Research on Posttraumatic Stress Disorder

MRI study of corpus callosum in patients with borderline personality *disorder***: A pilot study.** By: Zanetti, Marcus V. ; Soloff, Paul H.; Nicoletti, Mark A.; Hatch, John P.; Brambilla, Paolo; Keshavan, Matcheri S.; Soares, Jair C. Progress in Neuro-Psychopharmacology & Biological Psychiatry, Oct2007, Vol. 31 Issue 7, p1519-1525, 7p

Abstract: This pilot study examined the integrity of the corpus callosum in a sample of patients with borderline personality ***disorder*** (BPD), as abnormalities in inter-hemispheric communication could possibly be involved in illness pathophysiology. We utilized magnetic resonance imaging (MRI) signal intensity (SI) and morphometric measures. Ten BPD and 20 healthy control subjects were assessed for current and past Axis I and Axis II comorbidities and histories of childhood abuse. Regional CC SI and areas were measured with semi-automated software from three-dimensional gradient echo imaging scans. Analysis of covariance was conducted to evaluate the results. No significant differences were observed between BPD and controls in the SI or area of any CC region. Abnormalities in interhemispheric connectivity do not appear necessary for the development of BPD. Further studies with larger samples are needed to confirm this preliminary finding.

STRESS **SENSITIZATION AND FEAR LEARNING IN POSTTRAUMATIC** STRESS DISORDER**.** Psychophysiology, Sep2007 Supplement 1, Vol. 44, pS8-S9, 1p;

Abstract: The article presents abstracts of psychophysiological research. They include "Endocrine and Inflammatory Dysregulations in Posttraumatic ***Stress Disorder,"*** by Nicholas Rohleder and colleagues, "***Stress***-Induced Analgesia in Posttraumatic ***Stress Disorder***: An FMRI Study," by Michèle Wessa and colleagues and "Generalization of Conditioned Fear as a Pathogenic Marker of PTSD," by Shmuel Lissek and colleagues.

STRESS SENSITIZATION AND FEAR LEARNING IN POSTTRAUMATIC STRESS DISORDER Chair(s): Miche` le Wessa1, & Anke Karl2, 1University of Heidelberg'', 2University of Southampton

Abstract: Stress sensitization and altered fear learning have been proposed to be at the core of the development and maintenance of posttraumatic stress disorder (PTSD). Increased stress reactivity has been suggested to result in symptoms of hyperarousal, and its probable compensation by emotional numbing symptoms has been compared to the mechanism of stress-induced analgesia (SIA). On a physiological level, stress reactivity and hyperarousal have been found to covary with an increased central activity of endogenous stress systems, such as the hypothalamic-pituitary axis (HPA) or sympathetic-adrenal-medullary (SAM) system. Interestingly, central neuropeptides and peripheral hormones have been shown to impact fear conditioning, a process centrally implicated in the etiology of PTSD. This symposium will focus on psychobiological measures of both stress sensitization and fear conditioning in PTSD patients. Rohleder et al. present findings on alterations in endogeneous stress systems and their relation to the disinhibition of inflammatory mechanisms in PTSD patients. Wessa et al.'s study provides evidence for subjectively and centrally altered SIA in PTSD patients, which is related to increased emotional numbing. Lissek et al.extends previous findings on fear conditioning in PTSD by reporting generalization of conditioned fear in PTSD patients. Karl et al.'s treatment study indicates that some physiological variables represent learned fear responses which are no longer present after successful therapy, while others represent a generally increased physiological reactivity.

ENDOCRINE AND INFLAMMATORY DYSREGULATIONS IN POSTTRAUMATIC STRESS DISORDER Nicolas Rohleder1, Jutta M. Wolf1, Ljiljana Joksimovic2, & Clemens Kirschbaum3 1University of British Columbia, 2University of Du¨ sseldorf, 3Dresden University of Technology

Abstract: Posttraumatic stress disorder (PTSD) develops in response to severe psychological trauma and its psychiatric characteristics have been well described. In addition, it has been recognized that PTSD is not only accompanied by poor health but also by a number of specific and non-specific somatic pathologies such as cardiovascular, autoimmune and physical complaints/chronic pain. It has been hypothesized that alterations of the hypothalamic-pituitary (HPA) axis, the sympathetic-adrenal-medullary (SAM) and the immune system may mediate or facilitate these somatic conditions. We will

present data that is consistent with an over-active SAM system characterized by higher daily activity of salivary alpha-amylase, and an under-active HPA axis, characterized by lower daily cortisol secretion. Patients also presented with an increased in-vitro inflammatory response, and altered regulation of the inflammatory response by anti-inflammatory endocrine signals. These findings are consistent with the hypothesis that the specific alterations of HPA axis and SAM system permit disinhibition of inflammatory mechanisms, which in turn foster the development of somatic diseases as well as self-reported physical complaints.

