Consensus Statement Update on Posttraumatic Stress Disorder From the International Consensus Group on Depression and Anxiety

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Objective: To provide an update to the "Consensus Statement on Posttraumatic Stress Disorder From the International Consensus Group on Depression and Anxiety" that was published in a supplement to *The Journal of Clinical Psychiatry* (2000) by presenting important developments in the field, the latest recommendations for patient care, and suggestions for future research. *Participants:* The 4 members of the International Consensus Group on Depression and Anxiety were James C. Ballenger (chair), Jonathan R. T. Davidson, Yves Lecrubier, and David J. Nutt. Other faculty who were invited by the chair were Randall D. Marshall, Charles B. Nemeroff, Arieh Y. Shalev, and Rachel Yehuda. *Evidence:* The consensus statement is based on the 7 review articles in this supplement and the related scientific literature. *Consensus process:* Group meetings were held over a 2-day period. On day 1, the group discussed topics to be represented by the 7 review articles in this supplement, and the chair identified key issues for further debate. On day 2, the group discussed these issues to arrive at a consensus view. After the group meetings, the consensus statement was drafted by the chair and approved by all faculty. *Conclusion:* There have been advancements in the science and treatment of posttraumatic stress disorder. Attention to this disorder has increased with recent world events; however, continued efforts are needed to improve diagnosis, treatment, and prevention of posttraumatic stress disorder. *(J Clin Psychiatry 2004;65[suppl 1]:55–62)*

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The original Consensus Statement on Posttraumatic Stress Disorder (PTSD) from the International Consensus Group on Depression and Anxiety was published in 2000.[1] The purpose of that document was to increase awareness of PTSD by reviewing clinical presentation, prevalence, personal and societal burden, neurobiology, risk factors, clinical course, and clinical management. The consequences of exposure to traumatic events remain a highly active area of research in the domains of neurobiology, epidemiology, public health, prevention, and treatment. In keeping with the objectives of prior publications by our group, this update to the original Consensus Statement on PTSD strives to address important recent developments in the field, to provide clinicians with current recommendations for patient care, and to suggest areas in need of further research.

IMPLICATIONS OF EARLY-LIFE TRAUMA

Early-life trauma is now recognized as a significant risk factor for the development of psychiatric illness in

later life, including PTSD and major depressive disorder.[2] Ethological research suggests that experience of trauma during key development periods can result in persistent changes in brain morphology and function and lead to increased vulnerability to subsequent adversities.[2,3] Neurochemical and structural brain changes resulting from early developmental stress in animal models[4–6] and in maltreated children[7–9] are well documented. In contrast, the long-term effects of early-life trauma are just beginning to be studied in adults, and persistent morphologic changes in the brain[10] and alterations in neurotransmitter[11,12] and neuroendocrine[13,14] function have been demonstrated in PTSD populations as well as in others. Hopefully this burgeoning area of research will shed light on vulnerability and resilience factors associated with the development of PTSD and other psychiatric disorders.

Research Needs

1. Studies that examine the relationship between trauma-related alterations in brain morphology and function and clinical manifestations of psychiatric disorder and vulnerability.

2. Prospective studies of the neurobiological effects and long-term sequelae of early-life trauma. What is the effect of exposure to trauma during periods of high neuronal plasticity? Does trauma change brain function and morphology, and are the effects reversible?

3. Studies that expand the definition of early-life stress to include the trauma experienced by premature infants in a neonatal intensive care unit and by children who are exposed to prenatal insult in utero.

4. Differentiation of early-life trauma from emotional and/or physical neglect and prospective follow-up of the neurobiologic and behavioral sequelae of each.

5. Studies designed to differentiate the consequences of early-life trauma from those of trauma in adulthood. Does the adult brain respond differently than the infant/child one? Are the short-term and long-term consequences different? Does trauma in early life lead to different pathologies than trauma in adulthood?

