Etymology

[Raphael Lemkin](#), in his work *Axis Rule in Occupied Europe* (1944), coined the term "genocide" by combining Greek *genos* (γένος), "race, people" and Latin *cidere* "to kill".^[1]

Lemkin defined genocide as follows:

Generally speaking, genocide does not necessarily mean the immediate destruction of a nation, except when accomplished by mass killings of all members of a nation. It is intended rather to signify a coordinated plan of different actions aiming at the destruction of essential foundations of the life of national groups, with the aim of annihilating the groups themselves. The objectives of such a plan would be the disintegration of the political and social institutions, of culture, language, national feelings, religion, and the economic existence of national groups, and the destruction of the personal security, liberty, health, dignity, and even the lives of the individuals belonging to such groups.

The preamble to the [Genocide Convention](#) ("CPPCG") notes that instances of genocide have taken place throughout history,^[2] but it was not until Lemkin coined the term and the prosecution of perpetrators of the Holocaust at the [Nuremberg trials](#) that the United Nations defined the crime of genocide under [international law](#) in the Genocide Convention.

During an interview with Lemkin, the interviewer asked him about how he came to be interested in this genocide. He replied: "I became interested in genocide because it happened so many times. [It happened to the Armenians](#), then after the Armenians, [Hitler](#) took action."^{[3][4]}

Lemkin was also a close relative of genocide victims, losing 49 relatives in the Holocaust. However, his work on defining genocide as a crime dates to 1933, and it was prompted by the [Simele massacre](#) in Iraq.^[5]

2.2. Genocide as a crime

2.2.1. International law

[Buchenwald concentration camp](#) was not an extermination camp, though it was responsible for a vast number of deaths

After the Holocaust, which had been perpetrated by the [Nazi Germany](#) and its allies prior to and during [World War II](#), [Lemkin](#) successfully campaigned for the universal acceptance of international laws defining and forbidding genocide. In 1946, the first session of the [United Nations General Assembly](#) adopted a [resolution](#) that "affirmed" that genocide was a crime under international law, but did not provide a legal definition of the crime. In 1948, the UN General Assembly adopted the [Convention on the Prevention and Punishment of the Crime of Genocide](#) (CPPCG) which defined the crime of genocide for the first time.^[6]

The *CPPCG* was adopted by the UN General Assembly on 9 December 1948 and came into effect on 12 January 1951 (Resolution 260 (III)). It contains an internationally recognized definition of genocide which has been incorporated into the national criminal legislation of many countries, and was also adopted by the [Rome Statute of the International Criminal Court](#), which established the [International Criminal Court](#) (ICC). Article II of the Convention defines genocide as:

...any of the following acts committed with [intent to destroy, in whole or in part](#), a national, ethnical, racial or religious group, as such:

- (a) Killing members of the group;
- (b) Causing serious bodily or mental harm to members of the group;
- (c) Deliberately inflicting on the group conditions of life calculated to bring about its physical destruction in whole or in part;
- (d) Imposing measures intended to prevent births within the group;
- (e) Forcibly transferring children of the group to another group.

The first draft of the Convention included political killings, but these provisions were removed in a political and diplomatic compromise following objections from some countries, including the [USSR](#), a permanent Security Council member.^{[7][8]} The USSR argued that the Convention's definition should follow the etymology of the term,^[8] and may have feared greater international scrutiny of its own [Great Purge](#).^[7] Other nations feared that including political groups in the definition would invite international intervention in domestic politics.^[8]

The convention's purpose and scope was later described by the United Nations Security Council as follows:

The Convention was manifestly adopted for humanitarian and civilizing purposes. Its objectives are to safeguard the very existence of certain human groups and to affirm and emphasize the most elementary principles of humanity and morality.

When the Convention was drafted, it was already envisaged that it would apply not only to then existing forms of genocide, but also "to any method that might be evolved in the future with a view to destroying the physical existence of a group".^[9] As emphasized in the preamble to the Convention, genocide has marred all periods of history, and it is this very tragic recognition that gives the concept its historical evolutionary nature.

The Convention must be interpreted in good faith, in accordance with the ordinary meaning of its terms, in their context, and in the light of its object and purpose. Moreover, the text of the Convention should be interpreted in such a way that a reason and a meaning can be attributed to every word. No word or provision may be disregarded or treated as superfluous, unless this is absolutely necessary to give effect to the terms read as a whole.^[10]

Genocide is a crime under international law regardless of "whether committed in time of peace or in time of war" (art. I). Thus, irrespective of the context in which it occurs (for example, peace time, internal strife, international armed conflict or whatever the general overall situation) genocide is a punishable international crime.

— UN Commission of Experts that examined violations of international humanitarian law committed in the territory of the former Yugoslavia.^[11]

2.2.2. Specific provisions

"Intent to destroy"

In 2007 the [European Court of Human Rights](#) (ECHR), noted in its judgement on *Jorgic v. Germany* case that in 1992 the majority of legal scholars took the narrow view that "intent to destroy" in the CPPCG meant the intended physical-biological destruction of the protected group and that this was still the majority opinion. But the ECHR also noted that a minority took a broader view and did not consider biological-physical destruction was necessary as the intent to destroy a national, racial, religious or ethnic group was enough to qualify as genocide.^[12]

In the same judgement the ECHR reviewed the judgements of several international and municipal courts judgements. It noted that [International Criminal Tribunal for the Former Yugoslavia](#) and the [International Court of Justice](#) had agreed with the narrow interpretation, that biological-physical destruction was necessary for an act to qualify as genocide. The ECHR also noted that at the time of its judgement, apart from courts in Germany which had taken a broad view, that there had been few cases of genocide under other Convention States [municipal laws](#) and that "There are no reported cases in which the courts of these States have defined the type of group destruction the perpetrator must have intended in order to be found guilty of genocide".^[13]

"In part"

The phrase "in whole or in part" has been subject to much discussion by scholars of international humanitarian law.^[14] The [International Criminal Tribunal for the Former Yugoslavia](#) found in *Prosecutor v. Radislav Krstic – Trial Chamber I – Judgment – IT-98-33 (2001) ICTY8 (2 August 2001)*^[15] that Genocide had been committed. In *Prosecutor v. Radislav Krstic – Appeals Chamber – Judgment – IT-98-33 (2004) ICTY 7 (19 April 2004)*^[16] paragraphs 8, 9, 10, and 11 addressed the issue of *in part* and found that "the part must be a substantial part of that group. The aim of the Genocide Convention is to prevent the intentional destruction of entire human groups, and the part targeted must be significant enough to have an impact on the group as a whole." The Appeals Chamber goes into details of other cases and the opinions of respected commentators on the Genocide Convention to explain how they came to this conclusion.

The judges continue in paragraph 12, "The determination of when the targeted part is substantial enough to meet this requirement may involve a number of considerations. The numeric size of the targeted part of the group is the necessary and important starting point, though not in all cases the ending point of the inquiry. The number of individuals targeted should be evaluated not only in absolute terms, but also in relation to the overall size of the entire group. In addition to the numeric size of the targeted portion, its prominence within the group can be a useful consideration. If a specific part of the group is emblematic of the overall group, or is essential to its survival, that may support a finding that the part qualifies as substantial within the meaning of Article 4 [of the Tribunal's Statute]."^{[17][18]}

In paragraph 13 the judges raise the issue of the perpetrators' access to the victims: "The historical examples of genocide also suggest that the area of the perpetrators' activity and control, as well as the possible extent of their reach, should be considered. ... The intent to destroy formed by a perpetrator of genocide will always be limited by the opportunity presented to him. While this factor alone will not indicate whether the targeted group is substantial, it can—in combination with other factors—inform the analysis."^[16]

2.2.3. CPPCG coming into force

The Convention came into force as international law on 12 January 1951 after the minimum 20 countries became parties. At that time, however, only two of the five permanent members of the [UN Security Council](#) were parties to the treaty: France and the [Republic of China](#). The [Soviet Union](#) ratified in 1954, the United Kingdom in 1970, the People's Republic of China in 1983 (having replaced the Taiwan-based Republic of China on the UNSC in 1971), and the United States in 1988. This long delay in support for the Convention by the world's most powerful nations caused the Convention to languish for over four decades. Only in the 1990s did the international law on the crime of genocide begin to be enforced.

2.2.4. UN Security Council on genocide

[UN Security Council Resolution 1674](#), adopted by the United Nations Security Council on 28 April 2006, "reaffirms the provisions of paragraphs 138 and 139 of the 2005 [World Summit Outcome](#)

[Document](#) regarding the responsibility to protect populations from genocide, war crimes, ethnic cleansing and crimes against humanity".^[19] The [resolution](#) committed the Council to action to protect civilians in armed conflict.^[20]

In 2008 the UN Security Council adopted [resolution 1820](#), which noted that "rape and other forms of sexual violence can constitute war crimes, crimes against humanity or a constitutive act with respect to genocide".^[21]

2.2.5. Municipal law

Since the Convention came into effect in January 1951 about 80 United Nations member states have passed legislation that incorporates the provisions of CPPCG into their [municipal law](#).^[22]

2.3. Criticisms of the CPPCG and other definitions of genocide

William Schabas has suggested that a permanent body as recommended by the [Whitaker Report](#) to monitor the implementation of the Genocide Convention, and require States to issue reports on their compliance with the convention (such as were incorporated into the United Nations [Optional Protocol to the Convention against Torture](#)), would make the convention more effective.^[23]

Writing in 1998 Kurt Jonassohn and Karin Björnson stated that the CPPCG was a legal instrument resulting from a diplomatic compromise. As such the wording of the treaty is not intended to be a definition suitable as a research tool, and although it is used for this purpose, as it has an international legal credibility that others lack, [other definitions](#) have also been postulated. Jonassohn and Björnson go on to say that none of these alternative definitions have gained widespread support for various reasons.^[24]

Jonassohn and Björnson postulate that the major reason why no single generally accepted genocide definition has emerged is because academics have adjusted their focus to emphasise different periods and have found it expedient to use slightly different definitions to help them interpret events. For example Frank Chalk and Kurt Jonassohn studied the whole of human history, while [Leo Kuper](#) and [R. J. Rummel](#) in their more recent works concentrated on the 20th century, and [Helen Fein](#), Barbara Harff and Ted Gurr have looked at post World War II events. Jonassohn and Björnson are critical of some of these studies, arguing that they are too expansive, and conclude that the academic discipline of genocide studies is too young to have a canon of work on which to build an academic [paradigm](#).^[24]

The exclusion of social and political groups as targets of genocide in the CPPCG legal definition has been criticized by some historians and sociologists, for example M. Hassan Kakar in his book *The Soviet Invasion and the Afghan Response, 1979–1982*^[25] argues that the international definition of genocide is too restricted,^[26] and that it should include political groups or any group so defined by the perpetrator and quotes Chalk and Jonassohn: "Genocide is a form of one-sided mass killing in which a state or other authority intends to destroy a group, as that group and membership in it are defined by the perpetrator."^[27] While there are various definitions of the term, Adam Jones states that the majority of

genocide scholars consider that "intent to destroy" is a requirement for any act to be labelled genocide, and that there is growing agreement on the inclusion of the physical destruction criterion.^[28]

Barbara Harff and Ted Gurr defined genocide as "the promotion and execution of policies by a state or its agents which result in the deaths of a substantial portion of a group ...[when] the victimized groups are defined primarily in terms of their communal characteristics, i.e., ethnicity, religion or nationality."^[29] Harff and Gurr also differentiate between genocides and [politicides](#) by the characteristics by which members of a group are identified by the state. In genocides, the victimized groups are defined primarily in terms of their communal characteristics, i.e., ethnicity, religion or nationality. In politicides the victim groups are defined primarily in terms of their hierarchical position or political opposition to the regime and dominant groups.^{[30][31]} Daniel D. Polsby and Don B. Kates, Jr. state that "... we follow Harff's distinction between genocides and '[pogroms](#),' which she describes as 'short-lived outbursts by mobs, which, although often condoned by authorities, rarely persist.' If the violence persists for long enough, however, Harff argues, the distinction between condonation and complicity collapses."^{[32][33]}

According to R. J. Rummel, genocide has 3 different meanings. The ordinary meaning is murder by government of people due to their national, ethnic, racial, or religious group membership. The legal meaning of genocide refers to the international treaty, the *Convention on the Prevention and Punishment of the Crime of Genocide*. This also includes non-killings that in the end eliminate the group, such as preventing births or forcibly transferring children out of the group to another group. A generalized meaning of genocide is similar to the ordinary meaning but also includes government killings of political opponents or otherwise intentional murder. It is to avoid confusion regarding what meaning is intended that Rummel created the term [democide](#) for the third meaning.^[34]

Highlighting the potential for state and non-state actors to commit genocide in the 21st century, for example, in failed states or as non-state actors acquire weapons of mass destruction, Adrian Gallagher defined genocide as 'When a source of collective power (usually a state) intentionally uses its power base to implement a process of destruction in order to destroy a group (as defined by the perpetrator), in whole or in substantial part, dependent upon relative group size'.^[35] The definition upholds the centrality of intent, the multidimensional understanding of destroy, broadens the definition of group identity beyond that of the 1948 definition yet argues that a substantial part of a group has to be destroyed before it can be classified as genocide (dependent on relative group size).

A major criticism of the international community's response to the Rwandan Genocide was that it was reactive, not proactive. The international community has developed a mechanism for prosecuting the perpetrators of genocide but has not developed the will or the mechanisms for intervening in a genocide as it happens. Critics point to the [Darfur conflict](#) and suggest that if anyone is found guilty of genocide after the conflict either by prosecutions brought in the International Criminal Court or in an *ad hoc* International Criminal Tribunal, this will confirm this perception.

2.4.By ad hoc tribunals

[Nuon Chea](#), the Khmer Rouge's chief ideologist, before the [Cambodian Genocide Tribunal](#) on 5 December 2011.

All signatories to the CPPCG are required to prevent and punish acts of genocide, both in peace and wartime, though some barriers make this enforcement difficult. In particular, some of the signatories—namely, [Bahrain](#), [Bangladesh](#), [India](#), [Malaysia](#), the [Philippines](#), [Singapore](#), the [United States](#), [Vietnam](#), [Yemen](#), and former [Yugoslavia](#)—signed with the proviso that no claim of genocide could be brought against them at the [International Court of Justice](#) without their consent.^[36] Despite official protests from other signatories (notably [Cyprus](#) and [Norway](#)) on the ethics and legal standing of these reservations, the [immunity](#) from prosecution they grant has been invoked from time to time, as when the United States refused to allow a charge of genocide brought against it by former [Yugoslavia](#) following the 1999 [Kosovo War](#).^[37]

It is commonly accepted that, at least since [World War II](#), genocide has been illegal under [customary international law](#) as [a peremptory norm](#), as well as under [conventional international law](#). Acts of genocide are generally difficult to establish for prosecution, because a chain of accountability must be established. International criminal courts and tribunals function primarily because the states involved are incapable or unwilling to prosecute crimes of this magnitude themselves.

2.5. Nuremberg Tribunal (1945–1946)

Because the universal acceptance of [international laws](#), defining and forbidding genocide was achieved in 1948, with the promulgation of the *Convention on the Prevention and Punishment of the Crime of Genocide* (CPPCG), those criminals who were prosecuted after the war in international courts, for taking part in the Holocaust were found guilty of [crimes against humanity](#) and other more specific crimes like murder. Nevertheless the Holocaust is universally recognized to have been a genocide and the term, that had been coined the year before by [Raphael Lemkin](#),^[38] appeared in the [indictment of the 24 Nazi leaders](#), Count 3, which stated that all the defendants had "conducted deliberate and systematic genocide—namely, the extermination of racial and national groups..."^[39]

2.6. International Criminal Tribunal for the Former Yugoslavia (1993 to present)

The term *Bosnian Genocide* is used to refer either to [the genocide](#) committed by Serb forces in [Srebrenica](#) in 1995,^[40] or to ethnic cleansing that took place during the 1992–1995 [Bosnian War](#) (an interpretation rejected by a majority of scholars).^[41]

In 2001, the [International Criminal Tribunal for the Former Yugoslavia](#) (ICTY) judged that the 1995 [Srebrenica massacre](#) was an act of genocide.^[42]

On 26 February 2007, the [International Court of Justice](#) (ICJ), in the [Bosnian Genocide Case](#) upheld the ICTY's earlier finding that the Srebrenica massacre in Srebrenica and Zepa constituted genocide, but found that the Serbian government had not participated in a wider genocide on the territory of Bosnia and Herzegovina during the war, as the Bosnian government had claimed.^[43]

On 12 July 2007, [European Court of Human Rights](#) when dismissing the appeal by [Nikola Jorgić](#) against his conviction for genocide by a German court ([Jorgic v. Germany](#)) noted that the German courts wider interpretation of genocide has since been rejected by international courts considering similar cases.^{[44][45][46]} The ECHR also noted that in the 21st century "Amongst scholars, the majority have taken the view that [ethnic cleansing](#), in the way in which it was carried out by the Serb forces in Bosnia and Herzegovina in order to expel Muslims and Croats from their homes, did not constitute genocide. However, there are also a considerable number of scholars who have suggested that these acts did amount to genocide, and the ICTY has found in the Momcilo Krajisnik case that the actus reus, of genocide was met in Prijedor "With regard to the charge of genocide, the Chamber found that in spite of evidence of acts perpetrated in the municipalities which constituted the actus reus of genocide".^[47]

About 30 people have been indicted for participating in genocide or complicity in genocide during the early 1990s in [Bosnia](#). To date, after several [plea bargains](#) and some convictions that were successfully challenged on appeal two men, [Vujadin Popović](#) and [Ljubiša Beara](#), have been found guilty of committing genocide, [Zdravko Tolimir](#) has been found guilty of committing genocide and conspiracy to commit genocide, and two others, [Radislav Krstić](#) and Drago Nikolić, have been found guilty of aiding and abetting genocide. Three others have been found guilty of participating in genocides in Bosnia by German courts, one of whom [Nikola Jorgić](#) lost an appeal against his conviction in the [European Court of Human Rights](#). A further eight men, former members of the Bosnian Serb security forces were found guilty of genocide by the [State Court of Bosnia and Herzegovina](#) (See [List of Bosnian genocide prosecutions](#)).

[Slobodan Milošević](#), as the former President of Serbia and of Yugoslavia, was the most senior political figure to stand trial at the ICTY. He died on 11 March 2006 during his trial where he was accused of genocide or complicity in genocide in territories within Bosnia and Herzegovina, so no verdict was returned. In 1995, the ICTY issued a warrant for the arrest of Bosnian Serbs [Radovan Karadžić](#) and [Ratko Mladić](#) on several charges including genocide. On 21 July 2008, Karadžić was arrested in Belgrade, and he is currently in The Hague on trial accused of genocide among other crimes.^[48] Ratko Mladić was arrested on 26 May 2011 by Serbian special police in Lazarevo, Serbia.^[49]

2.7. International Criminal Tribunal for Rwanda

The [International Criminal Tribunal for Rwanda](#) (ICTR) is a court under the auspices of the United Nations for the prosecution of offenses committed in [Rwanda](#) during the [genocide which occurred there](#) during April 1994, commencing on 6 April. The ICTR was created on 8 November 1994 by the Security Council of the United Nations in order to judge those people responsible for the acts of genocide and other serious violations of the international law performed in the territory of Rwanda, or by Rwandan citizens in nearby states, between 1 January and 31 December 1994.

So far, the ICTR has finished nineteen trials and convicted twenty seven accused persons. On 14 December 2009 two more men were accused and convicted for their crimes. Another twenty five

persons are still on trial. Twenty-one are awaiting trial in detention, two more added on 14 December 2009. Ten are still at large.^[50] The first trial, of [Jean-Paul Akayesu](#), began in 1997. In October 1998, Akayesu was sentenced to life imprisonment. [Jean Kambanda](#), interim Prime Minister, pled guilty.