STRESS-INDUCED ANALGESIA IN POSTTRAUMATIC STRESS DISORDER: AN FMRI STUDY Miche` le Wessa, Slawomira Lipinski, Christoph Christmann, Stephanie Ridder, Simone Lang, & Herta Flor University of Heidelberg

Abstract: The inability to adaptively regulate levels of arousal and senzitization to stress have been proposed as core features of posttraumatic stress disorder (PTSD). Emotional numbing might represent one dysfunctional mechanism PTSD patients use to regulate their hyperarousal in analogy to the mechanism of stressinduced analgesia (SIA). Here, we investigated SIA in 12 PTSD patients, 12 trauma-exposed subjects without PTSD (nPTSD) and 12 healthy controls (HC) by use of a trauma-independent cognitive stressor (mental arithmetic plus noise). Participants underwent functional magnetic resonance imaging during painful stimulation before and after stress induction. Pain threshold and pain tolerance, self-reported pain intensity and unpleasantness ratings were sampled before and after stress induction. PTSD patients showed the most marked increase of pain threshold and pain tolerance and a decrease in pain ratings after stress induction. PTSD-patients displayed higher neural activity in brain regions related to pain modulation whereas both other groups activated more pain-processing brain structures. In PTSD patients, symptoms of emotional numbing were negatively correlated with neural activity post- versus pre-stress in brain areas related to the processing of pain. These findings point to enhanced stress-induced analgesia in PTSD patients on behavioural, self-report and neural levels. In addition, our findings suggest an association between emotional numbing and pain modulation.

GENERALIZATION OF CONDITIONED FEAR AS A PATHOGENIC MARKER OF PTSD Shmuel Lissek, Stephanie Rabin, Arter Biggs, Ruben Alvarez, Brian R. Cornwell, Meena Vythilingam, & Christian Grillon National Institute of Mental Health

Abstract: Heightened anxiety to conditioned stimuli (CSs) signaling safety is one of the more consistent conditioning correlates of PTSD found by lab-based studies. Whereas healthy controls display anxious arousal to CSs paired (CS1: danger cue) but not unpaired (CS – : safety cue) with an aversive unconditioned stimulus (US), PTSD patients tend to display fear responses to both CS1and CS – . **Given that CS1and CS – in this literature share many stimulus properties (e.g., size, shape, duration), such findings link PTSD to the tendency to generalize fear from danger cues to safety cues with overlapping features. This interpretation is consistent with the clinically observed PTSD process, by which fear to a traumatic event transfers to safe conditions that resemble aspects of the trauma (DSM-IV).** Unfortunately, little work on fear-generalization has been conducted in humans and no studies to our knowledge have systematically assessed generalization of conditioned fear in PTSD patients. The current effort introduces a generalization paradigm consisting of 10, quasi-randomly presented, rings of gradually increasing size. For half of participants the smallest ring is the CS1 (paired with an electric shock US) and for the other half, the largest serves as CS1. The 8 rings of intermediary size create a continuum of similarity from CS1to CS – and are included to assess generalization gradients of conditioned fear. Presented data provide psychophysiologic validation (startle EMG, SCR) of the paradigm as well as comparisons between generalization gradients among those with versus without PTSD. This work was supported by the Intramural Research Program of the National Institute of Mental Health.

BIOPSYCHOLOGICAL RISK FACTORS AND CORRELATES OF PTSD AND ITS SUCCESSFUL CBT TREATMENT Anke Karl1, Sirko Rabe2, Katja Pohnitzsch2, Tanja Zollner2, Andreas Maercker3, Klaus-Peter Lesch4, & Alexander Strobel2 1University of Southampton, 2Dresden University of Technology, 3University of Zurich, 4University of Wurzburg

Abstract: Affective processing in patients with PTSD is accompanied by a number of

psychophysiological alterations. In a recent study including treatment-seeking survivors of motor vehicle accidents, central (ERPs, EEG alpha asymmetry) and peripheral (heart rate, EMGstartle response) correlates of trauma-related stimulus processing were investigated pre and post treatment and at a 4month follow up. In addition, genetic samples were collected to determine the role of a polymorphism which indicates the efficiency of serotonergic neurotransmission (5HTTLPR). The short allele variant of this polymorphism has been related to serotonergic alterations and higher physiological reactivity (e.g., startle response). The results point to a pattern of increased heart rate, frontal P300 amplitude and EEG alpha asymmetry to trauma-related stimuli which was closely associated with the PTSD severity and thus the treatment outcome. On the other hand, contrary to our hypothesis, startle EMG and ERP (P200) were not related to PTSD and treatment outcome but to the presence of the short allele. Also, there was no evidence for a higher percentage of s allele carriers in the PTSD group. The results indicate that some physiological variables may indicate acquired aversive responses (e.g., conditioned fear) which are no longer present after successful CBT, while others may represent a generally increased physiological reactivity. The role of the latter for the etiology of PTSD needs to be further explored.

Fear conditioning in posttraumatic *stress disorder***: Evidence for delayed extinction of autonomic, experiential, and behavioural responses.** By: Blechert, Jens; Michael, Tanja; Vriends, Noortje; Margraf, Jürgen; Wilhelm, Frank H.. Behaviour Research & Therapy, Sep2007, Vol. 45 Issue 9, p2019-2033, 15p

Abstract: Aversive conditioning has been proposed as an important factor involved in the etiology of posttraumatic ***stress disorder*** (PTSD). However, it is not yet fully understood exactly which learning mechanisms are characteristic for PTSD. PTSD patients (), and healthy individuals with and without trauma exposure (TE group, ; nTE group,), underwent a differential fear conditioning experiment consisting of habituation, acquisition, and extinction phases. An electrical stimulus served as the unconditioned stimulus (US), and two neutral pictures as conditioned stimuli (, paired; , unpaired). Conditioned responses were quantified by skin conductance responses (SCRs), subjective ratings of CS valence and US-expectancy, and a behavioural test. In contrast to the nTE group, PTSD patients showed delayed extinction of SCRs to the . Online ratings of valence and US-expectancy as well as the behavioural test confirmed this pattern. These findings point to a deficit in extinction learning and highlight the role of affective valence appraisals and cognitive biases in PTSD. In addition, there was some evidence that a subgroup of PTSD patients had difficulties in learning the CS–US contingency, thereby providing preliminary evidence of reduced discrimination learning.