6. Assessment of whether psychopharmacologic agents that are effective in the treatment of PTSD or psychosocial interventions can prevent or reverse the neurobiological changes associated with early-life trauma. For example, will pharmacologic interventions (once developed) that target alterations in the corticotropin-releasing factor (CRF) systems (e.g., new putative treatments such as CRF receptor antagonists, glucocorticoid receptor antagonists, or currently useful treatment such as selective serotonin reuptake inhibitors [SSRIs]) reverse the neurobiologic changes?

7. More longitudinal studies of the natural course of PTSD or other psychiatric disorders in persons who experienced early-life trauma. There are some data on the influence of childhood adversity on the development of psychiatric disorders during adolescence and adulthood,[15–17] yet additional studies are needed.

IMPLICATIONS OF NEUROBIOLOGICAL/ NEUROIMAGING STUDIES IN PTSD

Tremendous advances in our understanding of the neurobiology of PTSD have been made in recent years. Neuroendocrine changes have been demonstrated involving the hypothalamic-pituitary-adrenal (HPA) axis in which patients with PTSD hypersecrete CRF but exhibit paradoxically low to normal cortisol levels in comparison to normal and depressed patients.[10,18,19] This finding is nonspecific to PTSD but does appear to be found in a limited number of medical and psychiatric conditions.[20] The most consistent HPA axis finding in PTSD is supersuppression of cortisol following a low-dose dexamethasone test in chronic PTSD across different patient populations.[20,21] It would be particularly useful to pursue comparisons across the other anxiety disorders, as this could inform more specific models of the relationships between clinical syndromes, peripheral endocrine systems, and central circuitry.[22]

Disruptions in the neurotransmitter systems involving norepinephrine, serotonin, glutamate, γ-aminobutyric acid (GABA), and endogenous opioids are also believed to play a role in the pathophysiology of PTSD.[23] We are beginning to study the neural circuits involved in the disturbances of sensory and memory processing and identify the loci for these functional deficits. Several structural neuroimaging studies have demonstrated reduced hippocampal volume in adults with PTSD, which may eventually prove useful in identifying at-risk individuals.[24,25] In studies of monozygotic twin pairs,[26] severity of PTSD negatively correlated with hippocampal volume of the twin exposed to trauma and the trauma-unexposed identical co-twin. Additionally, PTSD patients and their trauma-unexposed twins had significantly smaller hippocampi compared with twin pairs in which neither member had PTSD, which suggests that reduced hippocampal volume may be a preexisting trait linked in some way to a vulnerability for development of PTSD.

The field should be encouraged to conduct hypothesis-driven studies and, in particular, to exercise caution about overinterpreting single findings. Hopefully, neurobiologic and neuroimaging advances will shed light on which persons are at risk for PTSD or which factors are associated with treatment response and nonresponse.

Research Needs

1. Imaging studies to further characterize the neurocircuitry involved in the acute and chronic response

to trauma in normal subjects and patients with PTSD in other psychiatric populations.

2. Neurobiological probe studies that identify biological markers of risk for PTSD, such as CRF receptor ligand studies that identify at-risk persons with low CRF receptor density who hypersecrete CRF. Additionally, tryptophan depletion studies, which have been conducted in most other mood and anxiety disorders, are needed in PTSD.

3. Longitudinal studies that measure biological markers in high-risk persons before exposure to trauma and again after a traumatic event.

4. Studies of the serotonin transporters and receptors in PTSD patients during SSRI treatment to assess the relationship between the magnitude of transporter occupancy, receptor changes, and the completeness of the clinical response.

RISK AND RESILIENCE

One of the most enduring and interesting facets of PTSD is that, of the population of persons who experience a traumatic event, only a subset will ultimately develop the disorder. Neither trauma exposure nor a strong family history of the disorder is sufficient to cause PTSD.[27] Individual differences are clearly important, but what factors predispose a trauma victim to develop PTSD? Are neuroendocrine findings, such as low to normal baseline cortisol, markers for the ultimate development of PTSD? Of equal or possibly even greater interest are questions about which factors or conditions protect against the development of PTSD.

