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Post-traumatic Stress Disorder and Diabetes: Co-Morbidity and Outcomes in a Male Veterans Sample

Paula M. Trief,[1,2] Paige Ouimette,[2,3] Michael Wade,[2] Paul Shanahan,[4] and Ruth S. Weinstock[4,5]

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The purpose of this study was to assess the prevalence and correlates of comorbid diabetes and Post-Traumatic Stress disorder(PTSD)and potential relationships between PTSD and diabetes outcomes. Male patients enrolled in a VA primary care database ($N = 73{,}270$) were classified as having diabetes from pharmacy records ($N = 14{,}438$) and grouped into those with diagnoses of PTSD with depression ($N = 649$), PTSD-only ($N = 480$), Depression-only ($N = 1696$), Other psychiatric diagnosis ($N = 736$), or No psychiatric diagnosis ($N = 10{,}877$) based on the Purpose of Visit diagnoses in the medical record. Outcomes included glycemic control (HbA1c), cholesterol and tryglycerides. Correlates were age, substance use disorder, other psychiatric diagnosis, number of primary care encounters, and medications. The prevalence of comorbid diabetes and PTSD was 8% ($n = 1129$). Of these, 57% ($n = 649$) had comorbid depression. Patients with PTSD and depression had higher rates of substance use disorder and higher cholesterol and LDL. Patients with depression had poorer glycemic control. Patients with PTSD and depression weighed more and had higher BMI than patients with neither diagnosis. Thus, male diabetes patients with PTSD and depression may be vulnerable to substance use disorders and to weight/lipid problems that can affect health. Depression is a likely contributor to poor glycemic control. Careful screening for mental health comorbidities is needed for diabetes patients.

KEY WORDS: diabetes mellitus; post-traumatic stress disorder; glycemic control; veterans.

There has been a growing interest in the effect that co-morbid mental disorders may have on medical outcomes of patients who have diabetes (Carnethon *et al.*, 2003; Musselman *et al.*, 2003; Thomas *et al.*, 2003). Attention has primarily focused on diabetes and depression, with evidence that people who have diabetes are twice as likely to be depressed as people without chronic disease (Anderson *et al.*, 2001), and that depression is associated with hyperglycemia, mortality, poorer adherence, greater complication risk and poorer health behaviors (Clouse *et al.*, 2003; DeGroot *et al.*, 2001; Egede *et al.*, 2005; Lin *et al.*, 2004; Lustman *et al.*, 2000). Similarly, heightened anxiety symptoms have been reported in 40% of diabetes research participants (Peyrot and Rubin, 1997), and anxiety has been linked to poorer glycemic control, although conflicting results have been reported (Anderson *et al.*, 2002).

Post-traumatic stress disorder (PTSD) is a specific anxiety disorder with an estimated lifetime prevalence of 7.8–25.8% (Fairbank *et al.*, 1995; Kessler *et al.*, 1995; Resnick *et al.* 1993) in the general population (depending on gender of victim, type of trauma and study methodology) and of 14.7–30.9%

[1]Departments of Psychiatry and Medicine, State University of New York (SUNY) Upstate Medical University, Syracuse, NY, USA.
[2]Center for Integrated Healthcare, Veterans Affairs (VA) Medical Center, Syracuse, NY, USA.
[3]Department of Psychiatry, SUNY Upstate Medical University, Syracuse, NY, USA.
[4]Department of Medicine, VA Medical Center, Syracuse, NY, USA.
[5]Department of Medicine and Joslin Diabetes Center, SUNY Upstate Medical University, Syracuse, NY, USA.

in war veteran samples (Centers for Disease Control Vietnam Experience Study, 1988; Hoge *et al.*, 2004; Kang *et al.*, 2003; Kang and Hyams, 2005; Kulka *et al.*, 1990). Although PTSD is often comorbid with other psychiatric disorders (Davidson *et al.*, 1991; Kessler *et al.*, 1995; Kulka *et al.*, 1990), a recent review of the PTSD research literature states that it is "crystal clear that PTSD stands alone as a unique psychiatric disorder. It is not the same as depression, although many PTSD patients are also depressed, and it is not the same as the other anxiety disorders, although PTSD patients frequently also suffer with panic attacks, social avoidance, and obsessive ruminations" (Sullivan and Gorman, 2002, p. 463).

PTSD has been linked to poorer overall health and more somatic symptoms (Baker *et al.*, 1997). This relationship has been found when outcomes are subjectively defined through patient self report (Schnurr and Jankowski, 1999), or more objective physician ratings (Beckham *et al.*, 1998). In a longitudinal study of war veterans, PTSD was associated with higher rates of cardiovascular, musculoskeletal, dermatological and gastrointestinal medical diagnoses (Schnurr *et al.*, 2000). Similarly, more medical conditions, higher rates of circulatory and musculoskeletal conditions, and poorer health-related quality of life were found in VA patients with PTSD, even after controlling for comorbid depression, panic and generalized anxiety disorder (Ouimette *et al.*, 2004).