A Preliminary Investigation of the Relationship Between Emotion Regulation Difficulties and Posttraumatic *Stress* **Symptoms**. By: Tull, Matthew T.; Barrett, Heidi M.; McMillan, Elaine S.; Roemer, Lizabeth. Behavior Therapy, Sep2007, Vol. 38 Issue 3, p303-313, 11p, 4 charts

Abstract: This study examined the relationship between posttraumatic ***stress*** (PTS) symptoms and particular aspects of emotion regulation difficulties among trauma-exposed individuals. Participants were an ethnically diverse sample of 108 undergraduates from an urban university. PTS symptom severity was found to be associated with lack of emotional acceptance, difficulty engaging in goal-directed behavior when upset, impulse-control difficulties, limited access to effective emotion regulation strategies, and lack of emotional clarity. Further, overall difficulties in emotion regulation were associated with PTS symptom severity, controlling for negative affect. Finally, individuals exhibiting PTS symptoms indicative of a PTSD diagnosis reported greater difficulties with emotion regulation than those reporting PTS symptoms at a subthreshold level. The implications of these findings for research and treatment are discussed.

Adverse childhood experiences associated with sleep in primary insomnia. By: BADER, KLAUS; SCH&;#x00C4;FER, VALÉRIE; SCHENKEL, MAYA; NISSEN, LUKAS; SCHWANDER, JÜRG. Journal of Sleep Research, Sep2007, Vol. 16 Issue 3, p285-296, 12p, 4 charts

Abstract: The objectives were to explore the association between self-reported adverse childhood experiences (ACE) and sleep in adults suffering from primary insomnia and to examine the impact of presleep ***stress*** on this relationship. Fifty-nine patients with primary insomnia, aged 21–55 years, were administered the Childhood Trauma Questionnaire (CTQ) and then divided into two groups according to

the achieved scores: with moderate/severe or low/no reports of ACE. The participants spent three consecutive nights in the sleep laboratory in order to record polysomnographic and actigraphic sleep parameters. A ***stress*** induction technique was administered by activating negative autobiographical memories immediately before sleep in the second or third night. Results show that 46% of the insomniac patients reported moderate to severe ACE. This group exhibited a significantly greater number of awakenings and more movement arousals compared to patients with low or no reports of ACE. Actigraphic data also indicated more disturbed sleep and increased nocturnal activity for the high-ACE group. On the other hand, no specific group differences were found with regard to ***stress*** condition. The results support the assumption that it is possible to identify a subgroup among patients with primary insomnia who has experienced severe maltreatment in childhood and adolescence. This subgroup appears to differ in several sleep parameters, indicating a more disturbed sleep compared to primary insomniacs with low or no reports of ACE. With regard to sleep-disturbing nightly patterns of arousal, parallels between individuals with high ACE and trauma victims as well as ***post-traumatic stress disorder***-patients suggest themselves.

The Psychophysiology of Posttraumatic *Stress Disorder***: A Meta-Analysis.** By: Pole, Nnamdi. Psychological Bulletin, Sep2007, Vol. 133 Issue 5, p725-746, 22p, 6 charts
Abstract: This meta-analysis of 58 resting baseline studies, 25 startle studies, 17 standardized trauma cue studies, and 22 idiographic trauma cue studies compared adults with and without posttraumatic ***Stress disorder*** (PTSD) on psychophysiological variables: facial electromyography (EMG), heart rate (HR), skin conductance (SC), and blood pressure. Significant weighted mean effects of PTSD were observed for HR (r = .18) and SC (r = .08) in resting baseline studies; eyeblink EMG (r = .13), HR (r = .23), and SC habituation slope (r = .21) in startle studies; HR (r = .27) in standardized trauma cue studies; and frontalis EMG (r = .21), corrugator EMG (r = .34), HR (r = .22), and SC (r = .19) in idiographic trauma cue studies. The most robust correlates of PTSD were SC habituation slope, facial EMG during idiographic trauma cues, and HR during all study types. Overall, the results support the view that PTSD is associated with elevated psychophysiology. However, the generalizability of these findings is limited by characteristics of the published literature, including its disproportionate focus on male veterans and neglect of potential PTSD subtypes.

Post-traumatic stress disorder. Occupational Medicine, Aug2007, Vol. 57 Issue 6, p399-399, 1p
Abstract: ***Post-traumatic stress disorder*** (PTSD) is an increasingly recognized and potentially preventable condition. Certain factors, especially the severity of the trauma, perceived lack of social support and peri-***traumatic*** dissociation have been associated with its development. In recent years, a more robust evidence base regarding the management of individuals involved in ***traumatic*** events has emerged. Immediately after a ***traumatic*** event, simple practical, pragmatic support provided in a sympathetic manner by non-mental health professionals seems most likely to help. For individuals who develop persisting PTSD, trauma-focused cognitive behavioural therapy (TFCBT) may be beneficial within a few months of the trauma. For those who develop chronic PTSD, TFCBT and eye movement desensitization and reprocessing are best supported by the current evidence. Some anti-depressants appear to have a modest beneficial effect and are recommended as a second-line treatment. The current evidence base has allowed the development of guidelines that now require implementation. This has major implications in terms of planning and developing services that allow appropriately qualified and trained individuals to be available to cater adequately for the needs of survivors of ***traumatic*** events.

