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RISK FACTORS FOR PTSD

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PTSD was originally conceptualized as a direct consequence of exposure to a traumatic event in otherwise normal individuals. As originally described, the emphasis was on establishing the primacy of the trauma as the etiologic agent, rather than individual vulnerability factors. Yet it was clear from the beginning that not all trauma survivors developed permanent disorder. In fact, many recovered. Thus, the search for risk factors that increase vulnerability to chronic PTSD occurred early in the history of the disorder. Foy et al. (1984) published one of the first formal studies to look at risk factors for PTSD and reported characteristics of trauma exposure to be of central importance. Numerous studies have since observed a dose-response relationship between trauma severity and PTSD.

However, features of the trauma invariably account for a small proportion of the variance in PTSD symptoms (Yehuda & McFarlane, 1995). Epidemiological research has found the rate of exposure to trauma to far outweigh the prevalence of PTSD, indicating that most people do not ever develop PTSD following a traumatic event (Breslau et al., 1998). Moreover, PTSD is not the only possible psychological consequence of trauma. Elevated rates of major depression, panic, and substance abuse are commonly observed in traumatized populations (Shalev et al., 1998b), again calling into question the nature of the relationship between trauma and PTSD.

Currently, some investigators are calling for PTSD to be recast in a stress-diathesis model, with trauma characteristics and individual risk factors interacting to determining who develops the disorder. The study of risk factors has become increasingly popular, emphasizing environmental and demographic factors, personality and psychiatric history, dissociation, cognitive and biological systems, and genetic or familial risk (Yehuda, 1999a). All of these approaches are essential in directing and advancing the field as a whole. To date, several important risk factors have been identified.

Environmental Risk Factors. In addition to characteristics of stressor severity, a history of exposure *prior* to the focal trauma is also an important risk factor. A history of prior exposure to trauma or to chronic stress is an extremely potent risk factor for PTSD (Davidson et al., 1991), particularly if it is experienced at a young age (Bremner et al., 1993). In addition, Breslau et al. (1999a) found that the *type* of prior trauma exposure is important, prior assault being a particularly potent risk factor for the development of PTSD upon subsequent traumatization. Social factors may also affect risk. A history of family instability is associated with increased prevalence of PTSD (King et al., 1996), whereas good social support is associated with lower levels of symptoms (Solomon et al., 1988).

Demographic Risk Factors. Breslau et al. (1998) identified several demographic risk factors for the development of PTSD. Gender is an extremely salient risk factor, even controlling for differences in the type of events that are experienced by men compared to women. A consistent finding has been that the prevalence of PTSD is almost twice as high in women as it is in men. To date, there are no firm explanations for this finding, although gender (being female) is also a risk factor for other psychiatric disorders. Breslau et al. (1999b) found that the higher risk for PTSD in females is primarily due to a particular vulnerability to assaultive violence. Breslau et al. (1999b) suggest that assaultive violence is more threatening and injurous to females, most perpetrators being male and therefore wielding greater physical strength.

Lower levels of education and income, and being divorced or widowed are risk factors PTSD. In addition, some studies have reported a higher risk for PTSD amongst ethnic minorities (Breslau et al., 1998). However, not all studies have found ethnicity to be a risk factor for PTSD and instead have found that ethnic differences may interact with (Norris, 1992) or be attributable to (Breslau et al., 1991) other factors. Breslau et al. (1998) note that several demographic factors affect the risk of trauma exposure, including gender, age, and socioeconomic status, as well as ethnicity. This observation is important to the consideration of risk since trauma exposure does not occur in a vacuum. Some of the predictors

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of PTSD may actually be predictors of trauma exposure.

Prior Psychiatric Disorders and Personality Dimensions. A past history of behavioral or psychological problems has also been associated with the development of PTSD (McFarlane, 1989). Many different psychiatric disorders have been associated with past psychological problems, suggesting that this risk factor may be non-specific to poor coping rather than predictive of a specific disorder. Indeed, Breslau et al. (1998) found that prior affective, anxiety or substance abuse disorders all represented risk factors for the development of PTSD, and concluded that having a psychiatric history per se was a stronger predictor of PTSD than having a history of any one of the *specific* disorders. Personality dimensions are also important (for a review, see Schnurr & Vielhauer, 1999). Adult avoidant, antisocial (Schnurr et al., 1993), or neurotic personalities (Breslau et al., 1998) prior to the traumatic event have an increased risk for the development of PTSD.

Dissociation. There is ambiguity regarding whether dissociation should be considered a stable personality trait or a state-related cognitive response to trauma. Nonetheless, peritraumatic dissociation appears to be an important risk factor for the development of PTSD (Koopman et al., 1994), and PTSD subjects show elevated scores on measures of dissociative symptoms (Bremner et al., 1992). In a prospective study of injured trauma survivors, Shalev et al. (1996) found peritraumatic dissociation to be the best predictor of PTSD symptoms at 6 months post-trauma, explaining 30% of the variance in symptoms. Dissociative reactions may be adopted as a maladaptive coping strategy in response to childhood trauma or chronic stress (Spiegel, 1991). As such, they may partially mediate the relationship between prior traumatization and subsequently increased vulnerability to PTSD. Future studies examining dissociation in high risk groups will shed light on this issue.

Cognitive Risk Factors. Lower intellectual functioning has been found to be a risk factor for the development of PTSD. Macklin et al. (1998) assessed IQ in soldiers prior to entering the combat situation and found that lower precombat intelligence levels are associated with increased risk of developing PTSD on exposure to combat. This association remained significant even once an adjustment was made for degree of combat exposure. Controlling for degree of combat exposure is important because individuals with lower premorbid IQs are often placed in heavier combat situations than others.