2.8. Extraordinary Chambers in the Courts of Cambodia

The [Khmer Rouge](#), led by [Pol Pot](#), [Ta Mok](#) and other leaders, organized the mass killing of ideologically suspect groups. The total number of victims is estimated at approximately 1.7 million [Cambodians](#) between 1975–1979, including deaths from slave labour.^[51]

On 6 June 2003 the Cambodian government and the United Nations reached an agreement to set up the [Extraordinary Chambers in the Courts of Cambodia](#) (ECCC) which would focus exclusively on crimes committed by the most senior [Khmer Rouge](#) officials during the period of Khmer Rouge rule of 1975–1979.^[52] The judges were sworn in early July 2006.^{[53][54][55]}

The genocide charges related to killings of Cambodia's [Vietnamese](#) and [Cham](#) minorities, which is estimated to make up tens of thousand killings and possibly more^{[56][57]}

The investigating judges were presented with the names of five possible suspects by the prosecution on 18 July 2007.^{[53][58]}

- [Kang Kek Iew](#) was formally charged with war crime and [crimes against humanity](#) and detained by the Tribunal on 31 July 2007. He was indicted on charges of war crimes and crimes against humanity on 12 August 2008.^[59] His appeal against his conviction for war crimes and crimes against humanity was rejected on 3 February 2012, and he is serving a sentence of life imprisonment.^[60]
- [Nuon Chea](#), a former prime minister, who was indicted on charges of genocide, war crimes, crimes against humanity and several other crimes under Cambodian law on 15 September 2010. He was transferred into the custody of the ECCC on 19 September 2007. His trial, which is ongoing, started on 27 June 2011.^{[61][62]}
- [Khieu Samphan](#), a former head of state, who was indicted on charges of genocide, war crimes, crimes against humanity and several other crimes under Cambodian law on 15 September 2010. He was transferred into the custody of the ECCC on 19 September 2007. His trial, which is ongoing, started on 27 June 2011.^{[61][62]}
- [Ieng Sary](#), a former foreign minister, who was indicted on charges of genocide, war crimes, crimes against humanity and several other crimes under Cambodian law on 15 September 2010. He was transferred into the custody of the ECCC on 12 November 2007. His trial, which is ongoing, started on 27 June 2011.^{[61][62]}
- [Ieng Thirith](#), a former minister for social affairs and wife of Ieng Sary, who was indicted on charges of genocide, war crimes, crimes against humanity and several other crimes under

Cambodian law on 15 September 2010. She was transferred into the custody of the ECCC on 12 November 2007. Proceedings against her have been suspended pending a health evaluation.^{[62][63]}

There has been disagreement between some of the international jurists and the Cambodian government over whether any other people should be tried by the Tribunal.^[58]

2.9. By the International Criminal Court

Since 2002, the International Criminal Court can exercise its jurisdiction if national courts are unwilling or unable to investigate or prosecute genocide, thus being a "court of last resort," leaving the primary responsibility to exercise jurisdiction over alleged criminals to individual states. Due to the [United States concerns over the ICC](#), the United States prefers to continue to use specially convened international tribunals for such investigations and potential prosecutions.^[64]

2.10. Darfur, Sudan

There has been much debate over categorizing the situation in Darfur as genocide.^[65] The on-going conflict in [Darfur](#), Sudan, which started in 2003, was declared a "genocide" by [United States Secretary of State Colin Powell](#) on 9 September 2004 in testimony before the [Senate Foreign Relations Committee](#).^[66] Since that time however, no other permanent member of the UN Security Council followed suit. In fact, in January 2005, an International Commission of Inquiry on Darfur, authorized by [UN Security Council Resolution 1564](#) of 2004, issued a report to the Secretary-General stating that "the Government of the Sudan has not pursued a policy of genocide."^[67] Nevertheless, the Commission cautioned that "The conclusion that no genocidal policy has been pursued and implemented in Darfur by the Government authorities, directly or through the militias under their control, should not be taken in any way as detracting from the gravity of the crimes perpetrated in that region. International offences such as the crimes against humanity and war crimes that have been committed in Darfur may be no less serious and heinous than genocide."^[67]

In March 2005, the Security Council formally referred the situation in Darfur to the Prosecutor of the International Criminal Court, taking into account the Commission report but without mentioning any specific crimes.^[68] Two permanent members of the Security Council, the United States and [China](#), abstained from the vote on the referral resolution.^[69] As of his fourth report to the Security Council, the Prosecutor has found "reasonable grounds to believe that the individuals identified [in the [UN Security Council Resolution 1593](#)] have committed crimes against humanity and war crimes," but did not find sufficient evidence to prosecute for genocide.^[70]

In April 2007, the Judges of the ICC issued arrest warrants against the former Minister of State for the Interior, [Ahmad Harun](#), and a Militia [Janjaweed](#) leader, [Ali Kushayb](#), for crimes against humanity and war crimes.^[71]

On 14 July 2008, prosecutors at the [International Criminal Court](#) (ICC), filed ten charges of [war crimes](#) against Sudan's President [Omar al-Bashir](#): three counts of genocide, five of [crimes against](#)

[humanity](#) and two of murder. The ICC's prosecutors claimed that al-Bashir "masterminded and implemented a plan to destroy in substantial part" three tribal groups in Darfur because of their ethnicity.

On 4 March 2009, the ICC issued a warrant of arrest for Omar Al Bashir, President of Sudan as the ICC Pre-Trial Chamber I concluded that his position as head of state does not grant him immunity against prosecution before the ICC. The warrant was for war crimes and crimes against humanity. It did not include the crime of genocide because the majority of the Chamber did not find that the prosecutors had provided enough evidence to include such a charge.^[72]

Naked [Soviet POWs](#) in [Mauthausen concentration camp](#). "... the murder of at least 3.3 million Soviet POWs is one of the least-known of modern genocides; there is still no full-length book on the subject in English." —Adam Jones^[73]

The preamble to the [CPPCG](#) states that "genocide is a crime under international law, contrary to the spirit and aims of the United Nations and condemned by the civilized world," and that "at all periods of history genocide has inflicted great losses on humanity."

In many cases where accusations of genocide have circulated, partisans have fiercely disputed such an interpretation and the details of the event. This often leads to the promotion of vastly different versions of the event in question.

[Revisionist attempts](#) to challenge or affirm claims of genocide are illegal in some countries. For example, several European countries ban denying [the Holocaust](#), while in Turkey it is illegal to refer to [mass killings of Armenians](#), [Greeks](#) and [Assyrians](#) by the Ottoman Empire toward the end of the First World War as a genocide.^[74]

The 'Age of Totalitarianism' included nearly all of the infamous examples of genocide in modern history, headed by the Jewish Holocaust, but also comprising the mass murders and purges of the Communist world, other mass killings carried out by Nazi Germany and its allies, and also the Armenian genocide of 1915. All these slaughters, it is argued here, had a common origin, the collapse of the elite structure and normal modes of government of much of central, eastern and southern Europe as a result of the First World War, without which surely neither Communism nor Fascism would have existed except in the minds of unknown agitators and crackpots.

— William Rubinstein, *Genocide: a history*^[75]

2.11. Stages of genocide, influences leading to genocide, and efforts to prevent it

For genocide to happen, there must be certain preconditions. Foremost among them is a national culture that does not place a high value on human life. A [totalitarian](#) society, with its assumed superior ideology, is also a precondition for genocidal acts.^[76] In addition, members of the dominant society must perceive their potential victims as less than fully human: as "pagans," "savages," "uncouth barbarians," "unbelievers," "effete degenerates," "ritual outlaws," "racial inferiors," "class antagonists," "counterrevolutionaries," and so on.^[77] In themselves, these conditions are not enough for the

perpetrators to commit genocide. To do that—that is, to commit genocide—the perpetrators need a strong, centralized authority and bureaucratic organization as well as pathological individuals and criminals. Also required is a campaign of vilification and dehumanization of the victims by the perpetrators, who are usually new states or new regimes attempting to impose conformity to a new ideology and its model of society.^[76]

— M. Hassan Kakar^[78]

In 1996 [Gregory Stanton](#), the president of [Genocide Watch](#), presented a briefing paper called "The 8 Stages of Genocide" at the [United States Department of State](#).^[79] In it he suggested that genocide develops in eight stages that are "predictable but not inexorable".^{[79][80]}

The Stanton paper was presented to the State Department, shortly after the Rwandan Genocide and much of its analysis is based on why that genocide occurred. The preventative measures suggested, given the briefing paper's original target audience, were those that the United States could implement directly or indirectly by using its influence on other governments.

Stage	Characteristics	Preventive measures
1. Classification	People are divided into "us and them".	"The main preventive measure at this early stage is to develop universalistic institutions that transcend ... divisions."
2. Symbolization	"When combined with hatred, symbols may be forced upon unwilling members of pariah groups..."	"To combat symbolization, hate symbols can be legally forbidden as can hate speech ".
3. Dehumanization	"One group denies the humanity of the other group. Members of it are equated with animals, vermin, insects, or diseases."	"Local and international leaders should condemn the use of hate speech and make it culturally unacceptable. Leaders who incite genocide should be banned from international travel and have their foreign finances frozen."
4. Organization	"Genocide is always organized... Special army units or militias are often trained and armed..."	"The U.N. should impose arms embargoes on governments and citizens of countries involved in genocidal massacres , and create

		commissions to investigate violations"
5. Polarization	"Hate groups broadcast polarizing propaganda..."	"Prevention may mean security protection for moderate leaders or assistance to human rights groups...Coups d'état by extremists should be opposed by international sanctions."
6. Preparation	"Victims are identified and separated out because of their ethnic or religious identity..."	"At this stage, a Genocide Emergency must be declared. ..."
7. Extermination	"It is 'extermination' to the killers because they do not believe their victims to be fully human".	"At this stage, only rapid and overwhelming armed intervention can stop genocide. Real safe areas or refugee escape corridors should be established with heavily armed international protection."
8. Denial	"The perpetrators... deny that they committed any crimes..."	"The response to denial is punishment by an international tribunal or national courts"

In April 2012, it was reported that Stanton would soon be officially adding two new stages, Discrimination and Persecution, to his original theory, which would make for a 10-stage theory of genocide.^[81]

In a paper for the [Social Science Research Council](#) Dirk Moses criticises the Stanton approach concluding:

In view of this rather poor record of ending genocide, the question needs to be asked why the "genocide studies" paradigm cannot predict and prevent genocides with any accuracy and reliability. The paradigm of "genocide studies," as currently constituted in North America in particular, has both strengths and limitations. While the moral fervor and public activism is admirable and salutary, the paradigm appears blind to its own implication in imperial projects that are themselves as much part of the problem as they are part of the solution. The US government called Darfur a genocide to appease domestic lobbies, and because the statement cost it nothing. Darfur will end when it suits the great powers that have a stake in the region.

— Dirk Moses^[82]

Other authors have focused on the structural conditions leading up to genocide and the psychological and social processes that create an evolution toward genocide. [Helen Fein](#)^[83] showed that pre-existing [anti-Semitism](#) and systems that maintained anti-Semitic policies were related to the number of Jews killed in different European countries during the Holocaust. Ervin Staub showed that economic deterioration and political confusion and disorganization were starting points of increasing discrimination and violence in many instances of genocides and mass killing. They lead to scapegoating a group and ideologies that identified that group as an enemy. A history of devaluation of the group that becomes the victim, past violence against the group that becomes the perpetrator leading to psychological wounds, [authoritarian](#) cultures and political systems, and the passivity of internal and external witnesses (bystanders) all contribute to the probability that the violence develops into genocide.^[84] Intense conflict between groups that is unresolved, becomes intractable and violent can also lead to genocide. The conditions that lead to genocide provide guidance to early prevention, such as humanizing a devalued group, creating ideologies that embrace all groups, and activating bystander responses. There is substantial research to indicate how this can be done, but information is only slowly transformed into action.^[85]

Part III. Posttraumatic Stress Disorder (PTSD)

3.1. Terminology

The term **post-traumatic stress disorder (PTSD)** was coined in the mid-1970s,^[202] in part through the efforts of anti-Vietnam War activists and the antiwar group [Vietnam Veterans Against the War](#) and [Chaim F. Shatan](#), who worked with them and coined the term *post-Vietnam syndrome*; the condition was added to the [DSM-III](#) as **posttraumatic stress disorder** despite the odd double "t".^[205]

Early in 1978, the term was used in a working group finding presented to the Committee of Reactive Disorders.^[205] The term was formally recognized in 1980.^[202]

In the [DSM-IV](#), the spelling "posttraumatic stress disorder" is used, while in the [ICD-10](#), the spelling is "post-traumatic stress disorder".^[206]

Posttraumatic stress disorder (PTSD) may develop after a person is exposed to one or more traumatic events, such as [sexual assault](#), [warfare](#), [serious injury](#), or threats of imminent [death](#).^[1] The [diagnosis](#) may be given when a group of symptoms, such as disturbing recurring flashbacks, avoidance or numbing of memories of the event, and [hyperarousal](#), continue for more than a month after the occurrence of a traumatic event.^[1]

Most people having experienced a traumatizing event will not develop PTSD.^[2] Women are more likely to experience higher impact events, and are also more likely to develop PTSD than men.^[3] Children are less likely to experience PTSD after trauma than adults, especially if they are under ten years of age.^[2] [War veterans](#) are commonly at risk for PTSD.

3.2. Classification

Posttraumatic stress disorder is classified as an [anxiety disorder](#) in the DSM IV; the characteristic symptoms are not present before exposure to the violently traumatic event. In the typical case, the individual with PTSD persistently avoids all thoughts and emotions, and discussion of the stressor event and may experience amnesia for it. However, the event is commonly relived by the individual through intrusive, recurrent recollections, flashbacks, and nightmares.^[4] The characteristic symptoms are considered acute if lasting less than three months, and chronic if persisting three months or more, and with delayed onset if the symptoms first occur after six months or some years later. PTSD is distinct from the briefer [acute stress disorder](#), and can cause clinical impairment in significant areas of functioning.^{[5][6][7]}

3.3. Causes

PTSD is believed to be caused by the experience of a wide range of traumatic events and, in particular if the trauma is extreme, can occur in persons with no predisposing conditions.^{[9][10]}

Persons considered at risk include, for example, combat military personnel, victims of natural disasters, concentration camp survivors, and victims of violent crime. Individuals frequently experience "[survivor's guilt](#)" for remaining alive while others died. Causes of the symptoms of PTSD are the experiencing or witnessing of a stressor event involving death, serious injury or such threat to the self or others in a situation in which the individual felt intense fear, horror, or powerlessness.^[11] Persons employed in occupations that expose them to violence (such as soldiers) or disasters (such as [emergency service](#) workers) are also at risk.^[11]

Children or adults may develop PTSD symptoms by experiencing [bullying](#) or [mobbing](#).^[12]

3.3.1. Domestic violence

Trauma from domestic violence can predispose an individual to PTSD. Approximately 25% of children exposed to domestic violence can experience PTSD in a study of 337 school age children.^[14] Preliminary research suggests that child abuse may interact with mutations in a stress-related gene to increase the risk of PTSD in adults, in a cross-sectional study of 900 school age children.^[15] However, being exposed to a traumatic experience does not automatically indicate they will develop PTSD.^[5] It has been shown that the intrusive memories, such as flashbacks, nightmares, and the memories themselves, are greater contributors to the biological and psychological dimensions of PTSD than the event itself.^[18] These intrusive memories are mainly characterized by sensory episodes, rather than thoughts. People with PTSD have intrusive re-experiences of traumatic events that lack awareness of context and time. These episodes aggravate and maintain PTSD symptoms, since the individual re-experiences trauma as if it were happening in the present moment.^[19]

Multiple studies show that parental PTSD and other posttraumatic disturbances in parental psychological functioning can, despite a traumatized parent's best efforts, interfere with their response to their child as well as their child's response to trauma. For example, in two studies by Schechter, one of 67 mothers and another of 25 mothers, this was shown to be the case.^[20] Parents with violence-related PTSD may, for example, inadvertently expose their children to developmentally inappropriate violent media due to their need to manage their own emotional dysregulation.^[22] Clinical findings indicate that a failure to provide adequate treatment to children after they suffer a traumatic experience, depending on their vulnerability and the severity of the trauma, will ultimately lead to PTSD symptoms in adulthood.^[23]

3.3.2. Evolutionary psychology

[Evolutionary psychology](#) views different types of fears and reactions caused by fears as [adaptations](#) that may have been useful in the ancestral environment in order to avoid or cope with various threats. In general, [mammals](#) display several defensive behaviors roughly dependent on how close the threat is: avoidance, vigilant immobility, withdrawal, aggressive defense, appeasement, and finally complete frozen immobility (the last possibly to confuse a predator's attack reflex or to simulate a dead and contaminated body). PTSD may correspond to and be caused by overactivation of such fear circuits. Thus, PTSD avoidance behaviors may correspond to mammal avoidance of and withdrawal from

threats. Heightened memory of past threats may increase avoidance of similar situations in the future as well as be a prerequisite for analyzing the past threat and develop better defensive behaviors if the threat should recur. PTSD [hyperarousal](#) may correspond to vigilant immobility and aggressive defense. [Complex posttraumatic stress disorder](#) (and phenomena such as the [Stockholm syndrome](#)) may in part correspond to the appeasement stage and possibly the frozen immobility stage.^{[24][25]}

There may be evolutionary explanations for differences in resilience to traumatic events. Thus, PTSD is rare following traumatic fire that may be explained by events such as forest fires' long being part of the evolutionary history of mammals. On the other hand, PTSD is much more common following modern warfare, which may be explained by modern warfare's being a new development and very unlike the quick inter-group raids that are argued to have characterized the [paleolithic](#).^[26]

3.4. Risk factors

Most people (more than half) will experience at least one traumatizing event in their lifetime.^[36] Men are more likely to experience a traumatic event, but women are more likely to experience the kind of high impact traumatic event that can lead to PTSD, such as interpersonal violence and sexual assault.^[2] Only a minority of people who are traumatized will develop PTSD, but they are more likely to be women. The average risk of developing PTSD after trauma is around 8% for men, while for women it is just over 20%.^[2] The risk is believed to be higher in young urban populations (24%): 13% for men and 30% for women.^[2] Rates of PTSD are higher in combat veterans than other men, with a rate estimated at up to 20% for veterans returning from Iraq and Afghanistan.^[36]

Posttraumatic stress reactions have not been studied as well in children and adolescents as adults.^[2] The rate of PTSD may be lower in children than adults, but in the absence of therapy, symptoms may continue for decades.^[2] One estimate suggests that the proportion of children and adolescents having PTSD in a non-wartorn population in a developed country may be 1% compared to 1.5% to 3% of adults, and much lower below the age of 10 years.^[2]

Predictor models have consistently found that childhood trauma, chronic adversity, and familial stressors increase risk for PTSD as well as risk for biological markers of risk for PTSD after a traumatic event in adulthood.^{[37][38][39][40]} Peritraumatic dissociation in children is a predictive indicator of the development of PTSD later in life.^[28] This effect of childhood trauma, which is not well-understood, may be a marker for both traumatic experiences and attachment problems.^{[41][42]} Proximity to, duration of, and severity of the trauma also make an impact, and interpersonal traumas cause more problems than impersonal ones.^[43]

Quasi-experimental studies have demonstrated a relationship between intrusive thoughts and intentional control responses such that suppression increases the frequency of unwanted intrusive thoughts. These results suggest that suppression of intrusive thoughts may be important in the development and maintenance of PTSD.^[44]

3.4.1. Military experience

Schnurr, Lunney, and Sengupta^[33] identified risk factors for the development of PTSD in [Vietnam veterans](#). The subjects were 68 women and 414 men of whom 88 were white, 63 black, 80 Hispanic, 90 Native Hawaiian, and 93 Japanese American. Among their findings were:

- [Hispanic ethnicity](#), coming from an unstable family, being punished severely during childhood, childhood asocial behavior, and depression as pre-military factors
- War-zone exposure, [peritraumatic dissociation](#), depression as military factors
- Recent stressful life events, post-[Vietnam](#) trauma, and depression as post-military factors

They also identified certain protective factors, such as:

- [Japanese-American](#) ethnicity, high school degree or college education, older age at entry to war, higher socioeconomic status, and a more positive paternal relationship as pre-military protective factors
- Social support at homecoming and current social support as post-military factors.^[45] Other research also indicates the protective effects of social support in averting PTSD or facilitating recovery if it develops.^{[46][47]}

Glass and Jones found early intervention to be a critical preventive measure:^[48]

PTSD symptoms can follow any serious psychological trauma, such as exposure to combat, accidents, torture, disasters, criminal assault and exposure to atrocities or to the sequelae of such extraordinary events. Prisoners of war exposed to harsh treatment are particularly prone to develop PTSD. In their acute presentation these symptoms, which include subsets of a large variety of affective, cognitive, perceptual, emotional and behavioral responses which are relatively normal responses to gross psychological trauma. If persistent, however, they develop a life of their own and may be maintained by inadvertent reinforcement. Early intervention and later avoidance of positive reinforcement (which may be subtle) for such symptoms is a critical preventive measure.