Does *stress* **during childhood damage the hippocampus?** Nature Clinical Practice Neurology, Aug2007, Vol. 3 Issue 8, p419-419, 1/2p;
Abstract: This article presents a study regarding the symptoms of ***post-traumatic stress disorders***. Studies of adults have established an association between symptoms of ***post-traumatic stress disorder*** and smaller hippocampus size, but these outcomes have not been replicated in children. It also discusses the hypothesis that the hippocampus damage in adults might result from chronic exposure to neurotoxic levels of cortisol induced by ***stress*** during childhood development.;

Violence Exposure and Psychopathology in Urban Youth: The Mediating Role of Posttraumatic Stress**.** By: Ruchkin, Vladislav; Henrich, Christopher C.; Jones, Stephanie M.; Vermeiren, Robert; Schwab-Stone, Mary. Journal of Abnormal Child Psychology, Aug2007, Vol. 35 Issue 4, p578-593, 16p
Abstract: Understanding the mechanisms underlying the development of violence exposure sequelae is essential to providing effective treatments for traumatized youth. This longitudinal study examined the mediating role of posttraumatic ***stress*** in the relationship between violence exposure and psychopathology, and compared the mediated models by gender. Urban adolescents (n=1,358) were surveyed using the Social and Health Assessment. The proposed relationships were examined using Structural Equation Modeling. Posttraumatic ***stress*** fully mediated the relationships between victimization and depression and anxiety in girls, and partially so in boys. In addition, posttraumatic ***stress*** partially mediated the relationships between violence exposure and commission of violence in boys. Current findings support the longitudinal effects of violence exposure on adolescent mental health. Posttraumatic ***stress*** represents a unique mechanism for the development of psychopathology in girls and is also related to negative outcomes in boys. These findings have direct implications for prevention and rehabilitation efforts among violence exposed youth.

Keeping memories at an arm's length: Vantage point of trauma memories. By: Kenny, Lucy M.; Bryant, Richard A.. Behaviour Research & Therapy, Aug2007, Vol. 45 Issue 8, p1915-1920, 6p
Abstract: This study investigated the relationship between memory vantage point and avoidance following trauma. Sixty trauma survivors with differing levels of avoidance were interviewed about the vantage point of their memory for trauma, a positive memory, and a neutral memory. Avoidant individuals were more likely to remember their trauma from an observer perspective than individuals with a lower level of avoidance. Avoidance did not influence vantage point for positive or neutral memories. These data support the proposal that adoption of the observer vantage point for trauma memories may serve an avoidant function for people affected by trauma.

Neurobiological Alterations Associated With ***Traumatic Stress***. By: Weiss, Sandra J.. Perspectives in Psychiatric Care, Jul2007, Vol. 43 Issue 3, p114-122, 9p Abstract: PURPOSE. The purpose of this article is to describe the effects of ***traumatic stress*** on brain structure and function, and the relationship of these neurobiological changes to symptoms experienced after trauma. CONCLUSIONS. Exposure to ***traumatic stress*** is associated with changes in the limbic system, the hypothalamic–pituitary–adrenal axis, and key monoamine neurotransmitters. Different neurobiological alterations can be linked to specific symptoms of hyperarousal, dissociation/numbing, and reexperiencing of the trauma. PRACTICE IMPLICATIONS. Understanding what is happening in the brain can inform more targeted treatment for various symptoms that the individual may be experiencing.

Posttraumatic Stress Disorder. By: Stevens, Lise M.. JAMA: Journal of the American Medical Association, 8/1/2007, Vol. 298 Issue 5, p588-588, 1p;
Abstract: This article presents the patient education page where the topic of discussion is ***post-traumatic stress disorder*** (PTSD). PTSD is the development of symptoms that last more than one month and make normal functioning difficult following a life-threatening experience. The symptoms include flashbacks of the trauma with physical reactions such as a racing heart beat, avoiding people, places, or things that can trigger memories, feeling numb or emotionless and hyperarousal where the victim feels on guard all the time, can't sleep and is easily startled. The people who are at risk for PTSD are outlined. Treatment suggestions are offered from therapy to medications.;

Posttraumatic Stress Disorder

After people experience a very stressful event, they may feel that they should be able to move on and "just handle it" or "get over it." Some experiences, however, are so traumatic that some individuals have serious problems coping and functioning in their daily lives afterward. They may have **posttraumatic stress disorder** (**PTSD**). The August 1, 2007, issue of *JAMA* is a theme issue on violence and human rights. This Patient Page is based on one previously published in the August 2, 2006, issue of *JAMA*.

WHAT IS POSTTRAUMATIC STRESS DISORDER (PTSD)?

Posttraumatic stress disorder is the development of characteristic symptoms that last for more than 1 month, along with difficulty functioning after exposure to a life-threatening experience.