Individuals with PTSD show increased neurological soft signs, indicative of subtle nervous system dysfunction. Furthermore, they also report a larger number of developmental problems, suggesting that there are *preexisting* impairments in neurodevelopment which act as risk factors for the development of PTSD (Gurvits et al., 2000).

PTSD is also associated with specific impairments in explicit memory. Explicit memory deficits have been observed in combat veterans with PTSD compared to noncombat controls (Yehuda et al., 1995), and in rape victims with PTSD compared to rape victims without PTSD (Jenkins et al., 1998). Thus far memory function has not been assessed prior to trauma exposure. However, the possibility that (like IQ) lower mnemonic ability predates the trauma should be considered, particularly in the light of emergent evidence for premorbid abnomalities in HPA axis function (discussed below), this neuroendocrine system having well documented effects on explicit memory function. Again, examining memory in high risk groups will be informative.

Biological Risk Factors. The study of biological aspects of PTSD has identified several abnormalities that are present in trauma survivors with PTSD. In so far as these alterations are not observed in similarly exposed persons without PTSD, they are likely to be related to the pathophysiology of PTSD, not to trauma exposure per se. Any one of these abnormalities could, in theory, represent a pre-existing vulnerability to the effects of trauma. Indeed, recent evidence has suggested that at least some of the observed biological abnormalities represent risk factors for the development of PTSD (for a review see Yehuda, 1999a).

Shalev et al. (1998a) assessed heart rate in 86 trauma survivors at the time of presentation to the ER. Subjects who later developed PTSD had a higher heart rate at time of presentation compared to those who did not develop PTSD, consistent with there being an enhanced or prolonged catecholamine response to the trauma in this group. Heart rate no longer distinguished the PTSD and no PTSD groups at one month follow-up. Pitman (1989) has proposed that elevated norepinephrine during trauma may result in overconsolidation of memory and the subsequent development of intrusive symptoms.

Neurohormonal research has also uncovered a potential risk factor for the development of PTSD. Both combat-related and civilian PTSD are associated with chronically low levels of cortisol, a glucocorticoid secreted by the hypothalamic-pituitary-adrenal (HPA) axis. Underlying this observation are systematic alterations in overall HPA axis activity, which appear to be unique to PTSD. Yehuda et al. (1996) present compelling evidence that such alterations are symptomatic of enhanced negative feedback occurring in the HPA axis, mediated by increased glucocorticoid receptor sensitivity, and acting to keep cortisol levels chronically low.

Direct support for the idea that HPA axis abnormalities represent a risk factor for PTSD was provided by a study of cortisol levels in the immediate aftermath (i.e., within several hours) of rape (Resnick et al., 1995). Lower cortisol levels were observed in women with prior exposure to rape or assault, and prior exposure was a risk factor for the development of PTSD. This suggests that HPA axis alterations may underlie the association between prior exposure and increased vulnerability to PTSD. Furthermore, a second study found that individuals who developed PTSD following a motor vehicle accident showed a lower cortisol response to the accident compared to those who did not develop PTSD (Yehuda et al., 1998a).

Yehuda et al. (2000) report preliminary explorations of biologic alterations in a group known to be at high risk of

developing PTSD following trauma – adult children of Holocaust survivors (Yehuda et al., 1998a). Yehuda et al. (2000) found that these high risk offspring had lower cortisol levels relative to non-psychiatric children of non-trauma exposed parents, comparable to those observed in other groups of trauma survivors with PTSD. Low cortisol levels were particularly evident in those offspring who had been exposed to trauma, but those who have not experienced a life-threatening traumatic event also show cortisol levels that are somewhat reduced. Observations of biologic alterations in offspring of Holocaust survivors could be subject to different interpretations and do not directly address environmental or genetic factors.

Familial or "Genetic" Risk Factors? Several lines of evidence demonstrate familial transmission of PTSD. True et al. (1993) examined the prevalence of PTSD in monozygotic versus dyzygotic twin pairs and demonstrated that as much as 30% of some PTSD symptoms appear to have a genetic basis. Davidson et al. (1985) found that trauma survivors with PTSD were more likely to have parents and first-degree relatives with mood, anxiety, and substance abuse compared with trauma survivors who did not develop PTSD. More recently, Yehuda et al. (1998b) demonstrated that Holocaust survivors with PTSD are more likely to have children with PTSD compared to Holocaust survivors without PTSD.

Since all of the aforementioned studies confound genetic and environmental concordance, the extent which the findings are indicative of truly genetic phenomena is not yet clear. However, even in the absence of genetic heritability of risk factors for PTSD, family studies may ultimately demonstrate intergenerational effects of stress and trauma as the most potent of risk factors due to the persistent cognitive and neurobiologic changes that they induce.