Studies have shown that those prepared for the potential of a traumatic experience are more prepared to deal with the stress of a traumatic experience and therefore less likely to develop PTSD.^[5]

3.4.2. Drug misuse

[Alcohol abuse](#) and [drug abuse](#) commonly co-occur with PTSD.^[49] Recovery from posttraumatic stress disorder or other anxiety disorders may be hindered, or the condition worsened, by medication or substance overuse, abuse, or dependence; resolving these problems can bring about a marked improvement in an individual's mental health status and anxiety levels.^{[50][51]}

[Yohimbine](#) (not considered specifically appropriate for PTSD) increases arousal by increasing release of endogenous norepinephrine and can worsen PTSD symptoms.^[49]

3.4.3. Foster care

In the Casey Family Northwest Alumni Study, conducted in conjunction with researchers from the [Harvard Medical School](#) in Oregon and Washington state, the rate of PTSD in adults who were in [foster care](#) for one year between the ages of 14–18 was found to be higher than that of combat veterans. Up to 25% of those in the study meet the diagnostic criteria for PTSD as compared to 12–13% of Iraq war veterans and 15% of Vietnam War veterans, and a rate of 4% in the general population. The recovery rate for foster home alumni was 28.2% as opposed to 47% in the general population.^{[52][53]}

Dubner and Motta (1999)^[54] found that 60% of children in foster care having experienced sexual abuse had PTSD, and 42% of those having been physically abused met the PTSD criteria. PTSD was also found in 18% of the children not abused. These children may have developed PTSD due to witnessing violence in the home, or as a result of real or perceived parental abandonment.

3.5. Pathophysiology

3.5.1. Neuroendocrinology

PTSD symptoms may result when a traumatic event causes an over-reactive adrenaline response, which creates deep neurological patterns in the brain. These patterns can persist long after the event that triggered the fear, making an individual hyper-responsive to future fearful situations.^{[51][55]} During traumatic experiences the high levels of stress hormones secreted suppress [hypothalamic](#) activity that may be a major factor toward the development of PTSD.^[56]

PTSD causes [biochemical](#) changes in the brain and body that differ from other psychiatric disorders such as major depression. Individuals diagnosed with PTSD respond more strongly to a [dexamethasone suppression test](#) than individuals diagnosed with [clinical depression](#).^{[57][58]}

In addition, most people with PTSD also show a low secretion of [cortisol](#) and high secretion of [catecholamines](#) in [urine](#),^[59] with a [norepinephrine](#)/cortisol ratio consequently higher than comparable non-diagnosed individuals.^[60] This is in contrast to the normative [fight-or-flight response](#), in which both [catecholamine](#) and cortisol levels are elevated after exposure to a stressor.^[61]

Brain [catecholamine](#) levels are high,^[62] and [corticotropin-releasing factor](#) (CRF) concentrations are high.^{[63][64]} Together, these findings suggest abnormality in the [hypothalamic-pituitary-adrenal \(HPA\) axis](#).

The HPA axis is responsible for coordinating the hormonal response to stress.^[28] Given the strong cortisol suppression to [dexamethasone](#) in PTSD, HPA axis abnormalities are likely predicated on strong negative feedback inhibition of cortisol, itself likely due to an increased sensitivity of [glucocorticoid receptors](#).^[65]

Translating this reaction to human conditions gives a pathophysiological explanation for PTSD by a maladaptive learning pathway to fear response through a hypersensitive, hyperreactive, and hyperresponsive HPA axis.^[66]

Low [cortisol](#) levels may predispose individuals to PTSD: Following war trauma, [Swedish](#) soldiers serving in [Bosnia and Herzegovina](#) with low pre-service salivary cortisol levels had a higher risk of reacting with PTSD symptoms, following war trauma, than soldiers with normal pre-service levels.^[67] Because cortisol is normally important in restoring [homeostasis](#) after the stress response, it is thought that trauma survivors with low cortisol experience a poorly contained—that is, longer and more distressing—response, setting the stage for PTSD.

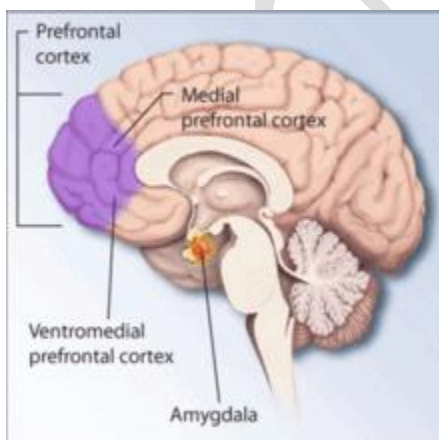
Other studies indicate that people that suffer from PTSD have chronically low levels of [serotonin](#), which contributes to the commonly associated behavioral symptoms such as anxiety, ruminations, irritability, aggression, suicidality, and impulsivity.^[18] Serotonin also contributes to the stabilization of glucocorticoid production.

[Dopamine](#) levels in patients with PTSD can help contribute to the symptoms associated. Low levels of dopamine can contribute to [anhedonia](#), [apathy](#), [impaired attention](#), and motor deficits. Increased levels of dopamine can cause [psychosis](#), [agitation](#), and restlessness.^[18]

Hyperresponsiveness in the norepinephrine system can be caused by continued exposure to high stress. Overactivation of norepinephrine receptors in the prefrontal cortex can be connected to the flashbacks and nightmares frequently experienced by those with PTSD. A decrease in other norepinephrine functions (awareness of the current environment) prevents the memory mechanisms in the brain from processing that the experience, and emotions the person is experiencing during a flashback are not associated with the current environment.^[18]

However, there is considerable controversy within the medical community regarding the neurobiology of PTSD. A review of existing studies on this subject showed no clear relationship between cortisol levels and PTSD. However, the majority of reports indicate people with PTSD have elevated levels of [corticotropin-releasing hormone](#), lower basal [cortisol](#) levels, and enhanced negative feedback suppression of the HPA axis by [dexamethasone](#).^{[28][68]}

3.5.2. Neuroanatomy



Regions of the brain associated with stress and posttraumatic stress disorder^[69]

Three areas of the brain in which function may be altered in PTSD have been identified: the [prefrontal cortex](#), [amygdala](#), and [hippocampus](#). Much of this research has utilised PTSD victims from the Vietnam War. For example, a prospective study using the Vietnam Head Injury Study showed that damage to the prefrontal cortex may actually be protective against later development of PTSD.^[70] In a study by Gurvits et al., combat veterans of the [Vietnam War](#) with PTSD showed a 20% reduction in the volume of their [hippocampus](#) compared with veterans having suffered no such symptoms.^[71] This finding could not be replicated in chronic PTSD patients traumatized at an [air show plane crash in 1988](#) (Ramstein, Germany).^[72]

In human studies, the amygdala has been shown to be strongly involved in the formation of emotional memories, especially fear-related memories. [Neuroimaging](#) studies in humans have revealed both morphological and functional aspects of PTSD.^[73] However, during high stress times the [hippocampus](#), which is associated with the ability to place memories in the correct context of space and time, and with the ability to recall the memory, is suppressed. This suppression is hypothesized to be the cause of the [flashbacks](#) that often plague PTSD patients. When someone with PTSD undergoes [stimuli](#) similar to the traumatic event, the body perceives the event as occurring again because the memory was never properly recorded in the patients memory.^[28]

The amygdalocentric model of PTSD proposes that it is associated with hyperarousal of the amygdala and insufficient top-down control by the medial [prefrontal cortex](#) and the [hippocampus](#) in particular during extinction.^[74] This is consistent with an interpretation of PTSD as a syndrome of deficient [extinction](#) ability.^{[74][75]} A study at the European Neuroscience Institute-Goettingen (Germany) found that fear extinction-induced [IGF2/IGFBP7](#) signalling promotes the survival of 17–19-day-old newborn hippocampal neurons. This suggests that therapeutic strategies that enhance IGF2 signalling and adult [neurogenesis](#) might be suitable to treat diseases linked to excessive fear memory such as [PTSD](#).^[76] Further animal and clinical research into the amygdala and [fear conditioning](#) may suggest additional treatments for the condition.

The maintenance of the fear involved with PTSD has been shown to include the HPA axis, the [locus coeruleus-noradrenergic](#) systems, and the connections between the [limbic system](#) and [frontal cortex](#). The HPA axis that coordinates the hormonal response to stress,^[77] which activates the LC-noradrenergic system, is implicated in the over-consolidation of memories that occurs in the aftermath of trauma.^[78] This over-consolidation increases the likelihood of one's developing PTSD. The [amygdala](#) is responsible for threat detection and the conditioned and unconditioned fear responses that are carried out as a response to a threat.^[28]

The [LC-noradrenergic](#) system has been hypothesized to mediate the over-consolidation of fear memory in PTSD. High levels of [cortisol](#) reduce noradrenergic activity, and because patients with PTSD tend to have reduced levels of cortisol, it is proposed that individuals with PTSD fail to regulate the increased noradrenergic response to traumatic stress.^[79] It is thought that the intrusive memories and conditioned fear responses to associated triggers is a result of this response. [Neuropeptide Y](#) has been reported to reduce the release of [norepinephrine](#) and has been demonstrated to have [anxiolytic](#) properties in animal models. Studies have shown people with PTSD demonstrate reduced levels of NPY, possibly indicating their increased anxiety levels.^[28]

The [basolateral](#) nucleus (BLA) of the amygdala is responsible for the comparison and development of associations between unconditioned and conditioned responses to stimuli, which results in the fear conditioning present in PTSD. The BLA activates the [central nucleus](#) (CeA) of the amygdala, which elaborates the fear response, (including behavioral response to threat and elevated startle response). Descending inhibitory inputs from the [medial prefrontal cortex](#) (mPFC) regulate the transmission from the BLA to the CeA, which is hypothesized to play a role in the extinction of conditioned fear responses.^[28]

3.6. Diagnosis

3.6.1. Diagnostic and Statistical Manual

The diagnostic criteria for PTSD, stipulated in the [Diagnostic and Statistical Manual of Mental Disorders IV \(Text Revision\)](#) (DSM-IV-TR), may be summarized as:^{[4][80]}

A: Exposure to a traumatic event. This must have involved *both* (a) loss of "physical integrity", or risk of serious injury or death, to self or others, and (b) a response to the event that involved intense fear, horror, or helplessness (or in children, the response must involve disorganized or agitated behavior). (The [DSM-IV-TR](#) criterion differs substantially from the previous DSM-III-R stressor criterion, which specified the traumatic event should be of a type that would cause "significant symptoms of distress in almost anyone," and that the event was "outside the range of usual human experience."^[81])

B: Persistent re-experiencing. One or more of these must be present in the victim: [flashback](#) memories, recurring distressing dreams, subjective re-experiencing of the traumatic event(s), or intense negative psychological or physiological response to any objective or subjective reminder of the traumatic event(s).

C: Persistent avoidance and emotional numbing. This involves a sufficient level of:

- avoidance of stimuli associated with the trauma, such as certain thoughts or feelings, or talking about the event(s)
- avoidance of behaviors, places, or people that might lead to distressing memories as well as the disturbing memories, dreams, flashbacks, and intense psychological or physiological distress^[18]
- inability to recall major parts of the trauma(s), or decreased involvement in significant life activities
- decreased capacity (down to complete inability) to feel certain feelings
- an expectation that one's future will be somehow constrained in ways not normal to other people.

D: Persistent symptoms of increased arousal not present before. These are all physiological response issues, such as difficulty falling or staying asleep, or problems with anger, concentration, or [hypervigilance](#). Additional symptoms include irritability, angry outbursts, increased startle response, and concentration or sleep problems.^[18]

E: Duration of symptoms for more than 1 month. If all other criteria are present, but 30 days have not elapsed, the individual is diagnosed with [acute stress disorder](#).^[18]

F: **Significant impairment.** The symptoms reported must lead to "clinically significant distress or impairment" of major domains of life activity, such as social relations, occupational activities, or other "important areas of functioning".^[82]

Assessment

Since the introduction of [DSM-IV](#), the number of possible events that might be used to diagnose PTSD has increased; one study suggests that the increase is around 50%.^[83] Various scales to measure the severity and frequency of PTSD symptoms exist.^{[84][85]} Standardized screening tools such as [Trauma Screening Questionnaire](#)^[86] and [PTSD Symptom Scale](#)^[87] can be used to detect possible symptoms of posttraumatic stress disorder and suggest the need for a formal diagnostic assessment.

DSM-5

In [DSM-5](#), published in May, 2013, PTSD is classified as a trauma- and stress-related disorder.^[1]

- Criterion A: (applicable to adults, adolescents and children over 6. There is a separate Posttraumatic stress disorder for children 6 years and younger.) Exposure to real or threatened death, injury, or sexual violence.
- Several items in Criterion B (intrusion symptoms) are rewritten to add or augment certain distinctions now considered important.
- Special consideration is given to developmentally appropriate criteria for use with children and adolescents. This is especially evident in the restated Criterion B—intrusion symptoms. Development of age-specific criteria for diagnosis of PTSD is ongoing at this time.
- Criterion C (avoidance and numbing) has been split into "C" and "D":
 - Criterion C (new version) now focuses solely on avoidance of behaviors or physical or temporal reminders of the traumatic experience(s). What were formerly two symptoms are now three, due to slight changes in descriptions.
 - New Criterion D focuses on negative alterations in cognition and mood associated with the traumatic event(s) and contains two new symptoms, one expanded symptom, and four largely unchanged symptoms specified in the previous criteria.
- Criterion E (formerly "D"), which focuses on increased arousal and reactivity, contains one modestly revised, one entirely new, and four unchanged symptoms.
- Criterion F (formerly "E") still requires duration of symptoms to have been at least one month.
- Criterion G (formerly "F") stipulates symptom impact ("disturbance") in the same way as before.
- Criterion H stipulated the disturbance is not due to the effects of a substance or another medical condition.

Specify whether:

With dissociative symptoms: (not due to effects of a substance or another medical condition)

1. In addition, meets the criteria of [Depersonalization](#)
2. In addition, meets the criteria of [Derealization](#)

Specify if:

With delayed expression Full criteria not met until more than 6 months after the event

Research-based groups

Emerging [factor analytic](#) research^[88] suggests that PTSD symptoms group empirically into four clusters, not the three currently described in the [Diagnostic and Statistical Manual of Mental Disorders](#).^[dated info] One model supported by this research divides the traditional avoidance symptoms into a cluster of numbing symptoms (such as loss of interest and feeling emotionally numb) and a cluster of behavioral avoidance symptoms (such as avoiding reminders of the trauma).^[89] An alternative model adds a fourth cluster of dysphoric symptoms. These include symptoms of emotional numbing, as well as anger, sleep disturbance, and difficulty concentrating (traditionally grouped under the hyperarousal cluster).^{[90][91]} A literature review^[92] and meta-analysis^[93] did not find strong support across the literature for one of these models over the other.

3.6.2. International Classification of Diseases

The diagnostic criteria for PTSD, stipulated in the [International Statistical Classification of Diseases and Related Health Problems](#) 10 (ICD-10), may be summarized as:^[94]

- Exposure to a stressful event or situation (either short or long lasting) of exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone.
- Persistent remembering or "reliving" the stressor by intrusive flash backs, vivid memories, recurring dreams, or by experiencing distress when exposed to circumstances resembling or associated with the stressor.
- Actual or preferred avoidance of circumstances resembling or associated with the stressor (not present before exposure to the stressor).
- Either (1) or (2):
 1. Inability to recall, either partially or completely, some important aspects of the period of exposure to the stressor
 2. Persistent symptoms of increased psychological sensitivity and arousal (not present before exposure to the stressor) shown by any two of the following:
 - difficulty in falling or staying asleep
 - irritability or outbursts of anger
 - difficulty in concentrating
 - hyper-vigilance
 - exaggerated startle response.

The [International Statistical Classification of Diseases and Related Health Problems](#) 10 diagnostic guidelines state:^[94] In general, this disorder should not be diagnosed unless there is evidence that it arose within 6 months of a traumatic event of exceptional severity. A "probable" diagnosis might still be

possible if the delay between the event and the onset was longer than 6 months, provided that the clinical manifestations are typical and no alternative identification of the disorder (e.g., as an anxiety or obsessive-compulsive disorder or depressive episode) is plausible. In addition to evidence of trauma, there must be a repetitive, intrusive recollection or re-enactment of the event in memories, daytime imagery, or dreams. Conspicuous emotional detachment, numbing of feeling, and avoidance of stimuli that might arouse recollection of the trauma are often present but are not essential for the diagnosis. The autonomic disturbances, mood disorder, and behavioural abnormalities all contribute to the diagnosis but are not of prime importance. The late chronic sequelae of devastating stress, i.e. those manifest decades after the stressful experience, should be classified under [F62.0](#).

3.6.3. Differential Diagnosis

A diagnosis of PTSD requires exposure to an extreme stressor such as one that is life-threatening. Any stressor can result in a diagnosis of [adjustment disorder](#) and it is an appropriate diagnosis for a stressor and a symptom pattern that does not meet the criteria for PTSD, for example a stressor like a partner being fired, or a spouse leaving. If any of the symptom pattern is present before the stressor, another diagnosis is required, such as [brief psychotic disorder](#) or [major depressive disorder](#). Other differential diagnoses are [schizophrenia](#) or other disorders with psychotic features such as Psychotic disorders due to a general medical condition. [Drug-induced psychotic disorders](#) can be considered if substance abuse is involved.^[4]

The symptom pattern for [acute stress disorder](#) must occur and be resolved within four weeks of the trauma. If it lasts longer, and the symptom pattern fits that characteristic of PTSD, the diagnosis may be changed.^[4]

[Obsessive compulsive disorder](#) may be diagnosed for intrusive thoughts that are recurring but not related to a specific traumatic event.^[4]

[Malingering](#) should be considered if a financial and/or legal advantage is a possibility.