The posttrauma environment has been shown to be perhaps the most important predictor of chronicity.[28] If an adverse environment can increase risk of PTSD after trauma, it is possible that enhanced posttrauma support or other interventions can reduce risk (i.e., promote resilience). Although research is limited, anecdotal experience suggests that attempts to facilitate a positive recovery environment by controlling unrestrained responses, alleviating secondary stressors, and enabling access to community support services may promote resilience in the aftermath of a traumatic experience, particularly after large-scale disasters or other forms of mass trauma.[29] Clearly, the response to trauma must be understood in the context of both vulnerability and resilience.

Risk research in psychiatry is an area of active study,[30] especially in PTSD. What is emerging is a sense that in adults, the presence of and interplay among the 3 factors of genetics, the nature of the trauma, and the recovery environment, work together to contribute to an individual's vulnerability or resilience to PTSD. The entire spectrum of early-life adverse events should be considered in studies of risk factors for PTSD. This includes very early-life events, such as may occur in premature infants who begin

life with an extended stay in a neonatal intensive care unit. Technological advances in the field of neonatology have enabled even extremely low–birth-weight infants to survive. However, the intensive care unit environment is, by necessity, a traumatic experience in which infants undergo multiple, regular invasive procedures, often without anesthesia. Care of these infants often requires administration of high-dose glucocorticoids, immobilization, lack of physical contact with a maternal surrogate, and constant light. The short- and long-term neurobiological and behavioral sequelae of these experiences are not known. Also, there is a growing literature on the outcome of children who experience prenatal insult (e.g., maternal infection, abuse of the mother, etc.)[31,32]

Research Needs

1. Family and genetic studies that assess factors associated with vulnerability or resilience to the psychological consequences of trauma. Investigators and study sponsors should be encouraged to pursue collection and banking of blood samples for purposes of genomic research in all clinical trials.

2. Development of standardized measures of vulnerability and resilience and assessment of how and to what degree risk can be mitigated.

3. Studies that assess the role of trauma type and severity, the individual response to trauma, and the effect of the recovery environment on subsequent development of psychopathology. These studies would identify factors that increase vulnerability or confer resilience to development of pathology as well as factors that are associated with vulnerability to subsequent trauma. The findings of these studies would inform primary and secondary prevention efforts.

4. Studies of multiple trauma exposures after an initial event, as in ongoing domestic violence in an adult who was sexually abused as a child, in order to determine the effect of early trauma on development of subsequent pathology.

5. Differentiation between risk of onset and risk of chronicity in the above studies, as has been fruitful in research with other psychiatric disorders.

DIAGNOSIS

Two major themes emerged as important unmet needs in the diagnosis of PTSD. The diagnosis of PTSD and acute stress disorder (ASD), despite revisions in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III, III-R, and IV), remain conceptually and clinically problematic.[33] Secondly, prior history of trauma and PTSD symptoms continues to be too-often overlooked both in the clinical setting and in the design of epidemiologic, genetic, and treatment studies of psychiatric illness.

Diagnostic Approach to PTSD

As stipulated in the DSM-IV,[34] a diagnosis of PTSD requires the development of a characteristic set of symptoms subsequent to a traumatic experience. According to the DSM-IV, persons with PTSD react to the trauma with intense fear, helplessness, or horror and develop symptoms of reexperiencing, avoidance of cues associated with the trauma, and hyperarousal lasting for more than 1 month. Although the widespread use of these diagnostic criteria has significantly advanced patient care, strong consideration should be given to a reevaluation of the nosology of PTSD. The fact that the International Statistical Classification of Diseases and Related Health Problems, Tenth Edition (ICD-10) diagnostic scheme for posttraumatic symptoms differs fundamentally from that of the DSM-IV suggests that fundamental concepts in the field continue to be controversial.[35] Thought should be given to placing greater emphasis on the frequency and occurrence of mood and anxiety symptoms. Also, consideration should be given to including a broader range of traumatic experiences than is presently described in the DSM-IV, particularly in the domain of adverse early-life events.