SELECTED ABSTRACTS

BRESLAU, N., KESSLER, R.C., CHILCOAT, H.D., SCHULTZ, L.R., DAVIS, G.C., & ANDRESKI, P. (1998). **Trauma and post-traumatic stress disorder in the community: The 1996 Detroit Area Survey of Trauma.** *American Journal of Psychiatry, 55,* 626-632. *Background:* The study estimates the relative importance of specific types of traumas experienced in the community in terms of their prevalence and risk of leading to PTSD. *Methods:* A representative sample of 2,181 persons in the Detroit area aged 18 to 45 years were interviewed by telephone to assess the lifetime history of traumatic events and PTSD, according to DSM-IV. PTSD was assessed with respect to a randomly selected trauma from the list of traumas reported by each respondent, using a modified version of the Diagnostic Interview Schedule, Version IV, and the World Health Organization Composite International Diagnostic Interview. *Results:* The conditional risk of PTSD following exposure to trauma was 9.2%. The highest risk of PTSD was associated with assaultive violence (20.9%). The trauma most often reported as the precipitating event among persons with PTSD (31 % of all PTSD cases) was sudden unexpected death of a loved one, an event experienced by 60% of the sample, and with a moderate risk of PTSD (14.3%). Women were at higher risk of PTSD than men, controlling for type of trauma. *Conclusions:* The risk of PTSD associated with a representative sample of traumas is less than previously estimated. Previous studies have overestimated the conditional risk of PTSD by focusing on the worst events the respondents had ever experienced. Although recent research has focused on combat, rape, and other assaultive violence as causes of PTSD, sudden unexpected death of a loved one is a far more important cause of PTSD in the community, accounting for nearly one third of PTSD cases.

BRESLAU, N., CHILCOAT, H.D., KESSLER, R.C., & DAVIS, G.C. (1999a). **Previous exposure to trauma and PTSD effects of subsequent trauma: Results from the Detroit Area Survey of Trauma.** *American Journal of Psychiatry, 156,* 902-907. *Objective:* With the exception of a few reports of higher rates of childhood trauma in Vietnam veterans with PTSD, little is known about the influence of previous exposure to trauma on the PTSD effects of subsequent trauma. The authors examine interrelated questions about the effects of previous exposure to trauma. *Method:* A representative sample of 2,181 individuals in southeast Michigan were interviewed by telephone to record lifetime history of traumatic events specified in DSM-IV as potentially leading to PTSD. PTSD was assessed with respect to randomly selected index trauma from the list of events reported by each respondent. *Results:* History of any previous exposure to traumatic events was associated with a greater risk of PTSD from the index trauma. Multiple previous events had a stronger effect than a single previous event. The effect of previous assaultive violence persisted over time with little change. When they examined several features of the previous exposure to trauma, the authors found that subjects who experienced multiple events involving assaultive violence in childhood were more likely to experience PTSD from trauma in adulthood. Furthermore, previous events involving assaultive violence — single or multiple, in childhood or later on — were associated with a higher risk of PTSD in adulthood. *Conclusions:* Previous exposure to trauma signals a greater risk of PTSD from subsequent trauma. Although these results are consistent with a sensitization hypothesis, like the results from previous research on PTSD, they do not address the mechanism of increased responsivity to trauma. Long-term observational studies can further elucidate these observations.

BRESLAU, N., CHILCOAT, H.D., KESSLER, R.C., PETERSON, E.L., & LUCIA, V.C. (1999b). **Vulnerability to assaultive violence: Further specification of the sex difference in post-traumatic stress disorder.** *Psychological Medicine, 29,* 813-821. *Background:* We examine potential sources of the sex differences in PTSD in the community. *Method:* Data were obtained from a representative sample of 2181 persons aged 18-45 years in the Detroit primary metropolitan statistical area, which is a six-county area containing more than four million residents. A random digit dialling method was used to select the sample and a computer-assisted telephone interview was used to obtain the data. DSM-IV PTSD was assessed with respect to a randomly selected trauma from the list of qualifying traumas reported by each respondent. *Results:* The lifetime prevalence of exposure and the mean number of traumas were lower in females than males. The overall conditional risk of PTSD (i.e. the probability of PTSD among those exposed to a trauma) was approximately twofold higher in females than males, adjusting for the sex difference in the distribution of trauma types. The sex difference was due primarily to females' greater risk following assaultive violence. The sex difference in the avoidance and numbing symptom group following assaultive violence exceeded the sex differences in other symptom groups. *Conclusions:* Future research should focus on sex differences in the response to assault-

ive violence, including potential explanations for females' greater probability to experience avoidance and numbing.

DAVIDSON, J., SWARTZ, M., STORCK, M., KRISHNAN, R.R., & HAMMETT, E. (1985). **A diagnostic and family study of posttraumatic stress disorder.** *American Journal of Psychiatry, 142*, 90-93. A family history study of 36 patients with chronic PTSD revealed a positive history of familial psychopathy in 66% of the patients. Alcoholism, depression, and anxiety disorders were the disorders most commonly found. The patients also had a higher prevalence of alcoholic siblings than did a retrospectively derived control group of depressed and anxious male patients. With respect to the proportion of familial anxiety to familial depression, the probands with PTSD more closely resembled probands with generalized anxiety than probands with depression. Every patient had experienced at least one significant psychiatric illness during his lifetime, most commonly alcohol abuse or depression.

FOY, D.W., SIPPRELLE, R.C., RUEGER, D.B., & CARROLL, E.M. (1984). **Etiology of posttraumatic stress disorder in Vietnam veterans: Analysis of premilitary, military, and combat exposure influences.** *Journal of Consulting and Clinical Psychology, 52*, 79-87. 43 Vietnam veterans seeking psychological services at a Los Angeles Veterans Administration medical center were assigned to positive and negative groups of (PTSD based on the DSM-III). Subjects were extensively assessed to examine the relative contributions of premilitary adjustment, military adjustment, and extent of combat exposure to the development of combat-related, chronic PTSD. In addition, groups were compared on profiles from the MMPI and a psychological problem checklist. Results of multiple regression analyses demonstrated that combat exposure and, to a lesser degree, military adjustment were significantly related to PTSD symptomatology, whereas premilitary adjustment was not. Discriminant function analyses showed that the MMPI had moderate ability to correctly classify subjects on the basis of PTSD diagnosis. However, problem checklist items indicative of anxiety-based disorders, particularly generalized anxiety and pervasive disgust, formed a discriminant function that correctly classified more than 90 percent of study subjects. Results were discussed in terms of implications for an empirically derived conceptualization of PTSD and further research directions.