SYMPTOMS

- Intrusion—memories of the trauma or "flashbacks" that occur unexpectedly; these may include nightmares or physical reactions such as a racing heart
- Avoidance—avoiding people, places, thoughts, or activities that bring back memories of the trauma; this may involve feeling numb or emotionless, withdrawing from family and friends, or "self-medicating" by abusing alcohol or other drugs
- Hyperarousal—feeling "on guard" or irritable, having sleep problems, having difficulty concentrating, feeling overly alert and being easily startled, having sudden outbursts of anger

- **WHO IS AT RISK FOR PTSD?**
- People with military combat experience or civilians who have been harmed by war
- People who have been raped, sexually abused, or physically abused
- People who have been involved in or who have witnessed a life-threatening event
- People who have been involved in a natural disaster, such as a tornado or an earthquake

TREATING PTSD

- Cognitive behavioral therapy with a trained psychiatrist, psychologist, or other professional can help change emotions, thoughts, and behaviors associated with PTSD and can facilitate managing panic, anger, and anxiety.
- Certain medications can reduce symptoms such as anxiety, impulsivity, depression, and insomnia and decrease urges to use alcohol and other drugs.
- Group therapy can help patients learn to communicate their feelings about the trauma and create a support network.
- Becoming informed about PTSD and sharing information with family and friends can create understanding and support during recovery.

Rethinking posttraumatic stress disorder. (cover story) Harvard Mental Health Letter, Aug2007, Vol. 24 Issue 2, p1-4, 4p;

Abstract: This article explores the pathology of ***post-traumatic stress disorder*** (PTSD). The authors begin by discussing three kinds of symptoms of PTSD: hyperarousal; re-experiencing or intrusion; and avoidance and emotional numbing. They cite some studies examining ***traumatic*** events and symptoms of PTSD. They also investigate the connection between gender and the development of PTSD. Further, the authors look at the diagnosis of the ***disorder***.;

Rethinking posttraumatic stress disorder

What is a traumatic event, and how does it produce symptoms?

"It could go on for years and years, and has, for centuries," wrote the author of the Sumerian epic of Gilgamesh in the third millennium, b.c., describing the suffering of a character who survived a violent encounter that killed his friend. That terrifying experiences oft en have lasting psychological consequences was well known for thousands of years before 1980, when the American Psychiatric Association classified posttraumatic stress disorder (PTSD) as a psychiatric disorder in the third edition of its diagnostic manual (DSM-III).

PTSD is one of the few psychiatric conditions to which the manual ascribes a definite cause. Although no one today doubts that emotional trauma can have devastating effects, a debate about this diagnosis has been ignited, and changes may be in store. War is a mother lode of traumatic experiences and the chief source of the concept of PTSD. In the American Civil War, the resulting symptoms were sometimes described as battle fatigue. In the First World War, it was called shell shock, and in the Second World War, combat neurosis or traumatic neurosis. Soldiers in those wars who succumbed to posttraumatic stress were sometimes regarded as weak or inadequate, but that view changed as understanding of their experiences improved. Physicians and mental health professionals came to see the symptoms as, in a sense, normal responses to abnormal circumstances. By the middle of the Korean War, DSM-I included a diagnosis of "gross stress reaction," and DSM-II described a "transient situational disturbance."

Establishing the diagnosis At the time DSM-III was compiled, professionals had begun to emphasize more lasting effects of trauma. We were in the aftermath of the Vietnam War, and some critics of the diagnosis of PTSD have suggested that it served a political purpose, in effect making the case that war is dangerous to mental health. The creators of DSM-III certainly sympathized with the veterans of a war many regarded as unjustified, and they looked for a pattern in the resulting suffering. At the same time, the women's movement was drawing new attention to the effects of sexual and physical abuse on women and children. All of this history influenced the psychiatric understanding of PTSD. As the disorder is defined today, it involves three kinds of symptoms:

1. ***Hyperarousal.*** Individuals with PTSD are irritable, easily startled, and constantly on guard. They sleep poorly and have difficulty concentrating.

2. ***Re-experiencing or intrusion.*** They recall the traumatic event involuntarily in the form of vivid memories, nightmares, and flashbacks. They may feel or even act as though it is happening again. Any object, situation, or feeling that reminds them of the trauma may cause intense distress.

3. ***Avoidance and emotional numbing.*** They avoid feelings, thoughts, persons, places, and situations that evoke memories of the trauma. They lose interest in their usual activities. They feel estranged from other people and even from their own feelings. These three sets of symptoms have a common theme—fixation on the trauma. The traumatic event dominates and controls the lives of people with PTSD. They have not assimilated the experience, so they repeatedly re-experience it in its original terrifying form. They are both emotionally numb and constantly on guard against a danger that no longer exists because they feel desperately conflicting needs for vigilance and repose.

What is a trauma? In DSM-III, a trauma was defined as an event beyond the range of ordinary human experience, one that would be distressing for almost anyone. Since then the definition has changed. In

the present edition of the diagnostic manual, DSM-IVTR, a "text revision" of the DSM-IV published in 2000, a traumatic experience is defined as one that involves a threat (or reality) of death, serious injury, or damage to physical integrity, and inspires intense fear, helplessness, or horror. The victim may experience the event directly, witness it, or be confronted with it in some other way. Some have interpreted these changes as shifting the focus away from the traumatic event itself and toward individual responses. The event is no longer necessarily utterly out of the ordinary or one that would be distressing to almost everyone. What arouses intense fear, helplessness, and horror in one person may have little effect on another. And in the DSM-IV-TR description, even immediate experience of the trauma is no longer necessary; being confronted with it could be interpreted to include hearing about it. There is no longer such an intimate relationship between a definite set of symptoms and a distinct kind of experience, so the theme of fixation on the trauma that links the symptoms is no longer so clear.