A DSM-IV diagnosis of PTSD and other mood and anxiety disorders requires categorical fulfillment of specific criteria, without which symptoms may be considered subsyndromal. There was consensus that PTSD and indeed some other major psychiatric disorders may better fit a diagnostic model that is dimensional rather than categorical in nature.[34,36] Categorical definitions of complex psychopathology have not always withstood the test of time. A preferred research diagnostic approach to the phenomenology of mood and anxiety disorders, including PTSD, might be to use both dimensional and categorical perspectives on the consequences of trauma. Until etiology is better understood for PTSD, however, a syndromal model will likely continue to be an efficient and scientifically valid way to study the relationship between clinical phenomenology and its biological correlates.[37] Associated features that occur in the aftermath of trauma in persons with an Axis I disorder should not be misinterpreted or misdiagnosed as an Axis II disorder.

Trauma Histories

A second theme of concern is that a history of trauma is very frequently missed, or never even queried, in both clinical and research settings. The pivotal features that suggest risk for PTSD or the presence of undiagnosed PTSD are exposure to traumatic events and a significant, negative change in life trajectory subsequent to the trauma. Clinicians are encouraged to always inquire about past and current trauma exposure in patients with mood/anxiety symptoms, dysfunctional personal relationships, substance/alcohol abuse, and vague, medically unexplained somatic complaints. Patients should be queried about major changes in their lives, such as health, well-being, and function at work, in school, and at home. Even the simple act of asking patients if they ever experienced significant trauma and if that experience had a lasting impact or changed their life may introduce a discussion that leads to a diagnosis and treatment.

Research Needs

1. Continued theoretical and empirical research into a nosology of the consequences of criterion A trauma.
2. Studies evaluating broader, more dimensional diagnostic criteria for PTSD that feature mood and anxiety symptoms, substance/alcohol abuse, dysfunctional interpersonal relationships, and overall symptom severity.
3. Studies that develop and validate standardized methodology for obtaining trauma histories in both the research and clinical setting. Such studies would facilitate assessment and rating of the importance of salient life events.
4. Studies that identify and quantify trauma severity.

TREATMENT

The group addressed several issues regarding treatment of PTSD, beginning with a discussion regarding how soon after a traumatic event definitive treatment should be initiated. Unfortunately, it is not known which symptom features occurring in the immediate or short-term aftermath of a trauma indicate a need for treatment. Furthermore, the likelihood of different types of PTSD symptoms vary dramatically with type and severity of trauma, such that there may be an interaction between type and severity of trauma, and normative versus pathologic acute symptom profile. Because some degree of distress after a trauma is normative and resolves in most individuals, pharmacotherapy and directed psychotherapeutic interventions should not be considered indiscriminately for all persons who exhibit posttrauma distress.

In the absence of definitive research, the group developed several practical recommendations. Persons who seek help after experiencing a trauma and who are in extreme distress (e.g., dissociating, inability to sleep) should receive symptomatic treatment. Although this approach generally should not be considered as preventive therapy for PTSD, short courses of cognitive-behavioral therapy (CBT) initiated soon after the trauma have been associated with lower rates of PTSD 3 months[38] or 6 months[39–41] later.

In contrast, individuals who continue to be markedly symptomatic after 3 to 4 weeks and who, after 2 or more serial assessments, have not improved or whose condition has deteriorated should be treated with psychotherapy, pharmacotherapy, or a combination of both. If the symptoms are sufficiently severe to prompt the patient to seek treatment and if the symptoms have resulted in continuing

social, interpersonal, and/or occupational impairment for at least 3 to 4 weeks, most clinicians recommend that definitive treatment be initiated. Symptoms suggesting the need for treatment include depressed mood, hyperarousal, intrusive recollections, avoidance, dissociation, and severely disrupted sleep.