GURVITS, T.G., GILBERSTON, M.W., LASKO, N.B., TARHAN, A.S., SIMEON, D., MACKLIN, M.L., ORR, S.P., & PITMAN, R.K. (2000). **Neurologic soft signs in chronic posttraumatic stress disorder.** *Archives of General Psychiatry, 57*, 181-186. *Background:* Subtle neurologic impairment has been reported in several mental disorders. The goals of the present study were to evaluate neurologic status in patients of both sexes with chronic PTSD from different traumatic experiences. *Methods:* 21 adult women who were sexually abused as children (12 with PTSD, 9 without) and 38 male Vietnam War combat veterans (23 with PTSD, 15 without) underwent examination for 41 neurologic soft signs, which were scored by the examiner as well as a blind rater observing videotapes. Subject history was obtained with special attention to neurodevelopmental problems. Psychometrics included the Wender Utah Rating Scale for symptoms of childhood attention-deficit / hyperactivity disorder and the Michigan Alcoholism Screening Test. Veterans also completed the Combat Exposure Scale and subtests of the Wechsler Adult Intelligence Scale-Revised. *Results:* Average neurologic soft sign scores (interrater reliability = 0.74) of women with PTSD owing to sexual abuse in childhood (mean [SD], 0.77 [0.32]) and veteran men (0.72 [0.20]) with combat-related PTSD were comparable and significantly ($P < .001$) higher than those of women sexually abused as children (0.42 [0.10]) and combat veteran men (0.43 [0.17]) without PTSD. This effect could not be explained by a history of alcoholism or head injury. Subjects with PTSD reported more neurodevelopmental problems and more childhood attention-deficit / hyperactivity disorder symptoms and had lower IQs, all of which were significantly correlated with neurologic soft signs. *Conclusion:* Neurologic compromise is evident from subject history and findings from physical examination in both women and men with chronic PTSD who had experienced different kinds of traumatic events in childhood and adulthood.

MACKLIN, M.L., METZGER, L.J., LITZ, B.T., MCNALLY, R.J., LASKO, N.B., ORR, S.P., & PITMAN, R.K. (1998). **Lower precombat intelligence is a risk factor for posttraumatic stress disorder.** *Journal of Consulting and Clinical Psychology, 66*, 323-326. The authors examined the relation between intelligence and PTSD by studying the association among precombat intelligence, current intelligence, and self-reported PTSD symptoms. Military aptitude test results were obtained in 59 PTSD and 31 non-PTSD Vietnam combat veterans who had undergone a psychodiagnostic interview and current intelligence testing. People with lower precombat intelligence were more likely to develop PTSD symptoms as assessed by the Clinician-Administered PTSD Scale even after adjustment for extent of combat exposure. The association between current intelligence and PTSD was no longer significant after adjusting for precombat intelligence. These results suggest that lower pretrauma intelligence increases risk for developing PTSD symptoms, not that PTSD lowers performance on intelligence tests.

MCFARLANE, A.C. (1989). **The aetiology of post-traumatic morbidity: Predisposing, precipitating and perpetuating factors.** *British Journal of Psychiatry, 154*, 221-228. A group of 469 firefighters were studied 4, 11 and 29 months after having an extreme exposure to a bushfire disaster. The relative importance of the impact of the disaster, personality and ways of coping were investigated as determinants of post-traumatic morbidity. Neuroticism and a past history of treatment for a psychological disorder were better predictors of post-traumatic morbidity than the degree of exposure to the disaster or the losses sustained. These results raise doubts about the postulated central aetiological role a traumatic event plays in the onset of morbidity.

RESNICK, H.S., YEHUDA, R., PITMAN, R.K., & FOY, D.W. (1995). **Effect of previous trauma on acute plasma cortisol level following rape.** *American Journal of Psychiatry, 152*, 1675-1677. *Objective:* The authors examined the relationships among history of previous assault, severity of rape, acute plasma cortisol level after rape, and development of rape-related PTSD. *Method:* Blood samples were drawn from 37 adult female rape victims within 51 hours after they had been raped. The subjects were assessed for history of previous assault and for the presence of PTSD 17-157 days (mean = 90 days) after the rape. *Results:* Women with a history of previous assault had a lower mean acute cortisol level after the rape but a higher probability of subsequently developing PTSD. A significant interaction between history of previous assault and the severity of the index rape was observed: only women who had never been assaulted before had higher cortisol levels following high-severity rapes (those which included injury or multiple types of penetration) than low-severity rapes. *Conclusions:* The authors conclude that previous traumatization may attenuate the acute cortisol response to trauma.