3.7. Prevention

Modest benefits have been seen from early access to [cognitive behavioral therapy](#), as well as from some medications such as [propranolol](#).^[95] [Critical incident stress management](#) has been suggested as a means of preventing PTSD, but subsequent studies suggest the likelihood of its producing [iatrogenic](#) outcomes.^{[96][97]} A [review](#) "...did not find any evidence to support the use of an intervention offered to everyone", and that "...multiple session interventions may result in worse outcome than no intervention for some individuals."^[98] The [World Health Organization](#) recommends against the use of [benzodiazepines](#) and [antidepressants](#) in those having experienced trauma.^[99]

3.7.1. Early detection

The ability to prescreen individuals would be of great help in getting treatment to those at risk of PTSD prior to development of the syndrome. Several biological indicators have been identified that are related to later PTSD development. First, Delhanty^[100] found that higher response times and a smaller

[hippocampal](#) volume were identified as linked to later PTSD development. However, both of these indicators are relatively difficult to test for and need specialized tests or equipment, or both, to identify. A blood biomarker is much easier to test for. Van Zuiden et al.^[101] found a biomarker when testing U.S. Army soldiers prior to deployment. They found that soldiers with more glucocorticoid receptors (GR) were more likely to be diagnosed with PTSD six months after deployment. However, higher GR levels have not been identified as a cause of PTSD, and may instead be an intermediary, or even an indicator that the individual has previously experienced traumatic events. There is a great deal of overlap between high GR levels and those later diagnosed with and without PTSD. Thus, the identification of high GR is simply a vulnerability indicator at this time.

Delhanty^[100] found that biological precursors existed directly following traumatic exposure in those that later developed chronic PTSD and were significantly different from those who did not. Directly following the traumatic event, later sufferers often have significantly lower levels of [hypothalamic](#) pituitary-adrenal activity and a corresponding decrease in Cortisol. Other methods of early detection include the identification of specific risk factors associated with later PTSD symptoms. Resnick, Acierno, Holmes, Kilpatrick, and Jager,^[102] for example, were able to identify that the forensic exam given to victims after a rape was associated with PTSD. Finally, global treatments attempt to avoid the problems of early detection by simply treating everyone involved. However, many studies^[100] have found this to be often ineffective and for global treatments to at times increase prevalence rates of PTSD. Stepped collaborative care is where individuals who are at risk are monitored for symptoms.^[95] As symptoms of PTSD appear the level of care is increased to treat those symptoms.

3.7.2. Psychological debriefing

Trauma-exposed individuals often receive a purported preventive treatment called *psychological debriefing*.^[95] Psychological debriefing is the most often used preventive measure, partly because of the relative ease with which this treatment can be given to individuals directly following an event. It consists of interviews that are meant to allow individuals to directly confront the event and share their feelings with the counselor and to help structure their memories of the event. However, research has revealed that psychological debriefing does not help trauma survivors, and it might even hurt them.^[95] Although from the outset, early psychological debriefing research yielded mixed results, some initial research suggested that psychological debriefings helped ameliorate peritraumatic symptoms and prevent post-traumatic symptom development.^[77] But as research progressed, several [meta-analyses](#) made it clear that psychological debriefing is unhelpful and potentially harmful.^{[103][104]} The first Cochrane meta-analysis concerned single-session debriefing. More recently a Cochrane review on multiple session interventions was conducted and also found negative results.^[105] The [American Psychological Association](#) judges the status of psychological debriefing as *No Research Support/Treatment is Potentially Harmful*.^[106]

3.7.3. Risk-targeted interventions

Risk-targeted interventions are those that attempt to mitigate specific formative information or events. It can target modeling normal behaviors, instruction on a task, or giving information on the event. For example,^[102] rape victims were given an instruction video on the procedures for a forensic exam. Also included in the video was advice on how to identify and stop avoidance behavior and control anxiety. Finally, the individuals modeling the forensic exam were shown to be calm and relaxed. PTSD diagnosis

for those having seen the video were 33% less than for those having gone through the standard forensic procedure.

3.7.4. Medications

Some medications have shown benefit in preventing PTSD or reducing its incidence, when given in close proximity to a traumatic event. These medications include:

Alpha-adrenergic agonists: Anecdotal report of success in using [clonidine](#) ("Catapres") to reduce traumatic stress symptoms^[107] suggests that it may have benefit in preventing PTSD.

Beta blockers: [Propranolol](#) ("Inderal"), similar to clonidine, may be useful if there are significant symptoms of "over-arousal". These may inhibit the formation of traumatic memories by blocking adrenaline's effects on the [amygdala](#).^[108]

Glucocorticoids: There is some evidence suggesting that administering [glucocorticoids](#) immediately after a traumatic experience may help prevent PTSD. Several studies have shown that individuals who receive high doses of [hydrocortisone](#) for treatment of [septic shock](#), or following surgery, have a lower incidence and fewer symptoms of PTSD.^{[109][110][111]}

Psychobiological treatments have also found success, especially with cortisol.^[95] Psychobiological treatments target biological changes that occur after a traumatic event. They also attempt to chemically alter learning or memory formation. Cortisol treatments after a traumatic event have found success in mitigating later diagnosis of PTSD. As discussed earlier, cortisol is often lower in individuals at risk of PTSD after a traumatic event than their counterparts. By increasing cortisol levels to normal levels this has been shown to reduce arousal post event as well prevent GR upregulation.

3.8. Management

3.8.1. Psychological

Many forms of psychotherapy have been advocated for trauma-related problems such as PTSD. Basic counseling practices common to many treatment responses for PTSD include education about the condition and provision of safety and support.^{[5][87]}

The psychotherapy programs with the strongest demonstrated efficacy include cognitive behavioral programs, variants of [exposure therapy](#), stress inoculation training (SIT), variants of cognitive therapy (CT), [eye movement desensitization and reprocessing](#) (EMDR),^[73] and many combinations of these procedures.^{[112][113]} A 2010 review disagrees that these treatments have proven efficacy and points out methodological flaws in the studies and previous meta-analyses.^[114]

EMDR and trauma-focused [cognitive behavioral therapy](#) (TFCBT) were recommended as first-line treatments for trauma victims in a 2007 review; however, "the evidence base [for EMDR] was not as strong as that for TFCBT ... Furthermore, there was limited evidence that TFCBT and EMDR were superior to supportive/non-directive treatments, hence it is highly unlikely that their effectiveness is due

to non-specific factors such as attention."^[115] A [meta-analytic](#) comparison of EMDR and [cognitive behavioral therapy](#) found both protocols indistinguishable in terms of effectiveness in treating PTSD; however, "the contribution of the eye movement component in EMDR to treatment outcome" is unclear.^[116]

3.8.2. Cognitive behavioral therapy

[Cognitive behavioral therapy](#) (CBT) seeks to change the way a trauma victim feels and acts by changing the patterns of thinking or behavior, or both, responsible for negative emotions. CBT has been proven to be an effective treatment for PTSD and is currently considered the standard of care for PTSD by the [United States Department of Defense](#).^[117] In CBT, individuals learn to identify thoughts that make them feel afraid or upset and replace them with less distressing thoughts. The goal is to understand how certain thoughts about events cause PTSD-related stress.

Recent research on contextually based third-generation [behavior therapies](#) suggests that they may produce results comparable to some of the better validated therapies.^[118] Many of these therapy methods have a significant element of exposure^[117] and have demonstrated success in treating the primary problems of PTSD and co-occurring depressive symptoms.^[119]

Exposure therapy is a type of cognitive behavioral therapy^[120] that involves assisting trauma survivors to re-experience distressing trauma-related memories and reminders in order to facilitate habituation and successful emotional processing of the trauma memory. Most exposure therapy programs include both imaginal confrontation with the traumatic memories and real-life exposure to trauma reminders; this therapy modality is well supported by clinical evidence. The success of exposure-based therapies has raised the question of whether exposure is a necessary ingredient in the treatment of PTSD.^[121] Some organizations^[which?] have endorsed the need for exposure.^{[122][123]} The US Department of Veterans Affairs has been actively training mental health treatment staff in [prolonged exposure therapy](#)^[124] and [Cognitive Processing Therapy](#)^[125] in an effort to better treat US Veterans with PTSD.

3.8.3. Eye movement desensitization and reprocessing

Eye movement desensitization and reprocessing (EMDR) is a form of psychotherapy developed and studied by [Francine Shapiro](#).^[126] She had noticed that, when she was thinking about disturbing memories herself, her eyes were moving rapidly. When she brought her eye movements under control while thinking, the thoughts were less distressing.^[126]

In 2002, Shapiro and Maxfield published a theory of why this might work, called adaptive information processing.^[127] This theory proposes that eye movement can be used to facilitate emotional processing of memories, changing the person's memory to attend to more adaptive information.^[128] The therapist initiates voluntary rapid eye movements while the person focuses on memories, feelings or thoughts about a particular trauma.^{[2][129]} The therapists uses hand movements to get the person to move their eyes backward and forward, but hand-tapping or tones can also be used.^[2] EMDR closely resembles [cognitive behavioral therapy](#) as it combines exposure (re-visiting the traumatic event), working on cognitive processes and relaxation/self-monitoring.^[2] However, exposure by way of being asked to think about the experience rather than talk about it has been highlighted as one of the more important distinguishing elements of EMDR.^[130]

There have been multiple small controlled trials of four to eight weeks of EMDR in adults^[131] as well as children and adolescents.^[129] EMDR reduced PTSD symptoms enough in the short term that one in two adults no longer met the criteria for PTSD, but the number of people involved in these trials was small.^[131] There was not enough evidence to know whether or not EMDR could eliminate PTSD.^[131] There was some evidence that EMDR might prevent depression.^[131] There were no studies comparing EMDR to other psychological treatments or to medication.^[131] Adverse effects were largely unstudied.^[131] The benefits were greater for women with a history of sexual assault compared with people who had experienced other types of traumatizing events (such as accidents, physical assaults and war). There is a small amount of evidence that EMDR may improve re-experiencing symptoms in children and adolescents, but EMDR has not been shown to improve other PTSD symptoms, anxiety, or depression.^[129]

The eye movement component of the therapy may not be critical for benefit.^{[2][128]} As there has been no major, high quality randomized trial of EMDR with eye movements versus EMDR without eye movements, the controversy over effectiveness is likely to continue.^[130]

3.8.4. Interpersonal psychotherapy

Other approaches, in particular involving social supports,^{[46][47]} may also be important. An open trial of interpersonal psychotherapy^[132] reported high rates of remission from PTSD symptoms without using exposure.^[133] A current, NIMH-funded trial in New York City is now (and into 2013) comparing interpersonal psychotherapy, [prolonged exposure therapy](#), and relaxation therapy.^{[134][135][136]}

3.8.5. Medication

A variety of medications has shown adjunctive benefit in reducing PTSD symptoms,^[137] but "there is no clear drug treatment for PTSD".^[138] In general, positive symptoms (re-experiencing, hypervigilance, increased arousal) respond better to medication than negative symptoms (avoidance, withdrawal), and it is recommended that any drug trial last for at least 6–8 weeks.^[138] With many medications, residual symptoms following treatment is the rule rather than the exception, which has led to increased research in the aggressive treatment of PTSD symptoms.^[139]

Some studies have shown that treatment with [hydrocortisone](#) shortly after a traumatic event, in comparison to a placebo, decreases the likelihood that the patient will suffer from PTSD. Other studies have indicated that [propranolol](#) administered within 6 hours of a traumatic event decreases the physiological reactivity to a reminder of the traumatic event. However propranolol had no effect on the rate of PTSD. Despite these studies, there is not significant evidence that medication can prevent PTSD, therefore none is routinely administered.^[140]

3.8.6. Symptom management

SSRIs ([selective serotonin reuptake inhibitors](#)). SSRIs are considered to be a [first-line](#) drug treatment.^{[141][142]} SSRIs for which there are data to support use include: [citalopram](#), [escitalopram](#),^[143] [fluoxetine](#),^[144] [fluvoxamine](#),^[145] [paroxetine](#),^[146] and [sertraline](#).^{[144][147]}

Among the anti-depressants described in this section, bupropion and [venlafaxine](#) have the lowest patient drop-out rates. Sertraline, [fluoxetine](#), and [nefazodone](#) have a modestly higher drop-out rate (~15%), and the heterocyclics and paroxetine have the highest rates (~20%+).^[148] Where drop-out is caused or feared because of medication side-effects, it should be remembered that most patients do not experience such side-effects.^[149]

Tricyclic antidepressants: [Amitriptyline](#) has shown benefit for positive distress symptoms and for avoidance, and [imipramine](#) has shown benefit for intrusive symptoms.^[144]

Alpha-adrenergic antagonists: [Prazosin](#), in a small study of combat veterans, has shown substantial benefit in relieving or reducing nightmares.^[49] Clonidine can be helpful with startle, hyperarousal, and general autonomic hyperexcitability.^[150]

Anti-convulsants, mood stabilizers, anti-aggression agents: [Carbamazepine](#) has likely benefit in reducing arousal symptoms involving noxious affect,^[144] as well as mood or aggression.^[151] [Topiramate](#)^[49] has been effective in achieving major reductions in flashbacks and nightmares, and no reduction of effect was seen over time.^[49] [Zolpidem](#) has also proven useful in treating sleep disturbances.^[150]

[Lamotrigine](#) may be useful in reducing reexperiencing symptoms, as well as avoidance and emotional numbing.^{[49][152][153][154]} [Valproic acid](#) and has shown reduction of symptoms of irritability, aggression, and impulsiveness, and in reducing flashbacks.^[150] Similarly, [lithium carbonate](#) has worked to control mood and aggressions (but not anxiety) symptoms.^[151] [Buspirone](#) has an effect similar to that of lithium, with the additional benefit of working to reduce hyperarousal symptoms.^[150]

Antipsychotics such as [risperidone](#) can be used to help with [dissociation](#), mood issues, and aggression.^[155]

Serotonin antagonists. [Cyproheptadine](#) can be used to help with sleep disorders and nightmares.^[156]

Atypical **antidepressants:**^[157] Nefazodone can be effective with sleep disturbance symptoms and with secondary depression, [anxiety](#), and [sexual dysfunction](#) symptoms.^[144] [Trazodone](#) can also reduce or eliminate problems with anger, anxiety, and disturbed sleep.^[144]

Beta blockers: [Propranolol](#) has demonstrated possibilities in reducing hyperarousal symptoms, including sleep disturbances.^{[108][150]}

Benzodiazepines: These drugs are not recommended by clinical guidelines for the treatment of PTSD due to a lack of evidence of benefit.^[158] Nevertheless some doctors use benzodiazepines with caution for short-term anxiety relief,^{[155][159]} hyperarousal, and sleep disturbance.^[150] However, some authors believe that the use of benzodiazepines is contraindicated for acute stress, as this group of drugs promotes dissociation and ulterior revivals.^[160] While benzodiazepines can alleviate acute anxiety, there is no consistent evidence that they can stop the development of PTSD, or are at all effective in the *treatment* of posttraumatic stress disorder. Additionally, benzodiazepines may reduce the effectiveness of psychotherapeutic interventions, and there is some evidence that benzodiazepines may actually contribute to the development and chronification of PTSD. Other drawbacks include the risk of

developing a [benzodiazepine dependence](#) and [withdrawal syndrome](#); additionally, individuals with PTSD are at an increased risk of [abusing benzodiazepines](#).^{[141][161]}

Glucocorticoids: In addition, post-stress high-dose [corticosterone](#) administration was recently found to reduce "PTSD-like" behaviors in a rat model of PTSD. In this study, corticosterone impaired memory performance, suggesting that it may reduce risk for PTSD by interfering with consolidation of traumatic memories.^[162] The neurodegenerative effects of the glucocorticoids, however, may prove this treatment counterproductive.^[163]

Monoamine-oxidase inhibitors (MAOIs): [Phenelzine](#) has for some time^[when?] been observed to be effective with hyperarousal and depression and is especially effective with nightmares.^[144]

3.8.7. Medications by symptom group affected

Medications can affect one or more of the symptoms, in one or more of the three major symptom classes^[4] involved in diagnosing PTSD, which can be summarized in the following table.^{[155][159][164]}

Symptom class	Symptom	Medication
<i>Reexperiencing</i>		
	intrusive recall	amitriptyline; fluoxetine; imipramine; lamotrigine; sertraline
	intrusive re-experiencing	amitriptyline; fluoxetine; imipramine; nefazodone; sertraline (women only); topiramate;
	sleep disturbance, nightmares	benzodiazepines; carbamazepine; clonidine; nefazodone; phenelzine; prazosin; topiramate; trazodone; zolpidem
	dissociative recall	risperidone
	intense psychological distress (anger, anxiety) when exposed to reminders of traumatic event(s)	benzodiazepines; buspirone; carbamazepine; lithium (not for anxiety); nefazodone; trazodone

<i>Avoidance</i>		
	avoidance	amitriptyline; fluoxetine; lamotrigine; nefazodone; sertraline
	feelings of detachment or estrangement from others	amitriptyline; risperidone
	restricted range of affect (numbing)	amitriptyline; lamotrigine; sertraline (women only)
<i>Hyperarousal</i>		
	general hyperarousal	amitriptyline; nefazodone; phenelzine; sertraline (women only)
	sleep disturbance, nightmares	benzodiazepines; carbamazepine; clonidine; nefazodone; phenelzine; trazodone; zolpidem
	irritability, anger (and impulsiveness)	carbamazepine; nefazodone; valproic acid
	anger	bupirone; fluoxetine; lithium; trazodone
	aggression	risperidone
	exaggerated startle response; general autonomic hyperexcitability	benzodiazepines; bupirone; carbamazepine; clonidine; propranolol; valproic acid

Some medications can also help with symptoms that may occur secondary to PTSD. [\[164\]](#)

Secondary symptom	Medication
Depression	nefazodone; phenelzine
dream content distortions	nefazodone
relapse of symptoms	carbamazepine
self-mutilation	clonidine; buprenorphine
sexual function reduction	nefazodone
sleep hours reduction	nefazodone

Other

Exercise, sport and physical activity

Physical activity can have an impact on people's psychological wellbeing^[165] and physical health.^[166] The U.S. National Center for PTSD recommends moderate exercise as a way to distract from disturbing emotions, build self-esteem and increase feelings of being in control again. They recommend a discussion with a doctor before starting an exercise program.^[167]

Some uncontrolled studies have found benefits for people with PTSD from exercise programs.^[165] A small trial studied adding a physical component to biofeedback-based CBT with traumatized refugees. The authors concluded that physical activity may lead to clinical improvement, but bigger trials are needed.^[168] More trials are underway.^{[169][170][171]}

Play therapy for children

Play is thought to help children link their inner thoughts with their outer world, connecting real experiences with abstract thought.^[172] Repetitive play can also be one of the ways a child relives traumatic events, and that can be a symptom of traumatization in a child or young person.^[173]

Play is a familiar way for children and young people to indirectly address what worries them, so it is often used as an element of psychological treatment – for example, using play materials or drawing to help a child focus on their feelings and events.^{[2][172]} Play therapy means using games, drawings and play materials to express, understand and control feelings rather than as a means of communication.^{[172][174]} Although it is commonly used, there have not been enough studies comparing outcomes in groups of children receiving and not receiving play therapy, so the effects of play therapy are not yet understood.^{[2][172]}

Research

To recapitulate some of the neurological and neurobehavioral symptoms experienced by the [veteran](#) population of recent conflicts in Iraq and Afghanistan, researchers at the [Roskamp Institute](#) and the James A Haley Veteran's Hospital (Tampa) have developed an animal model to study the consequences of [mild traumatic brain injury](#) (mTBI) and PTSD.^[207] In the laboratory, the researchers exposed mice to a repeated session of unpredictable stressor (i.e. predator odor while restrained), and physical trauma in

the form of inescapable foot-shock, and this was also combined with a mTBI. In this study, PTSD animals demonstrated recall of traumatic memories, anxiety, and an impaired social behavior, while animals subject to both mTBI and PTSD had a pattern of disinhibitory-like behavior. mTBI abrogated both contextual fear and impairments in social behavior seen in PTSD animals. In comparison with other animal studies,^{[207][208]} examination of neuroendocrine and neuroimmune responses in plasma revealed a trend toward increase in corticosterone in PTSD and combination groups.