There is a void in our understanding of the optimal therapeutic strategy for patients needing early intervention. However, for established PTSD, the efficacy of the SSRIs is supported by a large database of randomized, controlled trials.[42] The benzodiazepines are not effective for PTSD,[43] and single-session critical incident stress debriefing (CISD) has no preventative effect and may be harmful.[44,45] Though definitive data are lacking, treatment of established PTSD might be modeled after that used in other psychiatric disorders (e.g., schizophrenia, bipolar disorder, major depression) in which early intervention is more beneficial than delayed treatment. In the case of PTSD, it was concluded that definitive treatment with SSRIs and/or CBT should be initiated within 3 to 4 weeks of substantial symptomatology.

Long-Term Treatment

Because of preliminary evidence of the risk of relapse following discontinuation of pharmacotherapy in patients with chronic PTSD,[46] long-term treatment is often needed. In the absence of definitive research, it is recommended that, for patients with chronic PTSD (defined by the DSM-IV as full-criterion symptoms lasting 3 months or more), medication should be continued for at least 1 year, with regularly scheduled follow-up in order to prevent relapse. There is now a robust body of data on the efficacy of SSRI treatment of chronic PTSD. Concomitant CBT may increase treatment response or increase the durability of response as has been shown in social phobia.[47] Because CBT and the SSRIs are likely to have complementary rather than redundant mechanisms of action, combination treatment may be superior to either CBT or the SSRIs alone; in other cases, CBT may be effective for symptoms that do not respond to SSRIs.[48] Both hypotheses deserve systematic study.

Childhood Trauma and Psychopathology in an Adult

The presence of childhood trauma increases risk for a fairly wide range of psychiatric disorders and problems, including personality disorders.[2,16] The optimal and recommended approach for treating patients who present with current psychopathology and a history of childhood trauma is to address the current illness and assess the extent to which the relevant history contributes to or is part of the current problem.

Research Needs

1. Evaluation of the efficacy of early preventive treatment in high-risk patients.

2. Identification of clinical and biological markers suggesting the need for early treatment.
3. Further assessment of the role, if any, of non-antidepressant psychopharmacologic agents, such as antiadrenergic or mood-stabilizing agents, in the treatment of PTSD.
4. Studies to assess efficacy of management strategies to improve adherence to therapeutic regimens.
5. Studies of combined psychotherapy and pharmacotherapy to maximize treatment response and prevent relapse after treatment discontinuation.
6. Studies to identify patients with chronic PTSD who may be candidates for short- versus long-term treatment.
7. Studies of new theoretically useful agents such as CRF_1 receptor antagonists and other HPA modulators to prevent PTSD after trauma or to treat established PTSD.

PUBLIC HEALTH RECOMMENDATIONS

Ideally, the public health response in the aftermath of a mass trauma or large-scale disaster should serve multiple functions and therefore include coordinated needs assessment, case identification, treatment, training, and research efforts. More education is needed for mental health and medical professionals as to indications for specialized treatment versus short-term support. The current public education philosophy guided by the Federal Emergency Management Agency (FEMA) emphasizes the normative response to trauma, and these materials should probably include both negative (e.g., fear, helplessness) and positive (e.g., volunteerism, physical alertness) reactions. Our public educational efforts should promote resilience and recovery and discourage illness-promoting behavior (such as avoidance or catastrophic thinking or behavior).

It is imperative that therapeutic interventions for posttrauma psychopathology be provided exclusively by appropriately trained mental health professionals. Delayed-onset PTSD is extremely rare after community disaster.[49] Indiscriminate use of therapeutic interventions that have not been shown to be effective or may even be harmful, such as CISD,[44,45] should be avoided after serious trauma.