SCHNURR, P.P., FRIEDMAN, M.J., & ROSENBERG, S.D. (1993). **Premilitary MMPI scores as predictors of combat-related PTSD symptoms.** *American Journal of Psychiatry, 150,* 479-483. *Objective:* The authors used data collected before military service to assess predictors of combat-related lifetime symptoms of PTSD. *Method:* The subjects were 131 male Vietnam and Vietnam-era veterans who had taken the MMPI in college and who were interviewed as adults with the Structured Clinical Interview for DSM-III-R. Scores on the basic MMPI scales were used to predict combat exposure, lifetime history of any PTSD symptoms given exposure, and lifetime PTSD classification (symptoms only, subthreshold PTSD, or full PTSD). *Results:* Group means on the MMPI scales were within the normal range. No scale predicted combat exposure. Hypochondriasis, psychopathic deviate, masculinity-femininity, and paranoia scales predicted PTSD symptoms. Depression, hypomania, and social introversion predicted diagnostic classification among subjects with PTSD symptoms. The effects persisted when amount of combat exposure was controlled for. *Conclusions:* Premilitary personality can affect vulnerability to lifetime PTSD symptoms in men exposed to combat.

SHALEV, A.Y., PERI, T., CANETTI, L., & SCHREIBER, S. (1996). **Predictors of PTSD in injured trauma survivors: A prospective study.** *American Journal of Psychiatry, 153,* 219-225. *Objective:* The aim of this study was to prospectively examine the relationship between immediate and short-term responses to a trauma and the subsequent development of PTSD. *Method:* All patients consecutively admitted to a general hospital were screened for the presence of physical injury due to a traumatic event. 51 eligible subjects were assessed 1 week and 6 months after the trauma. The initial assessment included measures of event severity, peritraumatic dissociation, and symptoms of intrusion, avoidance, depression, and anxiety. The follow-up assessments added the PTSD module of the Structured Clinical Interview for DSM-III-R—Non-Patient Version and the civilian trauma version of the Mississippi Scale for Combat-Related PTSD. *Results:* 13 subjects (25.5%) met PTSD diagnostic criteria at follow-up. Subjects who developed PTSD had higher levels of peritraumatic dissociation and more severe depression, anxiety, and intrusive symptoms at the 1-week assessment. Peritraumatic dissociation predicted a diagnosis of PTSD after 6 months over and above the contribution of other variables and explained 29.4% of the variance of PTSD symptom intensity. Initial scores on the Impact of Event Scale predicted PTSD status with 92.3% sensitivity and 34.2% specificity. Symptoms of avoidance that were initially very mild intensified in the subjects who developed PTSD. *Conclusions:* Peritraumatic dissociation is strongly associated with the later development of PTSD. Early dissociation and PTSD symptoms can help the clinician identify subjects at higher risk for developing PTSD.

SHALEV, A.Y., SAHAR, T., FREEDMAN, S., PERI, T., GLICK, N., BRANDES, D., ORR, S.P., & PITMAN, R.K. (1998a). **A prospective study of heart rate response following trauma and the subsequent development of posttraumatic stress disorder.** *Archives of General Psychiatry, 55,* 553-559. *Background:* Physiological arousal during traumatic events may trigger the neurobiological processes that lead to PTSD. This study prospectively examined the relationship between heart rate and blood pressure recorded immediately following a traumatic event and the subsequent development of PTSD. *Method:* 86 trauma survivors who presented at the emergency department of a general hospital were followed up for 4 months. Heart rate and blood pressure were recorded on arrival at the emergency department. Heart rate, anxiety, depression, and PTSD symptoms were assessed 1 week, 1 month, and 4 months later. The Clinician-Administered PTSD Scale defined PTSD status at 4 months. *Results:* 20 subjects (23%) met PTSD diagnostic criteria at the 4-month assessment (PTSD group), and 66 (77%) did not (non-PTSD group). Subjects who developed PTSD had higher heart rates at the emergency department (95.5 ± 13.9 vs 83.3 ± 10.9 beats per minute, $t = 4.4$, $p < .001$) and 1 week later (77.8 ± 11.9 vs 72.0 ± 9.5 beats per minute, $t = 2.25$, $p < .03$), but not after 1 and 4 months. The groups did not differ in initial blood pressure measurement. Repeated-measures analysis of variance (ANOVA) for heart rate showed a significant group effect ($p < .02$), time effect ($p < .001$), and group X time interaction ($p < .001$). The time effect and group X time interaction remained significant when adjusted for sex, age, trauma severity, immediate response, and dissociation during the traumatic event. *Conclusion:* Elevated heart rate shortly after trauma is associated with the later development of PTSD.

TRUE, W.R., RISE, J., EISEN, S.A., HEATH, A.C., GOLDBERG, J., LYONS, M.J., & NOWAK, J. (1993). **A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms.** *Archives of General Psychiatry, 50,* 257-264. We studied 4042 Vietnam era veteran monozygotic and dizygotic male twin pairs to determine the effects of heredity, shared environment, and unique environment on the liability for 15 self-reported PTSD symptoms included in the symptom categories of reexperiencing the trauma, avoidance of stimuli related to the trauma, and increased arousal. Quantitative genetic analysis reveals that inheritance has a substantial influence on liability for all symptoms. Symptoms in the reexperiencing cluster and one symptom in the avoidance and numbing cluster are strongly associated with combat exposure, and monozygotic pairs are more highly concordant for combat exposure than dizygotic pairs. By fitting a bivariate genetic model, we show that there are significant genetic influences on symptom liability, even after adjusting for differences in combat exposure; genetic factors account for 13% to 30% of the variance in liability for symptoms in the reexperiencing cluster, 30% to 34% for symptoms in the avoidance cluster, and 28% to 32% for symptoms in the arousal cluster. There is no evidence that shared environment contributes to the development of PTSD symptoms.