For CUR students only

REFERENCES

References for Mental health and PTSD

1. American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing. pp. 271–280. [ISBN 978-0-89042-555-8](#).
2. National Collaborating Centre for Mental Health (UK) (2005). ["Post-Traumatic Stress Disorder: The Management of PTSD in Adults and Children in Primary and Secondary Care"](#). *NICE Clinical Guidelines, No. 26*. Gaskell (Royal College of Psychiatrists). [Lay summary – Pubmed Health \(plain English\)](#). 
3. National Collaborating Centre for Mental Health (UK) (2005). ["2: Post-traumatic stress disorder, section 2: Incidence and prevalence"](#). "Post-Traumatic Stress Disorder: The Management of PTSD in Adults and Children in Primary and Secondary Care". *NICE Clinical Guidelines, No. 26*. Gaskell (Royal College of Psychiatrists). [PMID 21834189](#). Retrieved 30 January 2014. [Lay summary – Pubmed Health \(plain English\)](#). 
4. American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders: DSM-IV*. Washington, DC: American Psychiatric Association. [ISBN 0-89042-061-0](#). ^{[[page needed](#)]}, [on-line](#).
5. Rothschild, Babette (2000). *The Body Remembers: The Psychophysiology of Trauma and Trauma Treatment*. New York: W.W. Norton & Company. [ISBN 0-393-70327-4](#). ^{[[page needed](#)]}
6. Kaplan, H. I.; Sadock, B. J. (1994). Grebb, J. A., ed. *Kaplan and Sadock's synopsis of psychiatry: Behavioral sciences, clinical psychiatry* (7th ed.). Baltimore: Williams & Williams. pp. 606–609. ^{[[page needed](#)]}
7. [Satcher D](#) (1999). ["Chapter 4"](#). *Mental Health: A Report of the Surgeon General*. [Surgeon General of the United States](#).
8. Robinson, Maisah (May 27, 2006). ["Review of Francisco Goya's Disasters of War"](#). Associated Press. ^{[[unreliable source?](#)]}
9. ["Post-Traumatic Stress Disorder \(PTSD\)"](#). *U.S. Department of Health and Human Services*. National Institute of Mental Health (NIMH). Retrieved 2011-12-16.
10. Mayo Clinic staff. ["Post-traumatic stress disorder \(PTSD\)"](#). Mayo Foundation for Medical Education and Research. Retrieved 2011-12-16. ^{[[scientific citation needed](#)]}
11. Fullerton CS, Ursano RJ, Wang L (2004). "Acute Stress Disorder, Posttraumatic Stress Disorder, and Depression in Disaster or Rescue Workers". *Am J Psychiatry* **161** (8): 1370–1376. [doi:10.1176/appi.ajp.161.8.1370](#). [PMID 15285961](#).
12. Kelleher I, Harley M, Lynch F, Arseneault L, Fitzpatrick C, Cannon M (November 2008). "Associations between childhood trauma, bullying and psychotic symptoms among a school-based adolescent sample". *Br J Psychiatry* **193** (5): 378–82. [doi:10.1192/bjp.bp.108.049536](#). [PMID 18978317](#). ^{[[non-primary source needed](#)]}
13. ["Are they really out to get your patient?"](#) (PDF). *Current Psychiatry* **8** (4). Archived from [the original](#) on 2010-05-09. ^{[[non-primary source needed](#)]}
14. McCloskey LA, Walker M (2000). "Posttraumatic stress in children exposed to family violence and single-event trauma". *Journal of the American Academy of Child and Adolescent Psychiatry* **39** (1): 108–15. [doi:10.1097/00004583-200001000-00023](#). [PMID 10638074](#). ^{[[non-primary source needed](#)]}
15. Binder EB, Bradley RG, Liu W, Epstein MP, Deveau TC, Mercer KB, Tang Y, Gillespie CF, Heim CM, Nemeroff CB, Schwartz AC, Cubells JF, Ressler KJ (March 2008). ["Association of](#)

- [FKBP5 polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults](#)". *JAMA* **299** (11): 1291–305. doi:10.1001/jama.299.11.1291. PMC 2441757. PMID 18349090.^[non-primary source needed]
16. Peggy Peck, Executive Editor (2008-03-09). "[Genes May Affect Lifelong Impact of Child Abuse](#)". MedPage Today. Archived from [the original](#) on 2014-02-20.
 17. Constance Holden (2008-03-18). "[Seeds of PTSD Planted in Childhood](#)". ScienceNOW Daily News. Archived from [the original](#) on 2008-06-03. (subscription required (help)).
 18. Olszewski TM, Varrasse JF (2005). "The neurobiology of PTSD: implications for nurses". *Journal of Psychosocial Nursing and Mental Health Services* **43** (6): 40–7. PMID 16018133.
 19. Ehlers A, Hackmann A, Michael T (2004). "Intrusive re-experiencing in post-traumatic stress disorder: Phenomenology, theory, and therapy". *Memory* **12** (4): 403–415. doi:10.1080/09658210444000025. PMID 15487537.
 20. Schechter DS, Coates SW, Kaminer T, Coots T, Zeanah CH, Davies M, Schonfeld IS, Marshall RD, Liebowitz MR, Trabka KA, McCaw JE, Myers MM (2008). "[Distorted maternal mental representations and atypical behavior in a clinical sample of violence-exposed mothers and their toddlers](#)". *J Trauma Dissociation* **9** (2): 123–47. doi:10.1080/15299730802045666. PMC 2577290. PMID 18985165.^[non-primary source needed]
 21. Schechter DS, Zygmont A, Coates SW, Davies M, Trabka K, McCaw J, Kolodji A, Robinson J (September 2007). "[Caregiver traumatization adversely impacts young children's mental representations on the MacArthur Story Stem Battery](#)". *Attach Hum Dev* **9** (3): 187–205. doi:10.1080/14616730701453762. PMC 2078523. PMID 18007959.^[non-primary source needed]
 22. Schechter DS, Gross A, Willheim E, McCaw J, Turner JB, Myers MM, Zeanah CH, Gleason MM (December 2009). "[Is maternal PTSD associated with greater exposure of very young children to violent media?](#)". *J Trauma Stress* **22** (6): 658–62. doi:10.1002/jts.20472. PMC 2798921. PMID 19924819.
 23. Clark C, Rodgers B, Caldwell T, Power C, Stansfeld S (June 2007). "[Childhood and adulthood psychological ill health as predictors of midlife affective and anxiety disorders: the 1958 British Birth Cohort](#)". *Arch Gen Psychiatry* **64** (6): 668–78. doi:10.1001/archpsyc.64.6.668. PMID 17548748.
 24. Chris Cantor (2005). [Evolution and posttraumatic stress: disorders of vigilance and defence](#). Routledge. ISBN 978-1-58391-771-8. Retrieved 2014-01-29.
 25. Cantor C, Price J (2007). "Traumatic entrapment, appeasement and complex post-traumatic stress disorder: Evolutionary perspectives of hostage reactions, domestic abuse and the Stockholm syndrome". *Australian and New Zealand Journal of Psychiatry* **41** (5): 377–384. doi:10.1080/00048670701261178. PMID 17464728.
 26. Bracha HS (2006). "Human brain evolution and the "Neuroevolutionary Time-depth Principle": Implications for the Reclassification of fear-circuitry-related traits in DSM-V and for studying resilience to warzone-related posttraumatic stress disorder". *Progress in Neuro-Psychopharmacology and Biological Psychiatry* **30** (5): 827–853. doi:10.1016/j.pnpbp.2006.01.008. PMID 16563589.
 27. True WR, Rice J, Eisen SA, Heath AC, Goldberg J, Lyons MJ, Nowak J (1993). "A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms". *Arch. Gen. Psychiatry* **50** (4): 257–64. doi:10.1001/archpsyc.1993.01820160019002. PMID 8466386.
 28. Skelton K, Ressler KJ, Norrholm SD, Jovanovic T, Bradley-Davino B (2012). "[PTSD and gene variants: New pathways and new thinking](#)". *Neuropharmacology* **62** (2): 628–637. doi:10.1016/j.neuropharm.2011.02.013. PMC 3136568. PMID 21356219.

29. Newton, Phil (16 November 2008). ["A gene for anxiety, depression and posttraumatic stress disorder: FKBP5"](#). Psychology Today. Retrieved 29 November 2011.
30. Binder EB, Bradley RG, Liu W, Epstein MP, Deveau TC, Mercer KB, Tang Y, Gillespie CF, Heim CM, Nemeroff CB, Schwartz AC, Cubells JF, Ressler KJ (2008). ["Association of FKBP5 polymorphisms and childhood abuse with risk of posttraumatic stress disorder symptoms in adults"](#). *JAMA* **299** (11): 1291–305. doi:10.1001/jama.299.11.1291. PMC 2441757. PMID 18349090.
31. Koenen KC, Saxe G, Purcell S, Smoller JW, Bartholomew D, Miller A, Hall E, Kaplow J, Bosquet M, Moulton S, Baldwin C (2005). "Polymorphisms in FKBP5 are associated with peritraumatic dissociation in medically injured children". *Mol Psychiatry* **10** (12): 1058–9. doi:10.1038/sj.mp.4001727. PMID 16088328.
32. Birmes P, Brunet A, Carreras D, Ducassé JL, Charlet JP, Lauque D, Sztulman H, Schmitt L (2003). "The predictive power of peritraumatic dissociation and acute stress symptoms for posttraumatic stress symptoms: a three-month prospective study". *Am J Psychiatry* **160** (7): 1337–9. doi:10.1176/appi.ajp.160.7.1337. PMID 12832251.
33. Schnurr PP, Lunney CA, Sengupta A (2004). "Risk factors for the development versus maintenance of posttraumatic stress disorder". *J Trauma Stress* **17** (2): 85–95. doi:10.1023/B:JOTS.0000022614.21794.f4. PMID 15141781.
34. Yehuda R, Cai G, Golier JA, Sarapas C, Galea S, Ising M, Rein T, Schmeidler J, Müller-Myhsok B, Holsboer F, Buxbaum JD (24 April 2009). "Gene expression patterns associated with posttraumatic stress disorder following exposure to the World Trade Center attacks". *Biol Psychiatry* **66** (7): 708–11. doi:10.1016/j.biopsych.2009.02.034. PMID 19393990.
35. Ressler KJ, Mercer KB, Bradley B, Jovanovic T, Mahan A, Kerley K, Norrholm SD, Kilaru V, Smith AK, Myers AJ, Ramirez M, Engel A, Hammack SE, Toufexis D, Braas KM, Binder EB, May V (February 24, 2011). ["Post-traumatic stress disorder is associated with PACAP and the PAC1 receptor"](#). *Nature* **470** (7335): 492–497. doi:10.1038/nature09856. PMC 3046811. PMID 21350482. Lay summary – SciGuru (March 2, 2011).
36. Spoont, Michele; Arbisi, P; Fu, S; Greer, N; Kehle-Forbes, S; Meis, L; Rutks, I. (January 2013). ["Screening for Post-Traumatic Stress Disorder \(PTSD\) in Primary Care: A Systematic Review"](#). Washington DC: Department of Veterans Affairs. PMID 23487872. Lay summary – Pubmed Health (plain English). 
37. Koenen KC, Moffitt TE, Poulton R, Martin J, Caspi A (February 2007). ["Early childhood factors associated with the development of post-traumatic stress disorder: results from a longitudinal birth cohort"](#). *Psychol Med* **37** (2): 181–92. doi:10.1017/S0033291706009019. PMC 2254221. PMID 17052377.
38. Lapp KG, Bosworth HB, Strauss JL, Stechuchak KM, Horner RD, Calhoun PS, Meador KG, Lipper S, Butterfield MI (September 2005). "Lifetime sexual and physical victimization among male veterans with combat-related post-traumatic stress disorder". *Mil Med* **170** (9): 787–90. PMID 16261985.
39. Otte C, Neylan TC, Pole N, Metzler T, Best S, Henn-Haase C, Yehuda R, Marmar CR (January 2005). "Association between childhood trauma and catecholamine response to psychological stress in police academy recruits". *Biol. Psychiatry* **57** (1): 27–32. doi:10.1016/j.biopsych.2004.10.009. PMID 15607297.
40. Resnick HS, Yehuda R, Pitman RK, Foy DW (November 1995). ["Effect of previous trauma on acute plasma cortisol level following rape"](#). *Am J Psychiatry* **152** (11): 1675–7. PMID 7485635.

41. Laor N, Wolmer L, Mayes LC, Golomb A, Silverberg DS, Weizman R, Cohen DJ (May 1996). "Israeli preschoolers under Scud missile attacks. A developmental perspective on risk-modifying factors". *Arch Gen Psychiatry* **53** (5): 416–23. [doi:10.1001/archpsyc.1996.01830050052008](https://doi.org/10.1001/archpsyc.1996.01830050052008). [PMID 8624185](https://pubmed.ncbi.nlm.nih.gov/8624185/).
42. Laor N, Wolmer L, Mayes LC, Gershon A, Weizman R, Cohen DJ (March 1997). "[Israeli preschool children under Scuds: a 30-month follow-up](#)". *J Am Acad Child Adolesc Psychiatry* **36** (3): 349–56. [doi:10.1097/00004583-199703000-00013](https://doi.org/10.1097/00004583-199703000-00013). [PMID 9055515](https://pubmed.ncbi.nlm.nih.gov/9055515/). (subscription required (help)).
43. Janoff-Bulman, R. (1992). *Shattered Assumptions: Toward a New Psychology of Trauma*. New York: Free Press. [\[page needed\]](#)
44. Falsetti, Sherry A.; Monier, Jeannine; Resnick, Jeannine (2005). "Chapter 2: Intrusive Thoughts In Posttraumatic Stress Disorder". In Clark, David A. *Intrusive Thoughts In Clinical Disorders. Theory, Research, and Treatment*. The Guilford Press. pp. 40–41. [ISBN 1-59385-083-2](#).
45. Price, Jennifer L. "[Findings from the National Vietnam Veterans' Readjustment Study – Factsheet](#)". *United States Department of Veterans Affairs*. National Center for PTSD. Archived from [the original](#) on 2009-04-30.
46. Brewin CR, Andrews B, Valentine JD (October 2000). "[Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults](#)". *J Consult Clin Psychol* **68** (5): 748–66. [doi:10.1037/0022-006X.68.5.748](https://doi.org/10.1037/0022-006X.68.5.748). [PMID 11068961](https://pubmed.ncbi.nlm.nih.gov/11068961/).
47. Ozer EJ, Best SR, Lipsey TL, Weiss DS (January 2003). "[Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis](#)". *Psychol Bull* **129** (1): 52–73. [doi:10.1037/0033-2909.129.1.52](https://doi.org/10.1037/0033-2909.129.1.52). [PMID 12555794](https://pubmed.ncbi.nlm.nih.gov/12555794/).
48. Glass, Albert Julius; Jones, Franklin D. "[Psychiatry In The U.S. Army: Lessons for Community Psychiatry](#)" (PDF).
49. Maxmen, J. S.; Ward, N. G. (2002). *Psychotropic drugs: fast facts* (3rd ed.). New York: W. W. Norton. p. 348. [ISBN 0-393-70301-0](#).
50. Cohen SI (February 1995). "[Alcohol and benzodiazepines generate anxiety, panic and phobias](#)". *J R Soc Med* **88** (2): 73–77. [PMC 1295099](https://pubmed.ncbi.nlm.nih.gov/7769598/). [PMID 7769598](https://pubmed.ncbi.nlm.nih.gov/7769598/).
51. Spates, R.; Souza, T. (2007). "[Treatment of PTSD and Substance Abuse Comorbidity](#)". *The Behavior Analyst Today* **9** (1): 11–26.
52. "[Former Foster Children in Oregon and Washington Suffer Posttraumatic Stress Disorder at Twice the Rate of U.S War Veterans](#)" (PDF) (Press release). Casey Family Programs, Harvard Medical School. 2005-04-05. Archived from [the original](#) on 2005-05-11. Retrieved 2010-03-23.
53. Casey Family Programs (2005). "[Assessing the Effects of Foster Care: Mental Health Outcomes from the Casey National Alumni Study](#)" (PDF). Retrieved 2014-01-29.
54. Dubner AE, Motta RW; Motta, RW (June 1999). "Sexually and physically abused foster care children and posttraumatic stress disorder". *Journal of Consulting and Clinical Psychology* **67** (3): 367–73. [doi:10.1037/0022-006X.67.3.367](https://doi.org/10.1037/0022-006X.67.3.367). [PMID 10369057](https://pubmed.ncbi.nlm.nih.gov/10369057/).
55. *The Secret Life of the Brain (Series), episode 4*. PBS. 2001. Retrieved 2014-01-29.
56. Joseph Zohar, Alzbeta Juven-Wetzler, Viki Myers, and Leah Fostic (2008). "Post-Traumatic stress disorder: Facts and Fiction". *Current opinion in psychiatry* **21** (1): 70–eoa. [doi:10.1097/YCO.0b013e3282f269ee](https://doi.org/10.1097/YCO.0b013e3282f269ee). [PMID 18281844](https://pubmed.ncbi.nlm.nih.gov/18281844/).
57. Yehuda R, Halligan SL, Golier JA, Grossman R, Bierer LM (2004). "Effects of trauma exposure on the cortisol response to dexamethasone administration in PTSD and major depressive disorder". *Psychoneuroendocrinology* **29** (3): 389–404. [doi:10.1016/S0306-4530\(03\)00052-0](https://doi.org/10.1016/S0306-4530(03)00052-0). [PMID 14644068](https://pubmed.ncbi.nlm.nih.gov/14644068/).