Although much is known about the basic mental health epidemiology after disaster, it is tragic that opportunities for the study of prevention and treatment of posttrauma psychopathology after disaster have been missed.[50] Policy makers must improve procedures to enable the initiation of rapid-response clinical trials in the immediate aftermath of a disaster. In addition, more efficient funding mechanisms must be in place to ensure that studies receive proper resources in a timely manner. Available services must distinguish wherever possible between victims who need immediate referral to trained mental health pro-

fessionals from those persons who will benefit from more basic community support. The mental health research community also has responsibilities in this domain. Because the treatment of PTSD is a relatively new area of study, most communities will not have sufficient capacity to provide evidence-based treatment after a major disaster. Usually, when a professional is also a member of the injured community, there is a genuine desire to help, and for many clinicians, this desire would be best served by obtaining additional training.

Research Needs

1. Expansion of early prevention/treatment research in trauma victims.
2. Studies evaluating public outreach and educational intervention programs, particularly in populations exposed to mass trauma.
3. Studies identifying persons who will benefit from early therapeutic intervention following disaster or mass trauma, including study of when to begin those treatments.

PRIMARY CARE RECOMMENDATIONS

This Consensus Group considered 2 scenarios related to PTSD as germane to the primary care setting: the patient who seeks help because of distress associated with a trauma and the patient who presents with either a symptom or symptom cluster (e.g., insomnia) or with comorbid disorders such as depression, anxiety, or substance/alcohol abuse or medically unexplained somatic symptoms.

Treatment-Seeking Patients
Following Trauma Exposure

The original Consensus Statement outlined a 3-tiered approach to the management of PTSD, which consists of education, psychotherapeutic support and/or treatment (i.e., CBT), and medication. The current Consensus Group emphasized the continued importance of psychoeducation and reinforcement of health-promoting behaviors by the primary care physician until further research can be conducted in this important but generally neglected area of public health. Primary care patients should be educated about the range of common responses to trauma, including both positive and negative psychological reactions. Single-session psychological debriefing is not recommended. The presence of extreme distress, such as inability to sleep, warrants treatment. The group recommended a short course of a nonbenzodiazepine sedative-hypnotic in this setting and cautioned against the use of traditional benzodiazepines because of associated withdrawal symptoms, lack of efficacy in the treatment of depression and PTSD, and interactions with alcohol.

Although the relief of distress in the immediate aftermath of a traumatic experience is considered important,

Table 1. Recommended Daily Doses of Selective Serotonin Reuptake Inhibitors (SSRIs) for Treatment of Posttraumatic Stress Disorder[a]

SSRI	Initial Dose (mg)	Usual Dose (mg)	Maximum Dose[b] (mg)
Paroxetine IR	10–20	20–50	50
Paroxetine CR	12.5–25	25–75	75
Sertraline	25–50	50–150	200
Fluoxetine	10–20	20–50	80
Fluvoxamine	50	100–250	300
Citalopram	20	20–40	60

[a]Adapted with permission from Foa et al.[51]
[b]Based on package insert.
Abbreviations: CR = controlled release, IR = immediate release.

clinicians should not rely on observations made during a single office visit to diagnose and begin treatment for PTSD. Symptoms of distress, such as anxiety, insomnia, nightmares, depressed affect, and irritability, are common, but transient in many persons. Before beginning directed pharmacotherapy, primary care physicians should note an accumulation of symptoms after the acute presentation, a lack of improvement or deterioration in the clinical picture, and the persistence or emergence of disability during a 3- to 4-week observation period.

Primary care is an ideal setting in which to educate patients about treatment options for PTSD, which are medication and/or psychotherapy. Patients should learn that psychotherapy for PTSD requires special training and should be referred to a therapist who has experience providing CBT. Such educational efforts must include informing patients that if they respond to medication therapy, treatment for 1 year or longer may be needed. Patients and family members should be warned that information about PTSD that is obtained from the Internet should be interpreted with caution. Internet sites from established health care agencies or patient advocacy organizations are recommended over chat rooms or nonspecialist or commercial sites.