YEHUDA, R., BIERER, L.M. SCHMEIDLER, J., AFERIAT, D.H., BRESLAU, I., & DOLAN, S. (2000). **Low cortisol and risk for PTSD in adult offspring of Holocaust survivors.** *American Journal of Psychiatry, 157. Objective:* The study examined the association between cortisol and putative risk factors for PTSD in a sample of subjects at increased risk for the development of PTSD. *Method:* 24-hour urinary cortisol excretion was measured in 35 adult offspring of Holocaust survivors and 15 healthy comparison subjects who were not offspring of Holocaust survivors. Subjects were also characterized with regard to clinical symptoms, presence or absence of psychiatric diagnoses including PTSD, and the presence or absence of PTSD in their parents. *Results:* Low cortisol levels were significantly associated with both PTSD in parents and lifetime PTSD in subjects, whereas having a current psychiatric diagnosis other than PTSD was relatively, but nonsignificantly, associated with higher cortisol levels. Offspring with both parental PTSD and lifetime PTSD had the lowest cortisol levels of all study groups. *Conclusions:* Parental PTSD, a putative risk factor for PTSD, appears to be associated with low cortisol levels in offspring, even in the absence of lifetime PTSD in the offspring. The findings suggest that low

cortisol levels in PTSD may constitute a vulnerability marker related to parental PTSD as well as a state-related characteristic associated with acute or chronic PTSD symptoms.

YEHUDA, R., MCFARLANE, A.C., & SHALEV, A.Y. (1998a). **Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event.** *Biological Psychiatry, 44,* 1305-1313. PTSD is a psychiatric condition that is directly precipitated by an event that threatens a person's life or physical integrity and that invokes a response of fear, helplessness, or horror. In recent years it has become clear that only a proportion of those exposed to fear-producing events develop or sustain PTSD. Thus, it seems that an important challenge is to elucidate aberrations in the normal fear response that might precipitate trauma-related psychiatric disorder. This paper summarizes the findings from recent studies that examined the acute and longer term biological response to traumatic stress in people appearing to the emergency room immediately following trauma exposure. In the aggregate, these studies have demonstrated increased heart rate and lower cortisol levels at the time of the traumatic event in those who have PTSD at a follow-up time compared to those who do not. In contrast, certain features associated with PTSD, such as intrusive symptoms and exaggerated startle responses, are only manifest weeks after the trauma. The findings suggest that the development of PTSD may be facilitated by an atypical biological response in the immediate aftermath of a traumatic event, which in turn leads to a maladaptive psychological state.

YEHUDA, R., SCHMEIDLER, J., GILLER, E.L., BINDER-BRYNES, K., & SIEVER, L.J. (1998b). **Relationship between PTSD characteristics of Holocaust survivors and their adult offspring.** *American Journal of Psychiatry, 155,* 841-843. *Objective:* There is controversy regarding the long-lasting effects of the Holocaust on the adult children of Holocaust survivors. In the present study the authors examined the relationship between characteristics of Holocaust survivors and their adult children to determine whether differences in symptoms severity or diagnostic status of parents would be associated with similar characteristics in their adult children. *Method:* Holocaust survivors (n = 22) and their offspring (n = 22) were interviewed with several instruments to assess lifetime trauma history, effect of trauma on one's life, level of intrusive PTSD, and current and lifetime axis I psychiatric disorder other than PTSD. *Results:* There were significant relationships between parents and children regarding the effect of trauma on one's life and level of intrusive, but not avoidance, symptoms in response to reminders of the Holocaust. Offspring with traumatic events were more likely to develop PTSD if their parents had PTSD. *Conclusions:* Symptoms in offspring may be related to presence and severity of symptoms in the parent. Furthermore, PTSD in the parent may be a risk factor for PTSD in offspring.

YEHUDA, R., SCHMEIDLER, J., WAINBERG, M., BINDER-BRYNES, K., & DUVDEVANI, T. (1998c). **Increased vulnerability to posttraumatic stress disorder in adult offspring of Holocaust survivors**. *American Journal of Psychiatry, 155,* 1163-1171. *Objective:* There has been considerable controversy regarding the impact of the Holocaust on the second generation, but few empirical data are available that systematically document trauma exposure and psychiatric disorder in these individuals. To obtain such data, the authors examined the prevalence of stress and exposure to trauma, current and lifetime PTSD, and other psychiatric diagnoses in a group of adult offspring of Holocaust survivors (n = 100) and a demographically similar comparison group (n = 44). *Method*: Subjects were recruited from both community and clinical populations and were evaluated with the use of structured clinical instruments. Stress and trauma history were evaluated with the Antonovsky Life Crises Scale and the Trauma History Questionnaire, PTSD was diagnosed with the Clinician Administered PTSD Scale, and other psychiatric disorders were evaluated according to the Structured Clinical Interview for DSM-IV. *Results:* The data show that although adult offspring of Holocaust survivors did not experience more traumatic events, they had a greater prevalence of current and lifetime PTSD and other psychiatric diagnoses than the demographically similar comparison subjects. This was true in both community and clinical subjects. *Conclusions:* The findings demonstrate an increased vulnerability to PTSD and other psychiatric disorders among offspring of Holocaust survivors, thus identifying adult offspring as a possible high-risk group within which to explore the individual differences that constitute risk factors for PTSD.

ADDITIONAL CITATIONS
Annotated by the Editor

BREMNER, J.D., SOUTHWICK, S.M., JOHNSON, D.R., YEHUDA, R., & CHARNEY, D.S. (1993). **Childhood physical abuse and combat-related posttraumatic stress disorder in Vietnam Veterans.** *American Journal of Psychiatry, 150,* 235-239.
Assessed premilitary stressful and traumatic events in male Vietnam veterans, 38 with PTSD and 28 without PTSD. Prevalence of childhood physical abuse was 26% in the PTSD group, compared with 7% in the no PTSD group. The PTSD group had a higher total number of premilitary traumatic events.