58. Yehuda R, Halligan SL, Grossman R, Golier JA, Wong C (2002). "The cortisol and glucocorticoid receptor response to low dose dexamethasone administration in aging combat veterans and holocaust survivors with and without posttraumatic stress disorder". *Biol Psychiatry* **52** (5): 393–403. [doi:10.1016/S0006-3223\(02\)01357-4](https://doi.org/10.1016/S0006-3223(02)01357-4). [PMID 12242055](https://pubmed.ncbi.nlm.nih.gov/12242055/).
59. Heim C, Ehler U, Hellhammer DH (2000). "The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders". *Psychoneuroendocrinology* **25** (1): 1–35. [doi:10.1016/S0306-4530\(99\)00035-9](https://doi.org/10.1016/S0306-4530(99)00035-9). [PMID 10633533](https://pubmed.ncbi.nlm.nih.gov/10633533/).
60. Mason JW, Giller EL, Kosten TR, Harkness L (1988). "Elevation of urinary norepinephrine/cortisol ratio in posttraumatic stress disorder". *J Nerv Ment Dis* **176** (8): 498–502. [doi:10.1097/00005053-198808000-00008](https://doi.org/10.1097/00005053-198808000-00008). [PMID 3404142](https://pubmed.ncbi.nlm.nih.gov/3404142/).
61. Bohnen N, Nicolson N, Sulon J, Jolles J (1991). "Coping style, trait anxiety and cortisol reactivity during mental stress". *J Psychosom Res* **35** (2–3): 141–7. [doi:10.1016/0022-3999\(91\)90068-Y](https://doi.org/10.1016/0022-3999(91)90068-Y). [PMID 2046048](https://pubmed.ncbi.nlm.nih.gov/2046048/).
62. Geraciotti TD, Baker DG, Ekhtor NN, West SA, Hill KK, Bruce AB, Schmidt D, Rounds-Kugler B, Yehuda R, Keck PE, Kasckow JW (2001). "CSF norepinephrine concentrations in posttraumatic stress disorder". *Am J Psychiatry* **158** (8): 1227–1230. [doi:10.1176/appi.ajp.158.8.1227](https://doi.org/10.1176/appi.ajp.158.8.1227). [PMID 11481155](https://pubmed.ncbi.nlm.nih.gov/11481155/).
63. Sautter FJ, Bissette G, Wiley J, Manguno-Mire G, Schoenbachler B, Myers L, Johnson JE, Cerbone A, Malaspina D (December 2003). "Corticotropin-releasing factor in posttraumatic stress disorder (PTSD) with secondary psychotic symptoms, nonpsychotic PTSD, and healthy control subjects". *Biol. Psychiatry* **54** (12): 1382–8. [doi:10.1016/S0006-3223\(03\)00571-7](https://doi.org/10.1016/S0006-3223(03)00571-7). [PMID 14675802](https://pubmed.ncbi.nlm.nih.gov/14675802/).
64. de Kloet CS, Vermetten E, Geuze E, Lentjes EG, Heijnen CJ, Stalla GK, Westenberg HG (2008). "Elevated plasma corticotrophin-releasing hormone levels in veterans with posttraumatic stress disorder". *Prog. Brain Res. Progress in Brain Research* **167**: 287–91. [doi:10.1016/S0079-6123\(07\)67025-3](https://doi.org/10.1016/S0079-6123(07)67025-3). [ISBN 978-0-444-53140-7](https://www.isbn-international.org/product/978-0-444-53140-7). [PMID 18037027](https://pubmed.ncbi.nlm.nih.gov/18037027/).
65. Yehuda R (2001). "Biology of posttraumatic stress disorder". *J Clin Psychiatry*. 62. Suppl 17: 41–6. [PMID 11495096](https://pubmed.ncbi.nlm.nih.gov/11495096/).
66. Yehuda R (2002). "Clinical relevance of biologic findings in PTSD". *Psychiatr Q* **73** (2): 123–33. [doi:10.1023/A:1015055711424](https://doi.org/10.1023/A:1015055711424). [PMID 12025720](https://pubmed.ncbi.nlm.nih.gov/12025720/).
67. Aardal-Eriksson E, Eriksson TE, Thorell LH (2001). "Salivary cortisol, posttraumatic stress symptoms, and general health in the acute phase and during 9-month follow-up". *Biol. Psychiatry* **50** (12): 986–93. [doi:10.1016/S0006-3223\(01\)01253-7](https://doi.org/10.1016/S0006-3223(01)01253-7). [PMID 11750895](https://pubmed.ncbi.nlm.nih.gov/11750895/).
68. Lindley SE, Carlson EB, Benoit M (2004). "Basal and dexamethasone suppressed salivary cortisol concentrations in a community sample of patients with posttraumatic stress disorder". *Biol. Psychiatry* **55** (9): 940–5. [doi:10.1016/j.biopsych.2003.12.021](https://doi.org/10.1016/j.biopsych.2003.12.021). [PMID 15110738](https://pubmed.ncbi.nlm.nih.gov/15110738/).
69. "[NIMH · Post Traumatic Stress Disorder Research Fact Sheet](https://www.nimh.nih.gov/health/publications/post-traumatic-stress-disorder-research-fact-sheet)". *National Institutes of Health*. Retrieved 2014-01-29.
70. Newton, Philip. "[From Mouse to Man; the Anatomy of Posttraumatic Stress Disorder](http://psychologytoday.com)". *Psychologytoday.com*. Retrieved 20 December 2009.
71. Carlson, Neil R. (2007). *Physiology of Behavior* (9 ed.). Pearson Education, Inc. ^[full citation needed]
72. Jatzko A, Rothenhöfer S, Schmitt A, Gaser C, Demirakca T, Weber-Fahr W, Wessa M, Magnotta V, Braus DF (2006). "[Hippocampal volume in chronic posttraumatic stress disorder \(PTSD\): MRI study using two different evaluation methods](https://pubmed.ncbi.nlm.nih.gov/16701903/)" (PDF). *Journal of Affective Disorders* **94** (1–3): 121–126. [doi:10.1016/j.jad.2006.03.010](https://doi.org/10.1016/j.jad.2006.03.010). [PMID 16701903](https://pubmed.ncbi.nlm.nih.gov/16701903/). Retrieved 2014-01-29.


73. Newport DJ, Nemeroff CB; Nemeroff, Charles B (2010). "Neurobiology of posttraumatic stress disorder". *Current Opinion in Neurobiology* **10** (2): 211–218. [doi:10.1016/S0959-4388\(00\)00080-5](https://doi.org/10.1016/S0959-4388(00)00080-5). PMID 10753802.
74. Milad MR, Pitman RK, Ellis CB, Gold AL, Shin LM, Lasko NB, Zeidan MA, Handwerker K, Orr SP, Rauch SL (2009). "[Neurobiological basis of failure to recall extinction memory in posttraumatic stress disorder](https://doi.org/10.1016/j.biopsych.2009.06.026)". *Biol Psychiatry* **66** (12): 1075–82. [doi:10.1016/j.biopsych.2009.06.026](https://doi.org/10.1016/j.biopsych.2009.06.026). PMC 2787650. PMID 19748076.
75. Stein MB, Paulus MP (2009). "[Imbalance of approach and avoidance: the yin and yang of anxiety disorders](https://doi.org/10.1016/j.biopsych.2009.09.023)". *Biol Psychiatry* **66** (12): 1072–4. [doi:10.1016/j.biopsych.2009.09.023](https://doi.org/10.1016/j.biopsych.2009.09.023). PMC 2825567. PMID 19944792.
76. Agis-Balboa RC, Arcos-Diaz D, Wittnam J, Govindarajan N, Blom K, Burkhardt S, Haladyniak U, Agbemenyah HY, Zovoilis A, Salinas-Riester G, Opitz L, Sananbenesi F, Fischer A (August 2011). "[A hippocampal insulin-growth factor 2 pathway regulates the extinction of fear memories](https://doi.org/10.1038/emboj.2011.293)". *EMBO J* **30** (19): 4071–83. [doi:10.1038/emboj.2011.293](https://doi.org/10.1038/emboj.2011.293). PMC 3209781. PMID 21873981.
77. Radley JJ, Kabbaj M, Jacobson L, Heydendaal W, Yehuda R, Herman JP (September 2011). "[Stress risk factors and stress related pathology: Neuroplasticity epigenetics and endophenotypes](https://doi.org/10.3109/10253890.2011.604751)". *Stress* **14** (5): 481–497. [doi:10.3109/10253890.2011.604751](https://doi.org/10.3109/10253890.2011.604751). PMC 3641164. PMID 21848436.
78. Pitman RK (1989). "Post-traumatic stress disorder, hormones, and memory". *Biological Psychiatry* **26** (3): 221–223. [doi:10.1016/0006-3223\(89\)90033-4](https://doi.org/10.1016/0006-3223(89)90033-4). PMID 2545287.
79. Zohar J, Juven-Wetzler A, Myers V, Fostick L (January 2008). "Post traumatic stress disorder: facts and fiction". *Current opinion in psychiatry* **21** (1): 74–7. [doi:10.1097/YCO.0b013e3282f269ee](https://doi.org/10.1097/YCO.0b013e3282f269ee). PMID 18281844.
80. "[NIMH · What are the symptoms of PTSD?](https://www.nimh.nih.gov/health/topics/ptsd/what-are-the-symptoms-of-ptsd.shtml)". *National Institute of Mental Health*. Retrieved 2014-01-30.
81. American Psychiatric Association (1987). *Diagnostic and Statistical Manual of Mental Disorders: DSM-III-R*. ISBN 0-89042-018-1.^[page needed]
82. American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders, quick reference*. Washington, D.C.: American Psychiatric Association. p. 211. ISBN 0-89042-063-7.
83. Breslau N, Kessler RC (2001). "The stressor criterion in DSM-IV posttraumatic stress disorder: an empirical investigation". *Biol. Psychiatry* **50** (9): 699–704. [doi:10.1016/S0006-3223\(01\)01167-2](https://doi.org/10.1016/S0006-3223(01)01167-2). PMID 11704077.
84. Blake DD, Weathers FW, Nagy LM, Kaloupek DG, Gusman FD, Charney DS, Keane TM (January 1995). "The development of a Clinician-Administered PTSD Scale". *J Trauma Stress* **8** (1): 75–90. [doi:10.1002/jts.2490080106](https://doi.org/10.1002/jts.2490080106). PMID 7712061.
85. Foa E: The Post Traumatic Diagnostic Scale Manual. Minneapolis, NCS, 1995.^[incomplete short citation]
86. Brewin CR, Rose S, Andrews B, Green J, Tata P, McEvedy C, Turner S, Foa EB (2002). "Brief screening instrument for post traumatic stress disorder". *British Journal of Psychiatry* **181**: 158–162. PMID 12151288.
87. Foa EB, Cashman L, Jaycox L, Perry K (1997). "The validation of a self-report measure of posttraumatic stress disorder: the Posttraumatic Diagnostic Scale". *Psychological Assessment* **9** (4): 445–451. [doi:10.1037/1040-3590.9.4.445](https://doi.org/10.1037/1040-3590.9.4.445).

88. Pietrzak RH, Southwick SM (June 2009). "The importance of four-factor emotional numbing and dysphoria models in PTSD". *Am J Psychiatry* **166** (6): 726–727. [doi:10.1176/appi.ajp.2009.09010032](https://doi.org/10.1176/appi.ajp.2009.09010032). PMID 19487403.
89. King DW, Leskin GA, King LA, Weathers FW (June 1998). "Confirmatory factor analysis of the clinician-administered PTSD Scale: Evidence for the dimensionality of posttraumatic stress disorder". *Psychol Assess* **10** (2): 90–96. [doi:10.1037/1040-3590.10.2.90](https://doi.org/10.1037/1040-3590.10.2.90).
90. Elklit A, Shevlin M (Sep 2007). "The structure of PTSD symptoms: A test of alternative models using confirmatory factor analysis". *Br J Clin Psychol* **46** (3): 299–313. [doi:10.1348/014466506X171540](https://doi.org/10.1348/014466506X171540). PMID 17535523.
91. Palmieri PA, Weathers FW, Difede J, King DW (May 2007). "Confirmatory factor analysis of the PTSD Checklist and the Clinician-Administered PTSD Scale in disaster workers exposed to the World Trade Center Ground Zero". *J Abnorm Psychol* **116** (2): 329–341. [doi:10.1037/0021-843X.116.2.329](https://doi.org/10.1037/0021-843X.116.2.329). PMID 17516765.
92. Elhai JD, Palmieri PA (2011). "The factor structure of posttraumatic stress disorder: A literature update, critique of methodology, and agenda for future research". *Journal of Anxiety Disorders* **25** (6): 849–854. [doi:10.1016/j.janxdis.2011.04.007](https://doi.org/10.1016/j.janxdis.2011.04.007). PMID 21793239.
93. Yufik T, Simms LJ (2011). "A meta-analytic investigation of the structure of posttraumatic stress disorder symptoms". *J Abnorm Psychol* **119** (4): 764–776. [doi:10.1037/a0020981](https://doi.org/10.1037/a0020981). PMID 21090877.
94. ["The ICD-10 Classification of Mental and Behavioural Disorders"](#) (PDF). World Health Organization. pp. 120–121. Retrieved 2014-01-29.
95. Feldner MT, Monson CM, Friedman MJ (2007). "A critical analysis of approaches to targeted PTSD prevention: current status and theoretically derived future directions". *Behav Modif* **31** (1): 80–116. [doi:10.1177/0145445506295057](https://doi.org/10.1177/0145445506295057). PMID 17179532.
96. Carlier, IVE; Lamberts, RD; van Uchelen, AJ; Gersons, BPR (1998). ["Disaster-related post-traumatic stress in police officers: A field study of the impact of debriefing"](#). *Stress Medicine* **14** (3): 143–8. [doi:10.1002/\(SICI\)1099-1700\(199807\)14:3<143::AID-SMI770>3.0.CO;2-S](https://doi.org/10.1002/(SICI)1099-1700(199807)14:3<143::AID-SMI770>3.0.CO;2-S).
97. Mayou RA, Ehlers A, Hobbs M (2000). "Psychological debriefing for road traffic accident victims. Three-year follow-up of a randomised controlled trial". *Br J Psychiatry* **176** (6): 589–93. [doi:10.1192/bjp.176.6.589](https://doi.org/10.1192/bjp.176.6.589). PMID 10974967.
98. Roberts NP, Kitchiner NJ, Kenardy J, Bisson JI (2009). ["Multiple session early psychological interventions for the prevention of post-traumatic stress disorder"](#). In Roberts, Neil P. "Cochrane Database of Systematic Reviews". Cochrane Database of Systematic Reviews. [doi:10.1002/14651858.CD006869.pub2](https://doi.org/10.1002/14651858.CD006869.pub2). Archived from [the original](#) on 2011-03-16. Retrieved April 27, 2011. (login required)
99. ["Assessment and Management of Conditions Specifically Related to Stress"](#) (PDF). Geneva: World Health Organization. 2013. ISBN 978-92-4-150593-2. Retrieved 2014-01-29.
100. Delahanty DL (2011). "Toward the predeployment detection of risk for PTSD". *American Psychiatric Assn* **168** (1): 9. [doi:10.1176/appi.ajp.2010.10101519](https://doi.org/10.1176/appi.ajp.2010.10101519). PMID 21205813.
101. van Zuiden M, Geuze E, Maas M, Vermetten E, Heijnen CJ, Kavelaars A (2009). "Deployment-related severe fatigue with depressive symptoms is associated with increased [glucocorticoid](#) binding to peripheral blood mono-nuclear cells". *Brain Behav Immun* **23** (8): 1132–1139. [doi:10.1016/j.bbi.2009.07.004](https://doi.org/10.1016/j.bbi.2009.07.004). PMID 19635550.
102. Resnick H, Acierno R, Holmes M, Kilpatrick DG, Jager N (1999). "Prevention of post-rape psychopathology: Preliminary findings of a controlled acute rape treatment study". *Journal of anxiety disorders* **13** (4): 359–370. [doi:10.1016/S0887-6185\(99\)00010-9](https://doi.org/10.1016/S0887-6185(99)00010-9). PMID 10504107.

103. van Emmerik AA, Kamphuis JH, Hulsbosch AM, Emmelkamp PM (2002). "Single session debriefing after psychological trauma: a meta-analysis". *The Lancet* **360** (9335): 766–771. doi:10.1016/S0140-6736(02)09897-5. PMID 12241834.
104. Rose S, Bisson J, Churchill R, Wessely S (January 21, 2009). "[Psychological debriefing for preventing post traumatic stress disorder \(PTSD\)](#)". In Rose, Suzanna C. "Cochrane summary on psychological debriefing". *Summaries.cochrane.org* (The Cochrane Collaboration) (2): CD000560. doi:10.1002/14651858.CD000560. PMID 12076399.
105. Roberts NP, Kitchiner NJ, Kenardy J, Bisson JI (April 14, 2010). "[Multiple session early psychological interventions for the prevention of post-traumatic stress disorder](#)". In Roberts, Neil P. "Cochrane summary on multiple session early psychological interventions for post-traumatic stress disorder". *Summaries.cochrane.org* (The Cochrane Collaboration). doi:10.1002/14651858.CD006869.pub2.
106. "[American Psychological Association on psychological debriefing](#)".
107. Khoshnu, E (October 2006). "[Clonidine for Treatment of PTSD](#)". *Clinical Psychiatry News* **34** (10): 22. doi:10.1016/S0270-6644(06)71796-9. Retrieved 9 February 2010. (login required)
108. Pitman RK, Sanders KM, Zusman RM, Healy AR, Cheema F, Lasko NB, Cahill L, Orr SP (2002). "Pilot study of secondary prevention of posttraumatic stress disorder with propranolol". *Biol. Psychiatry* **51** (2): 189–92. doi:10.1016/S0006-3223(01)01279-3. PMID 11822998.
109. Schelling G, Roozendaal B, Krauseneck T, Schmoelz M, DE Quervain D, Briegel J (2006). "Efficacy of hydrocortisone in preventing posttraumatic stress disorder following critical illness and major surgery". *Ann N Y Acad Sci* **1071** (1): 46–53. doi:10.1196/annals.1364.005. PMID 16891561.
110. Weis F, Kilger E, Roozendaal B, de Quervain DJ, Lamm P, Schmidt M, Schmölz M, Briegel J, Schelling G (2006). "Stress doses of hydrocortisone reduce chronic stress symptoms and improve health-related quality of life in high-risk patients after cardiac surgery: a randomized study". *J Thorac Cardiovasc Surg* **131** (2): 277–282. doi:10.1016/j.jtcvs.2005.07.063. PMID 16434254.
111. Schelling G, Kilger E, Roozendaal B, de Quervain DJ, Briegel J, Dagge A, Rothenhäusler HB, Krauseneck T, Nollert G, Kapfhammer HP (2004). "Stress doses of hydrocortisone, traumatic memories, and symptoms of posttraumatic stress disorder in patients after cardiac surgery: a randomized study". *Biol Psychiatry* **55** (6): 627–633. doi:10.1016/j.biopsych.2003.09.014. PMID 15013832.
112. Cahill, S. P.; Foa, E. B. (2004). Taylor, S., ed. *Advances in the Treatment of Posttraumatic Stress Disorder: Cognitive-behavioral perspectives*. New York: Springer. pp. 267–313.
113. Brom D, Kleber RJ, Defares PB (October 1989). "[Brief psychotherapy for posttraumatic stress disorders](#)". *J Consult Clin Psychol* **57** (5): 607–12. doi:10.1037/0022-006X.57.5.607. PMID 2571625.
114. Ehlers A, Bisson J, Clark DM, Creamer M, Pilling S, Richards D, Schnurr PP, Turner S, Yule W (March 2010). "[Do all psychological treatments really work the same in posttraumatic stress disorder?](#)". *Clin Psychol Rev* **30** (2): 269–76. doi:10.1016/j.cpr.2009.12.001. PMC 2852651. PMID 20051310.
115. Bisson JI, Ehlers A, Matthews R, Pilling S, Richards D, Turner S (2007). "[Psychological treatments for chronic post-traumatic stress disorder. Systematic review and meta-analysis](#)". *Br J Psychiatry* **190** (2): 97–104. doi:10.1192/bjp.bp.106.021402. PMID 17267924.