By the DSM-IV-TR definition, many kinds of events can be described as traumatic and many people can be said to have undergone a traumatic experience. The National Institute of Mental Health's Epidemiologic Catchment Area study found that more than 60% of men and more than 50% of women in the United States have had such an experience. The vast majority of people who have had a traumatic experience do not develop PTSD—nearly 90% of women and more than 97% of men in one large German study. In another recent study, researchers at Duke University interviewed hundreds of children and their parents at yearly intervals from ages 9 through 16, asking about traumatic events and symptoms of PTSD. More than two-thirds of the children had experienced at least one traumatic event, and a third had experienced more than one. The most common was witnessing or learning about a trauma suffered by another person. Only 13% re ported any symptoms typical of PTSD, and fewer than 1 in 200 had PTSD itself.

Just as trauma only occasionally causes PTSD symptoms, the symptoms associated with this diagnosis are not always the result of trauma. Some research suggests that people who experience "normal" stresses like illness, divorce, bereavement, or job loss develop such symptoms at the same rate as those who undergo traumatic stress. In a questionnaire survey of 600 undergraduates at Temple University, about 70% reported having had an experience they regarded as traumatic. About half of these events—for example, a romantic breakup or the anticipated death of a relative—were not traumatic by DSM-IV-TR or most other standards. But students who had had these apparently milder experiences reported just as much distress as those who suffered a catastrophic trauma.

Effects of gender It has become clear that people who develop PTSD differ from those who don't in a number of ways unrelated to the nature of the traumatic experience itself. To begin with, women seem to be two to three times as susceptible as men. They may be more biologically vulnerable for genetic or hormonal reasons. They also tend to undergo different kinds of trauma. Men suffer more non-sexual physical violence, women more rape and childhood sexual abuse. It is possible that female trauma is more often prolonged - the battered wife versus the street fighter, for example—and long-term stress can have more profound effects than single events. But even when both sexes have the same experience, women are more likely to develop PTSD. Six months after the bombing of the federal building in Oklahoma City, 45% of women exposed to the bombing had the disorder, and only 23% of the men did.

Differences in social support may be a factor; for example, wives may be better at soothing husbands than the other way around. Maybe women are more willing to admit that they have the symptoms and seek help, instead of retreating into solitary misery or disguising their problems with drinking and aggression. In a recent survey of 10,000 Australians, women reported the following traumatic symptoms more often than men did: avoiding thoughts and feelings related to a trauma, disturbed sleep, and intense startle reactions. Men reported one symptom more often than women did — emotional and social withdrawal.

Other individual differences Many other individual differences influence vulnerability. PTSD is more likely to arise in someone who has suffered previous traumatic experiences. Intentional injury—physical or sexual assault—creates a greater risk of PTSD than a natural disaster or an accident. The risk is even higher for victims who feel guilty because they believe that they bear some responsibility for the event.

High IQ may blunt the impact of a traumatic experience on mental health, and low IQ may exacerbate it. Depression, anxiety, alcohol and drug abuse, childhood behavior disorders and adolescent delinquency, antisocial personality, and other personality disorders also heighten vulnerability to PTSD.

German researchers interviewed a group of firefighters — for whom PTSD is an occupational hazard— immediately after their basic training and again periodically for two years. They found that men who showed more hostility and less confidence in their own abilities were more likely to develop PTSD symptoms. Twin and adoption studies suggest that heredity is a factor. In one study of identical twins, only one of each pair was a Vietnam combat veteran. About half of the veterans had been diagnosed with PTSD. Tests revealed subtle deficiencies in cognitive functioning that distinguished them from veterans who did not develop PTSD. But their identical twins who had not been in combat had the same deficiencies—which suggests that these were risk factors for PTSD rather than consequences of the traumatic experience.

Surprisingly, there is even some evidence that what happens to a person after the traumatic event influences the chance of developing PTSD as much as or more than what happens before. And we tend to revise our description of experiences in the light of later symptoms. In a study of veterans of the first Iraq war, 70% recalled a traumatic experience two years after returning but not after a month. Most veterans remembered more such experiences as time passed, especially the kind that did not involve a direct threat of death or physical injury to themselves.

Because memory is malleable and events before and after the trauma have so much influence on it, there is a risk that symptoms with other causes will be mistakenly attributed to a traumatic event. Many disability claims for PTSD have been made recently by Vietnamera veterans whose service ended 30 years ago. Critics point out that there are many reasons why people might want to make sense of their problems by ascribing them to a long-past experience. These critics fear that researchers and practitioners are not being careful enough to distinguish possible "pseudo-PTSD" from the real thing.

A distinctive diagnosis? The symptoms of PTSD overlap with the symptoms of other psychiatric disorders, especially depression and anxiety. In a Duke University study of children and adolescents, for example, being exposed to trauma did not result in PTSD symptoms but nearly doubled the rate of other psychiatric disorders. Researchers at Harvard studying men and women who volunteered for a study of depression found that by DSM-IV-TR standards, nearly 80% had undergone a traumatic experience and many also formally fit the diagnosis of PTSD. Australian psychologists tried to disentangle PTSD and depression among more than 350 people with serious injuries resulting from traffic accidents. Three months after the accident, 4% to 12% were diagnosed with PTSD, and another 16% to 30% were diagnosed with both PTSD and depression. In half of these patients, the diagnosis shifted from PTSD to depression or the other way around in the course of a year. The researchers suggest that PTSD symptoms are difficult to single out in reactions to traumatic stress.