BREMNER, J.D., SOUTHWICK, S.M., BRETT, E., FONTANA, A., ROSENHECK, R., & CHARNEY, D.S. (1992). **Dissociation and posttraumatic stress disorder in Vietnam combat veterans.** *American Journal of Psychiatry, 149,* 328-332.
Examined dissociative symptoms in male Vietnam combat veterans. Compared to veterans without PTSD (n = 32), those with PTSD (n = 53) reported a higher level of current dissociation and dissociation at the time of combat trauma. These differences we not explained by amount of combat exposure.

DAVIDSON, J.R.T., HUGHES, D., BLAZER, D.G., & GEORGE, L.K. (1991). **Post-traumatic stress disorder in the community: An epidemiological study.** *Psychological Medicine, 21,* 713-721.
Used data from one site in the Epidemiological Catchment Area study to examine predictors of PTSD in a community sample of 2985 adults. Although prevalence of PTSD was low (1.3%), PTSD was associated with family history of mental illness, parental poverty, child abuse, and parental separation or divorce.

JENKINS, M.A., LANGLAIS, P.J., DELIS, D., & COHEN, R. (1998). **Learning and memory in rape victims with posttraumatic stress disorder.** *American Journal of Psychiatry, 155,* 278-279.
Examined memory function in rape survivors with (n = 15) or without (n = 16) PTSD and in nontraumatized controls. The PTSD group, relative to the other two groups, performed more poorly on delayed free recall. These differences were not explained by comorbid depression, anxiety, or substance abuse.

KING, D.W., KING, L.A., FOY, D.W., & GUDANOWSKI, D.M. (1996). **Prewar factors in combat-related posttraumatic stress**

disorder: Structural equational modeling with a national sample of female and male Vietnam veterans. *Journal of Consulting and Clinical Psychology, 64,* 520-531.
Used structural equation modeling to examine prewar and warzone stressor variables in relation to current PTSD symptoms in 1632 male and female veterans. Among men, younger age at exposure and prior trauma directly predicted PTSD; other family and prewar characteristics had indirect effects. Among women, no prewar variables had direct effects.

KOOPMAN, C., CLASSEN, C., & SPIEGEL, D. (1994). **Predictors of posttraumatic stress symptoms among survivors of the Oakland/Berkeley, Calif., firestorm.** *American Journal of Psychiatry, 151,* 888-894.
Examined the relationship between peritraumatic dissociation and PTSD in a sample of 154 survivors of a large firestorm. Peritraumatic dissociation, loss of personal autonomy, and subsequent stressful life events were related to PTSD symptoms 7-9 months later. In stepwise multiple regression, dissociation was the first variable to enter the prediction equation.

NORRIS, F.H. (1992). **Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups.** *Journal of Consulting and Clinical Psychology, 60,* 409-418.
Examined trauma and PTSD in a sample of 1,000 adults drawn from 4 southeaster cities in the US. Women were more likely than men to have PTSD related to a crime, but did not differ from men in the likelihood of PTSD due to other events. Older, relative to middle-aged or younger individuals, were more likely to have PTSD due to any event, and to crime and accidents as well.

PITMAN, R.K. (1989). **Post-traumatic stress disorder, hormones, and memory.** *Biological Psychiatry, 26,* 221-223.
Discusses the question of how a traumatic event can lead to conditioned responses that are highly resistant to extinction. This question is discussed in terms of neurobiological findings on memory consolidation, a process described by the author as "superconditioning."

SCHNURR, P.P., & VIELHAUER, M.J. (1999). **Personality as a risk factor for PTSD.** In R. Yehuda (Ed.), *Risk factors for posttraumatic stress disorder* (pp. 191-222). Washington, DC: American Psychiatric Press.
Reviews findings on personality as a risk factor for PTSD. The authors present a model of how personality could influence risk, and interpret findings in terms of dimensions of normal personality: neuroticism, extraversion, and conscientiousness. Neuroticism is found to be the most consistent risk factor for PTSD.

SHALEV, A.Y., FREEDMAN, S., PERI, T., BRANDES, D., SAHAR, T., ORR, S.P., & PITMAN, R.K. (1998b). **Prospective study of posttraumatic stress disorder and depression following trauma.** *American Journal of Psychiatry, 155,* 630-637.
Prospectively assessed trauma survivors who presented to an emergency room 1 week, 1 month, 4 months, and 1 year after the trauma. Gender, psychiatric comorbidity, and heart rate in the emergency room were unrelated to the frequency of PTSD or depression at 1 or 4 months. Depression prior to the trauma was related to greater likelihood of PTSD following the trauma.

SOLOMON, Z., MIKULINCER, M., & AVITZUR, E. (1988). **Coping, locus of control, social support, and combat-related posttraumatic stress disorder: A prospective study.** *Journal of Personality and Social Psychology, 55,* 279-285.
Examined the relationship between PTSD symptoms and social support in 262 Israeli soldiers 2 and 3 years after their combat experience. Higher levels of social support were correlated with lower levels of PTSD. Increases in social support were correlated with decreases in PTSD over time.

SPIEGEL, D. (1991). **Dissociation and trauma.** In A. Tasman & S.M. Goldfinger (Eds.), *American Psychiatric Press, Review of Psychiatry, vol. 10* (pp. 261-275). Washington, DC: American Psychiatric Press.
Reviews research findings on peritraumatic dissociation and its relationship to PTSD. Other reactions to trauma are discussed as well, along with guidelines for psychotherapy to treat posttraumatic dissociation.