116. Seidler GH, Wagner FE (2006). "Comparing the efficacy of EMDR and trauma-focused cognitive-behavioral therapy in the treatment of PTSD: a meta-analytic study". *Psychol Med* **36** (11): 1515–22. [doi:10.1017/S0033291706007963](https://doi.org/10.1017/S0033291706007963). PMID 16740177.
117. Hassija, CM; Gray, MJ (2007). "[Behavioral Interventions for Trauma and Posttraumatic Stress Disorder](#)". *International Journal of Behavioral Consultation and Therapy* (Behavior Analyst Online) **3** (2): 166–175.
118. Mulick, P. S.; Landes, S.; Kanter, J. W. (2005). "[Contextual Behavior Therapies in the Treatment of PTSD: A Review](#)" (PDF). *International Journal of Behavioral Consultation and Therapy* **1** (3): 223–228.
119. Mulick, P. S.; Naugle, A. E. (2009). "[Behavioral Activation in the Treatment of Comorbid Posttraumatic Stress Disorder and Major Depressive Disorder](#)". *International Journal of Behavioral Consultation and Therapy* **5** (2): 330–339.
120. Grohol, JM. "[What is Exposure Therapy?](#)". *Psychcentral.com*. Retrieved 2010-07-14.
121. Joseph, J. S.; Gray, M. J. (2008). "[Exposure Therapy for Posttraumatic Stress Disorder](#)". *Journal of Behavior Analysis of Offender and Victim: Treatment and Prevention* **1** (4): 69–80. Retrieved 2010-05-10.
122. Ursano RJ, Bell C, Eth S, Friedman M, Norwood A, Pfefferbaum B, Pynoos JD, Zatzick DF, Benedek DM, McIntyre JS, Charles SC, Altshuler K, Cook I, Cross CD, Mellman L, Moench LA, Norquist G, Twemlow SW, Woods S, Yager J (November 2004). Work Group on ASD PTSD, Steering Committee on Practice Guidelines. "Practice guideline for the treatment of patients with acute stress disorder and posttraumatic stress disorder". *Am J Psychiatry* **161** (11 Suppl): 3–31. PMID 15617511.
123. *Committee on Treatment of Posttraumatic Stress Disorder, Institute of Medicine: Treatment of Posttraumatic Stress Disorder: An Assessment of the Evidence*. Washington, D.C.: National Academies Press. 2008. ISBN 0-309-10926-4.^[page needed]
124. "[Prolonged Exposure Therapy](#)". *U.S. Department of Veteran Affairs*. PTSD: National Center for PTSD. 2009-09-29. Retrieved 2010-07-14.
125. Karlin BE, Ruzek JI, Chard KM, Eftekhari A, Monson CM, Hembree EA, Resick PA, Foa EB (2010). "Dissemination of evidence-based psychological treatments for posttraumatic stress disorder in the Veterans Health Administration". *Journal of Traumatic Stress* **23** (6): 663–673. [doi:10.1002/jts.20588](https://doi.org/10.1002/jts.20588). PMID 21171126.
126. Shapiro, Francine (April 1989). "Efficacy of the eye movement desensitization procedure in the treatment of traumatic memories". *Journal of Traumatic Stress* **2** (2): 199–223. [doi:10.1002/jts.2490020207](https://doi.org/10.1002/jts.2490020207).
127. Shapiro F, Maxfield L (August 2002). "Eye Movement Desensitization and Reprocessing (EMDR): information processing in the treatment of trauma". *Journal of clinical psychology* **58** (8): 933–46. [doi:10.1002/jclp.10068](https://doi.org/10.1002/jclp.10068). PMID 12115716.
128. The Management of Post-Traumatic Stress Working Group (2010). "[VA/DoD clinical practice guideline for management of post-traumatic stress](#)". Department of Veterans Affairs, Department of Defense. p. Version 2.0. Retrieved 2 June 2013.
129. Gillies D, Taylor F, Gray C, O'Brien L, D'Abrew N (2012). Gillies, Donna, ed. "Psychological therapies for the treatment of post-traumatic stress disorder in children and adolescents". *Cochrane Database of Systematic Reviews* **12**: CD006726. [doi:10.1002/14651858.CD006726.pub2](https://doi.org/10.1002/14651858.CD006726.pub2). PMID 23235632. [Lay summary](#) – *Pubmed Health (plain English)*.

130. Jeffries FW, Davis P; Davis, P. (May 2013). "What is the role of eye movements in eye movement desensitization and reprocessing (EMDR) for post-traumatic stress disorder (PTSD)? a review". *Behavioural and cognitive psychotherapy* **41** (3): 290–300. [doi:10.1017/S1352465812000793](https://doi.org/10.1017/S1352465812000793). PMID 23102050.
131. Jonas, D. E.; Cusack, K.; Forneris, C. A. (April 2013). "[Psychological and pharmacological treatments for adults with posttraumatic stress disorder \(PTSD\)](#)". Comparative Effectiveness Reviews (92). Rockville, MD: US Agency for Healthcare Quality and Research. PMID 23658937. [Lay summary](#) – *Pubmed Health (plain English)*. 
132. Weissman, M. M.; Markowitz, J. C.; Klerman, G. L. (2007). *Clinician's Quick Guide to Interpersonal Psychotherapy*. New York: Oxford University Press. ^[page needed]
133. Bleiberg KL, Markowitz JC (January 2005). "A pilot study of interpersonal psychotherapy for posttraumatic stress disorder". *Am J Psychiatry* **162** (1): 181–3. [doi:10.1176/appi.ajp.162.1.181](https://doi.org/10.1176/appi.ajp.162.1.181). PMID 15625219.
134. "[Trauma and PTSD Program – Columbia University Department of Psychiatry](#)". Columbiatrauma.org. Retrieved 2014-01-29. ^[broken citation]
135. Markowitz JC, Milrod B, Bleiberg K, Marshall RD (March 2009). "[Interpersonal factors in understanding and treating posttraumatic stress disorder](#)". *J Psychiatr Pract* **15** (2): 133–40. [doi:10.1097/01.pra.0000348366.34419.28](https://doi.org/10.1097/01.pra.0000348366.34419.28). PMC 2852131. PMID 19339847.
136. Markowitz JC (October 2010). "[IPT and PTSD](#)". *Depress Anxiety* **27** (10): 879–81. [doi:10.1002/da.20752](https://doi.org/10.1002/da.20752). PMC 3683871. PMID 20886608.
137. Maxmen, J. S.; Ward, N. G. (1995). *Essential psychopathology and its treatment, 2nd ed., revised for DSM-IV* (2nd ed.). New York: W. W. Norton. p. 280. ISBN 0-393-70173-5.
138. Maxmen, J. S.; Ward, N. G. (2002). *Psychotropic drugs: fast facts* (3rd ed.). New York: W. W. Norton. p. 346. ISBN 0-393-70301-0.
139. Krystal JH, Neumeister A; Alexander, Neumeister (2009). "[Noradrenergic and serotonergic mechanisms in the neurobiology of posttraumatic stress disorder and resilience](#)". *Brain Research* **1293**: 13–23. [doi:10.1016/j.brainres.2009.03.044](https://doi.org/10.1016/j.brainres.2009.03.044). PMC 2761677. PMID 19332037.
140. Bisson, Jonathan I (2008). "Pharmacological treatment to prevent and treat post-traumatic stress disorder". *Torture* **18** (2): 104–104.
141. Berger W, Mendlowicz MV, Marques-Portella C, Kinrys G, Fontenelle LF, Marmar CR, Figueira I (Mar 2009). "[Pharmacologic alternatives to antidepressants in posttraumatic stress disorder: a systematic review](#)". *Prog Neuropsychopharmacol Biol Psychiatry* **33** (2): 169–80. [doi:10.1016/j.pnpbp.2008.12.004](https://doi.org/10.1016/j.pnpbp.2008.12.004). PMC 2720612. PMID 19141307.
142. Cooper J, Carty J, Creamer M (Aug 2005). "Pharmacotherapy for posttraumatic stress disorder: empirical review and clinical recommendations". *Aust N Z J Psychiatry* **39** (8): 674–82. [doi:10.1111/j.1440-1614.2005.01651.x](https://doi.org/10.1111/j.1440-1614.2005.01651.x). PMID 16050921.
143. Yehuda R (2000). "Biology of posttraumatic stress disorder". *J Clin Psychiatry* **61** (Suppl 7): 14–21. PMID 10795605.
144. Maxmen, J. S.; Ward, N. G. (2002). *Psychotropic drugs: fast facts* (3rd ed.). New York: W. W. Norton. p. 347. ISBN 0-393-70301-0.
145. Marshall RD, Beebe KL, Oldham M, Zaninelli R (December 2001). "[Efficacy and safety of paroxetine treatment for chronic PTSD: a fixed-dose, placebo-controlled study](#)". *Am J Psychiatry* **158** (12): 1982–8. [doi:10.1176/appi.ajp.158.12.1982](https://doi.org/10.1176/appi.ajp.158.12.1982). PMID 11729013.

146. Brady K, Pearlstein T, Asnis GM, Baker D, Rothbaum B, Sikes CR, Farfel GM (April 2000). ["Efficacy and safety of sertraline treatment of posttraumatic stress disorder: a randomized controlled trial"](#). *JAMA* **283** (14): 1837–44. doi:[10.1001/jama.283.14.1837](#). PMID [10770145](#).
147. Davidson JR, Rothbaum BO, van der Kolk BA, Sikes CR, Farfel GM (May 2001). ["Multicenter, double-blind comparison of sertraline and placebo in the treatment of posttraumatic stress disorder"](#). *Arch Gen Psychiatry* **58** (5): 485–92. doi:[10.1001/archpsyc.58.5.485](#). PMID [11343529](#).
148. Maxmen, J. S.; Ward, N. G. (1995). *Essential psychopathology and its treatment, 2nd ed., revised for DSM-IV* (2nd ed.). New York: W. W. Norton. p. 104. ISBN [0-393-70173-5](#).
149. Maxmen, J. S.; Ward, N. G. (1995). *Essential psychopathology and its treatment, 2nd ed., revised for DSM-IV* (2nd ed.). New York: W. W. Norton. p. 175. ISBN [0-393-70173-5](#).
150. Maxmen, J. S.; Ward, N. G. (2002). *Psychotropic drugs: fast facts* (3rd ed.). New York: W. W. Norton. p. 349. ISBN [0-393-70301-0](#).
151. Lacy CF, Armstrong LL et al. (2008). *Drug Information Handbook*. Lexi-Comp. pp. 260, 934. ISBN [978-1-59195-319-7](#).
152. ["Lamotrigine FAQ"](#). *Psychom.net*. Retrieved 2007-05-01.
153. Asnis GM, Kohn SR, Henderson M, Brown NL (2004). ["SSRIs versus Non-SSRIs in Post-traumatic Stress Disorder : An Update with Recommendations"](#). *Drugs* (Department of Psychiatry and Behavioral Sciences, Montefiore Medical Center, Albert Einstein College of Medicine, Bronx, New York) **64** (4): 383–404. doi:[10.2165/00003495-200464040-00004](#). PMID [14969574](#).
154. Hertzberg MA, Butterfield MI, Feldman ME, Beckham JC, Sutherland SM, Connor KM, Davidson JR (May 1999). ["A preliminary study of lamotrigine for the treatment of posttraumatic stress disorder"](#). *Biol. Psychiatry* **45** (9): 1226–9. doi:[10.1016/S0006-3223\(99\)00011-6](#). PMID [10331117](#).
155. Kapfhammer HP (December 2008). "[Therapeutic possibilities after traumatic experiences]". *Psychiatr Danub* **20** (4): 532–45. PMID [19011595](#).
156. Ahmadzadeh, G.; Asadolahi, G.; Mahmodi, G.; Farhat, A. (28 February 2006). ["AGP | Effect of cyproheptadine on combat related PTSD nightmares"](#). *Annals of General Psychiatry* **5** (Suppl 1): S159. doi:[10.1186/1744-859X-5-S1-S159](#). Retrieved 2014-01-29. 
157. Maxmen, J. S.; Ward, N. G. (1995). *Essential psychopathology and its treatment, 2nd ed., revised for DSM-IV* (2nd ed.). New York: W. W. Norton. p. 95. ISBN [0-393-70173-5](#).
158. Jain S, Greenbaum MA, Rosen C (February 2012). "Concordance between psychotropic prescribing for veterans with PTSD and clinical practice guidelines". *Psychiatr Serv* **63** (2): 154–60. doi:[10.1176/appi.ps.201100199](#). PMID [22302333](#).
159. Reist, C (2005). Post-traumatic Stress Disorder. Compendia, Build ID: F000005, published by Epocrates.com
160. Auxéméry Y (October 2012). "[Posttraumatic stress disorder (PTSD) as a consequence of the interaction between an individual genetic susceptibility, a traumatogenic event and a social context]". *Encephale* (in French) **38** (5): 373–80. doi:[10.1016/j.encep.2011.12.003](#). PMID [23062450](#).
161. Martényi F (Mar 2005). "[Three paradigms in the treatment of posttraumatic stress disorder]". *Neuropsychopharmacol Hung* **7** (1): 11–21. PMID [16167463](#).
162. Cohen H, Matar MA, Buskila D, Kaplan Z, Zohar J (2008). "Early post-stressor intervention with high-dose corticosterone attenuates posttraumatic stress response in an animal

- model of posttraumatic stress disorder". *Biol Psychiatry* **64** (8): 708–717. [doi:10.1016/j.biopsych.2008.05.025](https://doi.org/10.1016/j.biopsych.2008.05.025). PMID 18635156.
163. Sapolsky RM, Romero LM, Munck AU (2000). "How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions". *Endocr Review* **21** (1): 55–89. [doi:10.1210/er.21.1.55](https://doi.org/10.1210/er.21.1.55). PMID 10696570.
164. Maxmen, J. S.; Ward, N. G. (2002). *Psychotropic drugs: fast facts* (3rd ed.). New York: W. W. Norton. pp. 347–349. ISBN 0-393-70301-0.
165. Lawrence, Sue; De Silva, M; Henley, R (2010). Lawrence, Sue, ed. "[Sports and games for post-traumatic stress disorder \(PTSD\)](#)". *Cochrane Database of Systematic Reviews* (1). [doi:10.1002/14651858.CD007171.pub2](https://doi.org/10.1002/14651858.CD007171.pub2).
166. Jankowski, K. "[PTSD and physical health](#)". *Information on trauma and PTSD for professionals, National Center for PTSD*. U.S. Department of Veterans Affairs. Retrieved 8 June 2013.
167. U.S. Department of Veterans Affairs. "[Lifestyle Changes Recommended for PTSD Patients](#)". *Information on trauma and PTSD for veterans, general public and family from the National Center for PTSD*. U.S. Department of Veterans Affairs. Retrieved 8 June 2013.
168. Liedl A, Müller J, Morina N, Karl A, Denke C, Knaevelsrud C (1 February 2011). "Physical Activity within a CBT Intervention Improves Coping with Pain in Traumatized Refugees: Results of a Randomized Controlled Design". *Pain Medicine* **12** (2): 234–245. [doi:10.1111/j.1526-4637.2010.01040.x](https://doi.org/10.1111/j.1526-4637.2010.01040.x). PMID 21223501.
169. Rosenbaum S, Nguyen D, Lenehan T, Tiedemann A, van der Ploeg HP, Sherrington C (Jul 22, 2011). "[Exercise augmentation compared to usual care for post traumatic stress disorder: a randomised controlled trial \(the REAP study: Randomised Exercise Augmentation for PTSD\)](#)". *BMC Psychiatry* **11**: 115. [doi:10.1186/1471-244X-11-115](https://doi.org/10.1186/1471-244X-11-115). PMC 3151207. PMID 21777477.
170. Neylan, TC. "[Integrative Exercise for Post-Deployment Stress](#)". *ClinicalTrials.gov Identifier: NCT01674244*. US National Institutes of Health (NIH). Retrieved 8 June 2013.
171. Smits, J. "[Maximizing Treatment Outcome and Examining Sleep in Post-traumatic Stress Disorder \(PTSD\)](#)". US National Institutes of Health (NIH). NCT01199107. Retrieved 8 June 2013.
172. Wethington HR, Hahn RA, Fuqua-Whitley DS, Sipe TA, Crosby AE, Johnson RL, Liberman AM, Mościcki E, Price LN, Tuma FK, Kalra G, Chattopadhyay SK (31 August 2008). "[The Effectiveness of Interventions to Reduce Psychological Harm from Traumatic Events Among Children and Adolescents](#)". *American Journal of Preventive Medicine* **35** (3): 287–313. [doi:10.1016/j.amepre.2008.06.024](https://doi.org/10.1016/j.amepre.2008.06.024). PMID 18692745.
173. Fletcher, K. E.; Barkley, Russell A. (2003). "7". In Mash, Eric J. *Child psychopathology* (2nd ed.). New York: Guilford Press. pp. 330–371. ISBN 1-57230-609-2.
174. Hamblen, J; Barnett, E. (20 December 2012). "[PTSD in children and adolescents](#)". VA National Center for Posttraumatic Stress Disorder. Retrieved 10 June 2013.
175. "[Mortality and Burden of Disease Estimates for WHO Member States in 2004](#)". *World Health Organization*.
176. Brunet A, Akerib V, Birmes P (2007). "[Don't throw out the baby with the bathwater \(PTSD is not overdiagnosed\)](#)" (PDF). *Can J Psychiatry* **52** (8): 501–2; discussion 503. PMID 17955912. Retrieved 2008-03-12.
177. "[Mortality and Burden of Disease Estimates for WHO Member States: Persons, all ages \(2004\)](#)" (xls). *World Health Organization*. 2004. Retrieved 2009-11-12.

178. ["Mortality and Burden of Disease Estimates for WHO Member States: Females, all ages \(2004\)"](#) (xls). *World Health Organization*. 2004. Retrieved 2009-11-12.
179. ["Mortality and Burden of Disease Estimates for WHO Member States: Males, all ages \(2004\)"](#) (xls). *World Health Organization*. 2004. Retrieved 2009-11-12.
180. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB (December 1995). "Posttraumatic stress disorder in the National Comorbidity Survey". *Arch Gen Psychiatry* **52** (12): 1048–60. doi:10.1001/archpsyc.1995.03950240066012. PMID 7492257.
181. Sher, Leo (2010). "Neurobiology of suicidal behavior in post-traumatic stress disorder". *Expert Reviews* **10** (8): 1233–1235. doi:10.1586/ern.10.114. PMID 20662745.
182. Mintz, S. (2007). ["The War's Costs"](#). *Digital History*. Archived from [the original](#) on 2003-09-07.
183. ["Psychological Costs of War: Military Combat and Mental Health"](#). Journalistsresource.org. Retrieved 2014-01-29.
184. Department of Veterans Affairs Office of Inspector General (May 19, 2005). ["Review of State Variances in VA Disability Compensation Payments"](#) (PDF). Washington, DC: Department of Veterans Affairs Office of Inspector General. Retrieved 2011-09-03.
185. Hedenko, William. ["The Relationship Between PTSD and Suicide"](#). Retrieved 3 November 2011.
186. Secretary Nicholson (2005-11-10). "No Across-the-Board Review of PTSD Cases". The Department of Veterans Affairs.; ["No Across-the-Board Review of PTSD Cases – Secretary Nicholson"](#) (Press release). Washington, D.C.: United States Department of Veteran Affairs. November 10, 2005.
187. ["Marine Corps Offers Yoga, Massages to Marriages Strained by War"](#). Fox News Channel. Associated Press. 2008-04-02. Retrieved 2008-04-03.
188. ["Welcome to the American Legion Online"](#). Archived from [the original](#) on 2008-01-31. ^[broken citation]
189. ["Sierra Club Military Families and Veterans Initiative"](#). *Sierraclub.typepad.com*. Retrieved 2012-07-26.
190. Reed McManus (August 2011). ["A Missing Peace – Can wilderness adventure repair the ravages of war?"](#). *Sierra Magazine – Sierra Club*. Retrieved 2012-07-26.
191. ["Military Families and Veterans Initiative: Grizzly Tracking in Montana for Veterans, Service Members, and Families"](#). *Sierraclub.typepad.com*. Retrieved 2012-07-26.
192. Dixon, Laura (February 28, 2009). ["Lance Corporal Johnson Beharry accuses Government of neglecting soldiers"](#). *The Times* (London). Retrieved 2009-08-29. (login required)
193. Hickley, Matthew; Hope, Jenny (2009-03-02). ["British troops in Afghanistan face mental health timebomb 'on the scale of Vietnam'"](#). *Daily Mail* (London). Retrieved 2009-08-29.
194. ["UK | Full interview: L/Cpl Johnson Beharry"](#). BBC News. 2009-02-28. [Archived](#) from the original on 2014-02-19. Retrieved 2009-08-29.
195. ["The Operational Stress Injury Social Support \(OSISS\) Program for Canadian Veterans"](#). See also ["Evaluation of the OSISS Peer Support Network"](#) (PDF). Dept. of National Defence and Veterans Affairs Canada. January 2005.
196. Heber, A.; Grenier, S.; Richardson, D.; Darte, K. (2006). ["Combining Clinical Treatment and Peer Support: A Unique Approach to Overcoming Stigma and Delivering Care"](#) (PDF). *Human Dimensions in Military Operations – Military Leaders' Strategies for Addressing Stress and Psychological Support*. Neuilly-sur-Seine, France: Canadian Department Of National Defence. Retrieved 2014-01-30.