Changes in store As a result of the many questions raised by research, experts are reconsidering how to describe traumatic stress, PTSD symptoms, and the relationship between them. The fifth edition of the American Psychiatric Association's diagnostic manual may put less emphasis on the diagnosis of PTSD and more on a range of responses that depend on much besides the traumatic event alone. In the future, research may concentrate more on individual vulnerability and the lives of patients before and after the experience. With more long-term studies beginning immediately after an event, relying too much on memory may no longer be necessary. For now, it is important to remember that not all traumas are alike, that any trauma will affect different people differently, and that PTSD should not necessarily be the default diagnosis when symptoms appear after any particular traumatic experience. But however present controversies are resolved, the knowledge consolidated in the last century will not be lost—that traumatic events are a threat to mental health, that the effects can be lasting, and that sufferers often need and deserve help.

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In summary, we have found that young adults with a history of PTSD but no prior drug dependence experienced substantially higher 12-month incidence of drug abuse and/or drug dependence compared with young adults who were not exposed to trauma. Emerging dependence problems were also more likely among young adults with a history of PTSD. The observed RRs were attenuated after simultaneous statistical adjustment for early antecedents common to PTSD and drug use disorders (childhood conduct problems, risk taking, and family SES) as well as sex, age, ethnicity, and years of education at the time of the first young adult assessment. However, the fully adjusted RRs for all of the outcomes remained substantial when comparing the PTSD group with the group with no trauma exposure (eg, adjusted RR for drug abuse or dependence, 4.9). This effect size is similar in magnitude to the effect size reported by Chilcoat and Breslau[8] using a 10-year follow-up period.

Several study limitations merit mention. First, our sample was predominantly African American (> 70%) from an urban location. Whether samples from other places will produce similar associations is a question for future studies. Second, between the first young adult assessment and the 12-month follow-up assessment, 988 of 1436 eligible participants were reassessed before study funds were exhausted. As in other longitudinal studies, there is a chance that participants who were successfully contacted and assessed at follow-up differed from participants who were not included in the follow-up with respect to variables associated with key independent variables and the outcomes. We found that PTSD and exposure to trauma were not significantly associated with follow-up participation. Additionally, we considered whether ineligibility for inclusion (eg, current or lifetime drug use problems) at the time of the first young adult assessment was associated with follow-up participation. We regressed the count of problems of drug abuse or dependence at the first young adult assessment on a binary covariate indicating participation in the follow-up assessment (using negative binomial regression). For this analysis, the null hypothesis was that there was no association between problems of drug abuse or dependence at the first young adult assessment and participation at follow-up. The null hypothesis was not rejected.

As in other community studies, the cumulative incidence of PTSD in this sample up to the age at assessment (in contrast with exposure to trauma) was low. Further, the number of cases of drug use disorders was constrained by the short interval when new cases were identified. Despite these limitations on statistical power, we found substantial and, with the exception of drug dependence, moderately precise estimates of RR.

Finally, while our assessment of trauma and PTSD was a lifetime assessment and our assessment of early antecedents was made at approximately age 6 years, there remains some chance that for some subjects, trauma may have occurred prior to age 6 years and remained undetected by our PTSD assessment interview.

The study has several strengths. The prospective study design mitigates potential recall error. We have used a validated, structured interview protocol to assess exposure to *DSM-IV*–qualifying traumatic events and PTSD. While this procedure requires recall of past events, the young age of participants limits recall distortion because the risk for exposure to trauma primarily starts in midadolescence (as shown by Breslau et al[36, 43]). Inclusion of measures of potential confounders measured in early childhood is an important strength. The short follow-up period of 12 months is also important because this constrains the possible influence of unmeasured confounders that might have occurred during the follow-up period but before the onset of the outcomes of interest. Additionally, recall of drug problems is likely to be more accurate than is the case when respondents are asked to review their memory for events that have occurred during long periods.

Other investigators[57-62] have found that individuals with emerging drug use problems (1 or 2 clinical features of dependence) may constitute a group distinct from both cases of drug dependence and individuals with no emerging problems of dependence. Hasin and Paykin[57, 62] have reported that in follow-up assessment, some members of the group with emerging

Incidence of Drug Problems in Young Adults Exposed to Trauma and Posttraumatic Stress Disorder
Do Early Life Experiences and Predispositions Matter?
Philip L. Reed, PhD; James C. Anthony, PhD; Naomi Breslau, PhD
Arch Gen Psychiatry. 2007;64(12):1435-1442.
Conclusions Association of PTSD with subsequent incident drug use disorders remained substantial after statistical adjustment for early life experiences and predispositions reported in previous studies as carrying elevated risk for both disorders. Posttraumatic stress disorder might be a causal determinant of drug use disorders, possibly representing complications such as attempts to self-medicate troubling trauma-associated memories, nightmares, or painful hyperarousal symptoms.

problems progressed into having *DSM-IV* dependence, whereas others moved back into the group with no clinical features of dependence. Our finding of an elevated risk for emerging problems among the PTSD group (adjusted RR = 3.3) leads us to speculate that PTSD might be 1 factor accounting for these differences in the progression from problems to diagnosable dependence. This suggests the possibility that early intervention to halt the progression to drug dependence for individuals with isolated problems of drug use might focus on trauma victims with PTSD.