YEHUDA, R. (1999a). **Biological factors associated with susceptibility to posttraumatic stress disorder.** *Canadian Journal of Psychiatry, 44,* 34-39.
Discusses biological and familial risk factors for PTSD, e.g., among children of Holocaust survivors. The author notes that it is difficult to interpret the increased susceptibility of family members to PTSD following traumatic exposure as evidence of biological / genetic versus experiential factors.

YEHUDA, R. (1999b). *Risk factors for posttraumatic stress disorder.* Washington, DC: American Psychiatric Press.
Summarizes research findings on risk factors for PTSD. Following discussion of methodological issues in the study of risk factors, chapters address epidemiology, genetic factors, family studies, intergenerational transmission, neurocognitive, psychophysiological, and acute responses, and personality.

YEHUDA, R., KEEFE, R.S.E., HARVEY, P.D., LEVENGOOD, R.A., GERBER, D.K., GENI, J., & SIEVER, L.J. (1995). **Learning and memory in combat veterans with posttraumatic stress disorder.** *American Journal of Psychiatry, 152,* 137-139.
Assessed intellectual function in 20 male combat veterans with PTSD and 12 normal controls. The groups were comparable in terms of initial attention, immediate memory, cumulative learning, and active interference, but the PTSD group had more retroactive interference.

YEHUDA, R., & MCFARLANE, A.C. (1995). **Conflict between current knowledge about posttraumatic stress disorder and its original conceptual basis.** *American Journal of Psychiatry, 152,* 1705-1713.
Reviews the historical, political, and social influences on the current understanding of trauma as a cause of PTSD. The authors suggest that although PTSD appears to be a distinct diagnostic entity, that the interpretation of PTSD as a normative response to trauma is inconsistent with research findings.

YEHUDA, R., TEICHER, M.H., TRESTMAN, R.L., LEVENGOOD, R.A., & SIEVER, L.J. (1996). **Cortisol regulation in post-traumatic stress disorder and major depression: A chronobiological analysis.** *Biological Psychiatry, 40,* 79-88.
Assessed predictors of cortisol, MHPG, and PTSD in a sample of 20 female rape survivors. Prior physical of sexual assault was associated with lower cortisol. MHPG was associated with injury during the rape. PTSD status 3 months after the rape was associated with prior assault or injury during the rape, but not cortisol or MHPG.

PILOTS UPDATE

PILOTS database users are often frustrated by the difficulties they face in obtaining copies of the publications their searches discover. Researchers based at major universities may have access to million-volume libraries, and clinicians practicing at large teaching hospitals may have thousands of journals within reach; but the traumatic stress literature is a large and varied one, and even the greatest libraries cover it incompletely. The clinician in private practice, or the academic at a small rural college, has a harder time of it.

Libraries have traditionally relied upon interlibrary loan and document delivery services to supply materials that they themselves cannot provide for their patrons. A national network of medical libraries, coordinated by the National Library of Medicine, makes it possible for small libraries and individual practitioners in the United States to obtain publications that are unavailable locally. The British Library Document Supply Centre serves libraries and individuals throughout the world, and several commercial enterprises offer document delivery as well.

Although our PTSD Resource Center contains the world's largest collection of traumatic stress literature—including every publication indexed in the PILOTS database, except for dissertations and theses—we are not able to supply copies of original documents. In many cases, the journal publishers who provide us with complimentary subscriptions to facilitate our bibliographical work have stipulated that we not provide photocopies from their publications to people outside the National Center. Because most of our collection consists of copies that we have made of copyrighted material (under the "fair use" provision of the copyright law), we cannot legally make further copies from them. And in any case we have neither the staff nor the resources to make copies and to keep the records that copyright laws and guidelines require.

But new forms of access are emerging. Scientific, technical, and medical publishers are increasingly making their publications available to users of the World Wide Web, and offering a range of mechanisms for selling access to them. The traditional library subscription model is one of them, with Internet software making it possible for people affiliated with subscribing institutions to read or print out articles in their homes or offices rather than having to go to the library. And many publishers now sell individual articles to non-subscribers, who can use their credit cards to purchase instant access to the material they need.

Journal publishers and database producers are cooperating to make it easier to go from a bibliographical citation to the full text of the underlying publication. Many citations on PubMed, the free Web interface to the MEDLINE database, include a direct link to the publisher's website. There, upon entry of a password or payment of a fee, the full text can be read, downloaded, or printed. Such links to full text are becoming increasingly common on bibliographical databases—including PILOTS.

We have modified the structure of PILOTS records to allow us to incorporate hypertext links to publishers' websites. We are investigating the mechanisms by which we shall be able to ensure the permanance of these links. And we are beginning by providing links to National Center publications on our own website.

The first of these links already exist. The 66 records in the PILOTS database representing articles that have appeared in the *PTSD Research Quarterly* contain links to the PDF versions of those issues on our website. As we place articles from the *NC-PTSD Clinical Quarterly* and articles written by National Center staff members on the website, we shall incorporate links to those in their PILOTS database records. As with the database itself, no account or password will be required for access to this material.

Based on the experience we gain from this, and on our negotiations with organizations working to facilitate reference linking on the World Wide Web, we shall in future be able to provide links to full text on external websites. This will not only better serve the informational needs of researchers and clinicians; it will also increase the opportunities for the authors of publications on traumatic stress to reach and influence their colleagues, and to receive recognition for their work.

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