197. J Don Richardson, Kathy Darte, Stéphane Grenier, Allan English, Joe Sharpe (2008). ["Operational Stress Injury Social Support: a Canadian innovation in professional peer support"](#). *Canadian Military Journal* **9** (1): 57–64. Retrieved 2014-01-30.
198. ["The New Veterans Charter for CF Veterans and their Families"](#). Vac-Acc.Gc.Ca. 2006-07-12. Archived from [the original](#) on 2006-06-19. Retrieved 2009-08-29.
199. ["Henry IV, Part I, Act II, Scene 3 : |: Open Source Shakespeare"](#). Opensourceshakespeare.org. Retrieved 2014-01-30.
200. Shay, Jonathan (1994). *Achilles in Vietnam: Combat Trauma and the Undoing of Character*. Scribner. pp. 165–66.
201. [World War One – A New Kind of War | Part II](#), From 14 – 18 *Understanding the Great War*, by Stéphane Audoin-Rouzeau, Annette Becker^{[[incomplete short citation](#)]}
202. ["When trauma tips you over: PTSD Part 1"](#). All in the Mind. Australian Broadcasting Commission. 9 October 2004.
203. [Andreasen, Nancy C.](#) (Feb 19, 2004). *Brave New Brain: Conquering Mental Illness in the Era of the Genome*. New York: Oxford University Press. p. 303. [ISBN 978-0-19-516728-3](#).
204. Jones, Joshua A. 2013, VOL. 5 NO. 02 pp. 1-3. ["Nostalgia to Post-Traumatic Stress Disorder: A Mass Society Theory of Psychological Reactions to Combat"](#) *The International Student Journal*
205. Shalev, Arieh Y.; Yehuda, Rachel; Alexander C. McFarlane (2000). *International handbook of human response to trauma*. New York: Kluwer Academic/Plenum Press. [ISBN 0-306-46095-5](#).^{[[page needed](#)]},[on-line](#).
206. ["International Statistical Classification of Diseases and Related Health Problems 10th Revision Version for 2007"](#). World Health Organization (UN). 2007. Retrieved October 3, 2011.
207. Ojo, J; Greenberg, M (December 2014). "213". "Neurobehavioral, neuropathological and biochemical profiles in a novel mouse model of co-morbid post-traumatic stress disorder and mild traumatic brain injury.". *Front Behav Neurosci.* **8**. [doi:10.3389/fnbeh.2014.00213](#). [PMID 25002839](#).
208. Poulos, AM; Reger, M (August 2014). "Amnesia for early life stress does not preclude the adult development of posttraumatic stress disorder symptoms in rats.". *Biol Psychiatry.* **15** (76): 306–14. [doi:10.1016/j.biopsych.2013.10.007](#). [PMID 24231200](#).
209. Emerson A, Ponté L, Jerome L, Doblin R (2014). "History and future of the Multidisciplinary Association for Psychedelic Studies (MAPS)". *J Psychoactive Drugs* **46** (1): 27–36. [doi:10.1080/02791072.2014.877321](#). [PMID 24830183](#).
210. ["A Manual for MDMA-Assisted Psychotherapy in the Treatment of Posttraumatic Stress Disorder"](#). [Multidisciplinary Association for Psychedelic Studies](#). 4 January 2013. Retrieved 31 May 2014.
211. White, C. Michael (16 April 2014). ["3,4-Methylenedioxymethamphetamine's \(MDMA's\) Impact on Posttraumatic Stress Disorder"](#). *Annals of Pharmacotherapy*.
212. de Kleine RA, Rothbaum BO, van Minnen A (17 Oct 2014). ["Pharmacological enhancement of exposure-based treatment in PTSD: a qualitative review"](#). *Eur J Psychotraumatology* **4**. [doi:10.3402/ejpt.v4i0.21626](#). [PMC 3800126](#). [PMID 24147208](#).

References for Genocide

1. [Jump up](#)^ *genocide* in the [Oxford English Dictionary](#), 2nd ed.—"1944R. Lemkin *Axis Rule in Occupied Europe* ix. 79 By 'genocide' we mean the destruction of a nation or of an ethnic group."
2. [Jump up](#)^ Office of the High Commissioner for Human Rights, [Convention on the Prevention and Punishment of the Crime of Genocide Archived](#) 2 May 2008 at the [Wayback Machine](#)
3. [Jump up](#)^ [Video interview with Raphael Lemkin CBS news](#) on [YouTube](#)
4. [Jump up](#)^ New York Times "[1]"
5. [Jump up](#)^ William Korey, "Raphael Lemkin: 'The Unofficial Man'," *Midstream*, June–July 1989, p. 45–48
6. [Jump up](#)^ Rubinstein, W. D. (2004). [Genocide: a history](#). Pearson Education. p. 308. [ISBN 0-582-50601-8](#).
7. [Jump up to:](#)^{a b} Robert Gellately & Ben Kiernan (2003). [The Specter of Genocide: Mass Murder in Historical Perspective](#). Cambridge, UK: Cambridge University Press. p. 267. [ISBN 0-521-52750-3](#).
8. [Jump up to:](#)^{a b c} Staub, Ervin (31 July 1992). [The Roots of Evil: The Origins of Genocide and Other Group Violence](#). Cambridge, UK: Cambridge University Press. p. 8. [ISBN 0-521-42214-0](#).
9. [Jump up](#)^ From a statement made by Mr. Morozov, representative of the Union of Soviet Socialist Republics, on 19 April 1948 during the debate in the Ad Hoc Committee on Genocide (E/AC.25/SR.12).
10. [Jump up](#)^ See Vienna Convention on the Law of Treaties, opened for signature on 23 May 1969, United Nations Treaty Series, vol. 1155, No. I-18232.
11. [Jump up](#)^ [Mandate, structure and methods of work: Genocide I](#) of the [UN Commission of Experts](#) to examine violations of international humanitarian law committed in the territory of the former Yugoslavia, created by [Security Council resolution 780](#) (1992) of 6 October 1992.
12. [Jump up](#)^ European Court of Human Rights [Judgement in Jorgic v. Germany \(Application no. 74613/01\)](#) paragraphs 18, 36,74
13. [Jump up](#)^ European Court of Human Rights [Judgement in Jorgic v. Germany \(Application no. 74613/01\)](#) paragraphs 43–46
14. [Jump up](#)^ [What is Genocide?](#) McGill Faculty of Law ([McGill University](#))
15. [Jump up](#)^ [Prosecutor v. Radislav Krstic – Trial Chamber I – Judgment – IT-98-33 \(2001\) ICTY8 \(2 August 2001\)](#)

16. [^ Jump up to:^a ^b Prosecutor v. Radislav Krstic – Appeals Chamber – Judgment – IT-98-33 \(2004\) ICTY 7 \(19 April 2004\)](#)
17. [Jump up^ Prosecutor v. Radislav Krstic – Appeals Chamber – Judgment – IT-98-33 \(2004\) ICTY 7 \(19 April 2004\)](#) See Paragraph 6: "Article 4 of the Tribunal's Statute, like the Genocide Convention, covers certain acts done with "intent to destroy, in whole or in part, a national, ethnical, racial or religious group, as such."
18. [Jump up^ Statute of the International Tribunal for the Prosecution of Persons Responsible for Serious Violations of International Humanitarian Law Committed in the Territory of the Former Yugoslavia since 1991](#), U.N. Doc. S/25704 at 36, annex (1993) and S/25704/Add.1 (1993), adopted by Security Council on 25 May 1993, [Resolution 827](#) (1993).
19. [Jump up^ Resolution Resolution 1674 \(2006\)](#)
20. [Jump up^ Security Council passes landmark resolution – world has responsibility to protect people from genocide Oxfam](#) Press Release – 28 April 2006
21. [Jump up^ http://www.un.org/News/Press/docs/2008/sc9364.doc.htm](#)
22. [Jump up^ The Crime of Genocide in Domestic Laws and Penal Codes](#) website of [prevent genocide international](#).
23. [Jump up^](#) William Schabas *War crimes and human rights: essays on the death penalty, justice and accountability*, Cameron May 2008 [ISBN 1-905017-63-4](#), [ISBN 978-1-905017-63-8](#). p. 791
24. [^ Jump up to:^a ^b](#) Kurt Jonassohn & Karin Solveig Björnson, *Genocide and Gross Human Rights Violations in Comparative Perspective: In Comparative Perspective*, Transaction Publishers, 1998,[ISBN 0-7658-0417-4](#), [ISBN 978-0-7658-0417-4](#). pp. 133–135
25. [Jump up^](#) M. Hassan Kakar *Afghanistan: The Soviet Invasion and the Afghan Response, 1979–1982* [University of California press 1995 The Regents of the University of California](#).
26. [Jump up^](#) M. Hassan Kakar [4. The Story of Genocide in Afghanistan: 13. Genocide Throughout the Country](#)
27. [Jump up^](#) Frank Chalk, Kurt Jonassohn *The History and Sociology of Genocide: Analyses and Case Studies*, Yale University Press, 1990, [ISBN 0-300-04446-1](#)
28. [Jump up^](#) Jones, Adam. *Genocide: A Comprehensive Introduction*, Routledge/Taylor & Francis Publishers, 2006. [ISBN 0-415-35385-8](#). [Chapter 1: The Origins of Genocide](#) pp.20–21
29. [Jump up^](#) [What is Genocide?](#) McGill Faculty of Law ([McGill University](#)) source cites Barbara Harff and Ted Gurr *Toward empirical theory of genocides and politicides*, International Studies Quarterly, 37:3, 1988
30. [Jump up^](#) [Origins and Evolution of the Concept](#) in the Science Encyclopedia by Net Industries. states "Politicide, as [Barbara] Harff and [Ted R.] Gurr define it, refers to the killing of groups of people who are targeted not because of shared ethnic or communal traits, but because of 'their

- hierarchical position or political opposition to the regime and dominant groups' (p. 360)". But does not give the book title to go with the page number.
31. [Jump up](#) Staff. [*There are NO Statutes of Limitations on the Crimes of Genocide!*](#) On the website of the American Patriot Friends Network. Cites Barbara Harff and Ted Gurr "Toward empirical theory of genocides and politicides," *International Studies Quarterly* 37, 3 [1988].
 32. [Jump up](#) Polsby, Daniel D.; Kates, Don B., Jr. (3 November 1997). "[OF HOLOCAUSTS AND GUN CONTROL](#)". *Washington University Law Quarterly* 75 (Fall): 1237. (cites Harff 1992, see other note)
 33. [Jump up](#) Harff, Barbara (1992). Fein, Helen, ed. "Recognizing Genocides and Politicides". *Genocide Watch* (New Haven, CT: Yale University Press) 27: 37, 38.
 34. [Jump up](#) [Domocide versus genocide; which is what?](#)
 35. [Jump up](#) Adrian Gallagher, *Genocide and Its Threat to Contemporary International Order* (Palgrave Macmillan, 2013) p. 37.
 36. [Jump up](#) [United Nations Treaty Collection \(As of 9 October 2001\): Convention on the Prevention and Punishment of the Crime of Genocide](#) on the web site of the [Office of the United Nations High Commissioner for Human Rights](#)
 37. [Jump up](#) (See for example the submission by Agent of the United States, Mr. David Andrews to the ICJ Public Sitting, 11 May 1999)
 38. [Jump up](#) [Oxford English Dictionary](#): 1944 R. Lemkin *Axis Rule in Occupied Europe* ix. 79 "By 'genocide' we mean the destruction of a nation or of an ethnic group."
 39. [Jump up](#) [Oxford English Dictionary](#) "Genocide" citing Sunday Times 21 October 1945
 40. [Jump up](#) Staff. [Bosnian genocide suspect extradited](#), [BBC](#), 2 April 2002
 41. [Jump up](#) [European Court of Human Rights](#). [Jorgic v. Germany Judgment](#), 12 July 2007. § 47
 42. [Jump up](#) The [International Criminal Tribunal for the Former Yugoslavia](#) found in [Prosecutor v. Radislav Krstic – Trial Chamber I – Judgment – IT-98-33 \(2001\) ICTY8 \(2 August 2001\)](#) that genocide had been committed. (see paragraph 560 for name of group in English on whom the genocide was committed^{[who?][clarification needed]}). It was upheld in [Prosecutor v. Radislav Krstic – Appeals Chamber – Judgment – IT-98-33 \(2004\) ICTY 7 \(19 April 2004\)](#)
 43. [Jump up](#) ["Courte: Serbia failed to prevent genocide, UN court rules"](#). *The San Francisco Chronicle*. Associated Press. 26 February 2007.
 44. [Jump up](#) ECHR *Jorgic v. Germany*. § 42 citing *Prosecutor v. Krstic*, IT-98-33-T, judgment of 2 August 2001, §§ 580
 45. [Jump up](#) ECHR *Jorgic v. Germany Judgment*, 12 July 2007. § 44 citing *Prosecutor v. Kupreskic and Others* (IT-95-16-T, judgment of 14 January 2000), § 751. In 14 January 2000, the

- ICTY ruled in the [Prosecutor v. Kupreskic and Others](#) case that the killing of 116 Muslims in order to expel the Muslim population from a village amounted to persecution, not genocide.
46. [Jump up](#)[^] [ICJ press release 2007/8](#) 26 February 2007
 47. [Jump up](#)[^] http://icty.org/x/cases/krajisnik/cis/en/cis_krajisnik_en.pdf
 48. [Jump up](#)[^] Staff (5 November 2009). "[Q&A: Karadzic on trial](#)". BBC News. Retrieved 28 January 2010.
 49. [Jump up](#)[^] Staff (26 May 2011). "[Q&A: Ratko Mladic arrested: Bosnia war crimes suspect held](#)". BBC News. Retrieved 28 May 2011.
 50. [Jump up](#)[^] These figures need revising they are from the [ICTR](#) page which says see www.ictor.org
 51. [Jump up](#)[^] [Cambodian Genocide Program](#), [Yale University's](#) MacMillan Center for International and Area Studies
 52. [Jump up](#)[^] "[A/RES/57/228B](#)". 2003-05-022. Retrieved 11 December 2010. Check date values in: |date= ([help](#))
 53. [^] [Jump up to:](#)^{a b} Doyle, Kevin. "[Putting the Khmer Rouge on Trial](#)",*Time*, 26 July 2007
 54. [Jump up](#)[^] MacKinnon, Ian "[Crisis talks to save Khmer Rouge trial](#)",*The Guardian*, 7 March 2007
 55. [Jump up](#)[^] [The Khmer Rouge Trial Task Force](#), Royal Cambodian Government
 56. [Jump up](#)[^] [Case 002](#) The Extraordinary Chambers in the Courts of Cambodia. Retrieved 14 August 2014
 57. [Jump up](#)[^] [Former Khmer Rouge leaders begin genocide trial](#) BBC. 30 July 2014
 58. [^] [Jump up to:](#)^{a b} Buncombe, Andrew (11 October 2011). "[Judge quits Cambodia genocide tribunal](#)". *The Independent* (London).
 59. [Jump up](#)[^] Ker Munthit (12 August 2008). "[Cambodian tribunal indicts Khmer Rouge jailer](#)". *USA Today*. Associated Press. Retrieved April 2012.
 60. [Jump up](#)[^] "[Kaing Guek Eav alias Duch Sentenced to Life Imprisonment by the Supreme Court Chamber](#)". Extraordinary Chambers in the Courts of Cambodia. 3 February 2012. Retrieved April 2012.
 61. [^] [Jump up to:](#)^{a b c} "[Case 002](#)". Extraordinary Chambers in the Courts of Cambodia. Retrieved April 2012.
 62. [^] [Jump up to:](#)^{a b c d} "[002/19-09-2007: Closing Order](#)" (PDF). Extraordinary Chambers in the Courts of Cambodia. 15 September 2010. Retrieved April 2012.

63. [Jump up](#) [^] ["002/19-09-2007: Decision on immediate appeal against Trial Chamber's order to release the accused Ieng Thirith"](#)(PDF). Extraordinary Chambers in the Courts of Cambodia. 13 December 2011. Retrieved April 2012.
64. [Jump up](#) [^] [Statement by Carolyn Willson, Minister Counselor for International Legal Affairs, on the Report of the ICC, in the UN General Assembly](#) PDF (123 KB) 23 November 2005
65. [Jump up](#) [^] Jafari, Jamal and [Paul Williams](#) (2005) ["Word Games: The UN and Genocide in Darfur"](#) *JURIST*
66. [Jump up](#) [^] [POWELL DECLARES KILLING IN DARFUR 'GENOCIDE'](#), [The NewsHour with Jim Lehrer](#), 9 September 2004
67. [^] [Jump up to:](#) ^a ^b [Report of the International Commission of Inquiry on Darfur to the United Nations Secretary-General](#) PDF (1.14 MB), 25 January 2005, at 4
68. [Jump up](#) [^] [Security Council Resolution 1593 \(2005\)](#) PDF (24.8 KB)
69. [Jump up](#) [^] [SECURITY COUNCIL REFERS SITUATION IN DARFUR, SUDAN, TO PROSECUTOR OF INTERNATIONAL CRIMINAL COURT](#), UN Press Release SC/8351, 31 March 2005
70. [Jump up](#) [^] [Fourth Report of the Prosecutor of the International Criminal Court, to the Security Council pursuant to UNSC 1593 \(2005\)](#) PDF (597 KB), Office of the Prosecutor of the International Criminal Court, 14 December 2006.
71. [Jump up](#) [^] [Statement by Mr. Luis Moreno Ocampo, Prosecutor of the International Criminal Court, to the United Nations Security Council pursuant to UNSCR 1593 \(2005\), International Criminal Court](#), 5 June 2008
72. [Jump up](#) [^] [ICC issues a warrant of arrest for Omar Al Bashir, President of Sudan](#) (ICC-CPI-20090304-PR394), ICC press release, 4 March 2009
73. [Jump up](#) [^] [Adam Jones](#) (2010), *Genocide: A Comprehensive Introduction* (2nd ed.), p.271.
74. [Jump up](#) [^] [Pair guilty of 'insulting Turkey'](#), *BBC News*, October 11, 2007.
75. [Jump up](#) [^] [Rubinstein, W. D.](#) (2004). *Genocide: a history*. Pearson Education. p.7. [ISBN 0-582-50601-8](#)
76. [^] [Jump up to:](#) ^a ^b M. Hassan Kakar [Chapter 4. The Story of Genocide in Afghanistan](#) Footnote 9. Citing Horowitz, quoted in Chalk and Jonassohn, *Genocide*, 14.
77. [Jump up](#) [^] M. Hassan Kakar [Chapter 4. The Story of Genocide in Afghanistan](#) Footnote 10. Citing For details, see Carlton, *War and Ideology*.
78. [Jump up](#) [^] M. Hassan Kakar, [Afghanistan: The Soviet Invasion and the Afghan Response, 1979–1982](#), [University of California](#) Press, 1995.

79. ^ [Jump up to:](#)^{a b} [Gregory Stanton. The 8 Stages of Genocide, Genocide Watch](#), 1996
80. [Jump up](#)^ The [FBI](#) has found somewhat similar stages for [hate groups](#).
81. [Jump up](#)^ <http://aipr.wordpress.com/2012/04/19/genprev-in-the-news-19-april-2012/>
82. [Jump up](#)^ Dirk Moses [Why the Discipline of "Genocide Studies" Has Trouble Explaining How Genocides End?](#), [Social Science Research Council](#), 22 December 2006
83. [Jump up](#)^ Fein, H. (1979). Accounting for genocide: Victims and survivors of the Holocaust. New York: Free Press.^[page needed]
84. [Jump up](#)^ Staub, E (1989). The roots of evil: The origins of genocide and other group violence. New York: Cambridge University Press.^[page needed]
85. [Jump up](#)^ Staub, E. (2011) [Overcoming evil: Genocide, violent conflict and terrorism](#) New York: Oxford University Press.^[page needed]