In conclusion, this prospective study of young adults free of drug use problems found that trauma victims with PTSD were at markedly increased risk for incident drug use disorders in a 1-year follow-up period and that trauma victims who did not develop PTSD were not at increased risk for incidence of drug use problems. The association of PTSD with incident drug use disorders remained substantial even with statistical adjustment for early life experiences and predispositions that have been reported previously as carrying elevated risk for drug use disorders, exposure to trauma, and PTSD. Early cognitive achievement, conduct problems, family SES, and the predisposition for risk taking are important potential confounders and potential independent causal factors for incidence of drug use disorders in adulthood. To our knowledge, these common antecedents have not been taken into account in previous studies of the association of PTSD with subsequent first-onset drug use disorders.

The findings described here support the notions that the observed PTSD–drug use disorder associations might at least in part be causal and that the association is not fully accounted for by early experiences. An alternative to the early-experience explanation with growing empirical support is a self-medication explanation.[8, 27-28,63-65] A reliance on psychoactive drugs to relieve symptoms of PTSD might hinder the development of other coping strategies and, at the same time, lead to a more perilous drug-related trajectory.

• Violent behaviour and post-traumatic stress disorder. Begic, Drazen; Jokic-Begic, Natasa; Current Opinion in Psychiatry, Vol 15(6), Nov 2002. pp. 623-626. [Journal Article] Abstract: Purpose of review: There is a dual connection between violent behaviour and posttraumatic ***stress disorder***. On one hand, exposure to violence leads to ***post-traumatic stress disorder*** symptoms, and on the other hand some of the symptoms of ***post-traumatic stress disorder*** are violent behaviour and ***aggression***. In other words, violence creates ***post-traumatic stress disorder***, and posttraumatic ***stress disorder*** contains violence. The frequency of violent behaviour as a background for the development of posttraumatic ***stress disorder*** is increasing. Exposure to violence, criminal and terrorist attacks, sexual (especially in early childhood) and physical abuse lead to anxiety, ***aggression***, depression and ***post-traumatic stress disorder*** symptoms. Recent findings: Different types of violence and their psychological and psychiatric consequences are now being researched to determine whether there are any differences in exposure to violence and its consequences with regard to age, sex, ethnicity, and sociodemographic characteristics. Apart from the individual and his/her family, the effects of violence on the wider community are being examined. In contrast, the well-described ***post-traumatic stress disorder***, within the frame of increased alertness, can contain violence, ***aggression***, anger, and impulsivity, which are a big problem for the patient and his/her family. These are the most common reasons for requesting psychiatric treatment. Summary: Future research into violent behaviour and ***post-traumatic stress disorder*** will reveal the risk factors for this ***disorder***, and try to explain what it is that, after exposing an individual to psychological trauma, leads to ***post-traumatic stress disorder***. Possible protective factors and mechanisms to prevent the occurrence of ***post-traumatic stress disorder*** will be described. (PsycINFO Database Record (c) 2007 APA, all rights reserved)

Aggressive behavior in combat veterans with post-traumatic stress disorder. Begic, Drazen; Jokic-Begic, Nataša; Military Medicine, Vol 166(8), Aug 2001. pp. 671-676. [Journal Article] Abstract: The incidence of ***aggression*** and violent behavior in combat veterans varies and can be observed with regard to the presence or absence of ***post-traumatic stress disorder*** (PTSD). In this study the authors examined violent behavior in 116 male combat veterans (aged 22-45 yrs), 79 of whom had been diagnosed with PTSD. Results show that a significantly greater occurrence of ***aggression*** was observed in combat veterans with PTSD compared with those without PTSD. There were various types of aggressive behavior that frequently are combined. Autoaggressive (suicidal) and heteroaggressive (interpersonal violence) behaviors predominate, with dominating verbal ***aggression*** and impulsive somatic reactions. Impulsive reactions are more frequently directed toward unknown persons, whereas verbal ***aggression*** is mostly aimed at known people. In the occurrence of aggressive behavior in combat veterans with PTSD, important roles are played by education level, low socioeconomic status, maltreatment in childhood, and previous types of violent behavior (before participation in war events). (PsycINFO Database Record (c) 2007 APA, all rights reserved)

Dangerous misidentification of people due to flashback phenomena in posttraumatic stress disorder. Silva, J. Arturo; Leong, Gregory B.; Harry, Bruce E.; Journal of Forensic Sciences, Vol 43(6), Nov 1998. pp. 1107-1111. [Journal Article] Abstract: Misidentification of people may occur in the context of visual flashback phenomena associated with ***post-traumatic stress disorder***. People who misidentify someone during a flashback associated with previous war combat experience may perceive and conceptualize the misidentified object as an enemy who may be both feared and disliked. This might make the misidentified objects become the targets of violent attacks by the affected person. In this article, the authors present 5 cases of flashback-induced misidentification of people who were subsequently attacked within the context of the flashback experience. Ss were males (aged 44-53 yrs) who were all involved in combat during

the Vietnam War. Ss were treated with various psychotherapeutic methods and drug therapies with varying results. The nature of the misidentification of persons due to flashback experiences is discussed, as is the association between the type of misidentification and ***aggression***.