The SSRIs must be considered first-line psychopharmacologic treatment for PTSD because of their demonstrated efficacy in this disorder, relative safety in overdose, and efficacy in the treatment of other common comorbid psychiatric disorders (e.g., depression, panic disorder).[1,51] As outlined in treatment guidelines for primary care that were published in 1999,[51] a course of SSRI treatment should be initiated with low doses of an SSRI, which are increased gradually to the maximally effective and tolerated dose (Table 1). An initial 2- to 3-month course of antidepressant treatment is warranted, and the patient should be observed for improvement in specific symptoms, including intrusive reexperiencing (flashbacks, nightmares), hyperarousal (insomnia, startle), and avoidance. If no response is observed after 8 weeks of full, therapeutic doses, consideration can be given to switching to another SSRI or either venlafaxine or mirtazapine. Patients with partial responses may need longer to respond to

the first medication (SSRI). Responding patients should continue on full-dose medication for 12 months or longer as required by the severity of their illness.

Psychotherapy or pharmacotherapy alone is often appropriate for patients with mild PTSD, but a combined treatment strategy is warranted for more severely ill patients. Psychological treatments for PTSD consist of cognitive-behavioral approaches, including exposure therapy, stress inoculation training, and cognitive therapy.[1] Psychotherapy for PTSD generally requires referral to a specialist who is trained in cognitive-behavioral management techniques. Referral to specialized psychiatric care also should be considered for patients who do not respond to an adequate trial of full-dose pharmacotherapy or those who have significant psychiatric comorbidity, suicidality, or ongoing life stressors.

Consider PTSD in High-Risk Patients

PTSD remains a remarkably underrecognized and underdiagnosed illness in primary care patients for many reasons. Patients may be reluctant to acknowledge past or current histories of domestic violence or physical or sexual abuse, or they may not consider these traumatic experiences to be related to their current health problems. Primary care physicians are encouraged to maintain a high degree of clinical suspicion for past or present trauma in their patients who present with medically unexplained somatic symptoms, depression, substance or alcohol abuse, suicidal thoughts or behavior, and dysfunctional lives. By simply asking patients about negative experiences that were sufficiently severe to change their lives, primary care physicians are in an ideal position to improve the recognition of PTSD in their practices.

Research Needs

1. More epidemiologic studies to determine prevalence of PTSD in primary care.
2. Development and validation of clinically useful tools for the screening and diagnosis of PTSD in primary care.

CONCLUSIONS

In this publication, we have attempted to expand upon the principles that were outlined in our original Consensus Statement on PTSD[1] by identifying broad goals and unmet needs for research and clinical practice. It is our hope that these efforts will encourage further study and continued improvements in patient care:

- Increase awareness for traumatic experiences that occur in early life and understand the potential long-term psychological sequelae
- Better understand and delineate the factors leading to vulnerability and resilience to PTSD

- Identify risk factors and optimal treatment strategies for PTSD among persons who are exposed to large-scale disasters or other forms of mass trauma
- Support the efforts of public health organizations and governmental agencies in educating the public about health-promoting behaviors, normative reactions to trauma, and availability of support services, as well as the more appropriate use of trained mental health professionals in the diagnosis and treatment of the psychological sequelae of trauma in the subset of individuals with severe and persistent symptoms
- Appreciate the emerging neuroscience knowledge base, which offers the promise of developing biological markers to identify at-risk persons, predict treatment response, and develop targeted pharmacotherapeutic agents for PTSD
- Expand existing diagnostic strategies for PTSD to include early-life adversity and a more dimensional view of the usual symptom profile of patients with this disorder
- Encourage clinicians and researchers in other areas to be more cognizant of trauma and its short-term and long-term sequelae by integrating trauma histories into their routine clinical and research assessment procedures.

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