Diabetes is a disease that may be particularly vulnerable to an effect of PTSD. From a biomedical perspective, chronic stress has been linked to endocrinological abnormalities such as thyroid function changes, hormonal changes, HPA (hypothalamic-pituitary-adrenal) axis dysregulation, which may also relate to diabetes and insulin resistance (Friedman and Schnurr, 1995). Also, recent data showing that PTSD patients have lower cortisol levels, higher T-cell counts, and lower natural killer cell activity (Boscarino, 2004; Ironson *et al.*, 1997) suggest a relationship between PTSD and autoimmune diseases. Type 1 diabetes is an automimmune disease (we do not know if autoimmunity compromise plays a role in type 2 diabetes, the focus of this paper). From a behavioral perspective, PTSD can be related to the unhealthy lifestyle behaviors that lead to obesity and diabetes, such as smoking, lack of exercise, over-eating and alcohol consumption (Solomon, 1988).

A few studies have also suggested that PTSD may be a risk factor for diabetes. For example, Weisberg *et al.* (2002) found that 37% of a primary care sample with anxiety disorders met DSM-IV criteria for PTSD, and these patients had higher rates of various medical conditions, including diabetes, than patients with other anxiety disorders. Similarly, Goodwin and Davidson (2005), drawing on the large ($N = 5877$) National Comorbidity Survey dataset of adults in the community, found that self-reported diabetes was significantly associated with a higher likelihood of PTSD. This was true after adjusting for sociodemographic variables and other mental disorders. Interestingly, diabetes was not associated with depression, or any other mental disorder, in this sample. Frayne *et al.* (2004) studied a large ($N = 4348$) group of female veterans and found that patients with comorbid PTSD and depression reported more medical conditions, and much poorer physical function, than women with depression alone, and described this as the "greater burden of medical illness" (p.1306) for women with PTSD.

These studies are limited by the fact that diagnoses of PTSD and of the medical conditions generally relied on self-report. For example, Frayne *et al.* (2004) classified a subject as having a specific condition, e.g., depression, if she answered yes to the question: "Has a doctor ever told you that you have any of the following: depression. . ." (p. 1308).

With increasing numbers of veterans now serving in combat operations, concerns about the mental health effects of this involvement have increased. Hoge *et al.* (2004) assessed depression, generalized anxiety and PTSD (using standardized questionnaires) of four combat infantry units serving in Iraq or Afghanistan. They found that rates of PTSD were significantly higher after combat duty, with 9.4% prevalence before deployment, and 11.5–19.9% after deployment, and increasing rates were related to increasing exposure to trauma. They argue for the importance of screening for, and treating, PTSD. Given the links identified earlier between PTSD and health outcomes, we believe it is important to also assess the effect of PTSD on physical health.

The purpose of the present study was to answer the following questions:

1. What is the prevalence of PTSD and co-morbid diabetes in a large VA sample?
2. What are the medical/behavioral and psychological correlates of this co-morbidity?
3. Do individuals with co-morbid PTSD and diabetes have poorer diabetes-related medical outcomes?

RESEARCH DESIGN AND METHODS

Study Population

Participants were selected from a search of computerized records of all veterans enrolled in primary care ($N = 73{,}270$) who were released medications between 7/1/03 and 10/01/04 in the VA Healthcare Network Upstate New York (Veterans Integrated Service Network 2). The purposes of visits to providers were extracted, including all primary care providers and psychiatric providers who saw primary care patients in mental health clinics.

Participants were classified as having diabetes if their pharmacy records indicated they had been released insulin or an oral glycemia medication (acarbose, acetohexamide, chlorpropamide, glimepiride, glipizide, glyburide, metformin, miglitol, nateglinide, pioglitazone, repaglinide, rosiglitazone, tolazamide, tolbutamide). A total of 14,795 veterans were thus identified as diabetes patients. Female veterans ($N = 357$ or 2.4%) were excluded due to their small numbers, leaving us with 14,438 male veterans for data analysis.

Procedures

Data were extracted from the VA's information technology system, VISTA, using a data extraction tool, FILEMAN, by the fourth author (PS). The following categories of information were culled from the database: age, glycemic control (measured by HbA1c, a measure of blood glucose control over the preceding 2–3 months), cholesterol (total, LDL and HDL), triglycerides, weight, body mass index (BMI)), number of primary care visits and released medication (insulin, other diabetes medications, psychiatric medications). Data from the most recent visit was used for each variable. Medical and psychiatric diagnoses were based on ICD-9-CM and were noted by providers as the purpose for a visit. A list of codes used for each diagnostic category is available from the second author. The labs and encounter diagnoses both took place in the same time interval. The total duration of the psychiatric diagnosis is unknown, what is known is that the patient was being treated for the psychiatric problem in the interval. The primary care encounter occurred (diagnosis mentioned) between 7/1/03 and 10/1/04. The lab data was obtained in the 1 year interval ending on the date of lab data sampling, roughly 10/4/03 to 10/4/04.

All data extracted from the database were de-identified and this project was approved by the Institutional Review Board of the Syracuse VA Medical Center and met the criteria for exemption from review by the Institutional Review Board of SUNY Upstate Medical University.

Statistical Analyses

Patients in four diagnostic groups (PTSD and depressive disorder, PTSD only, depressive disorder only, and neither PTSD nor depressive disorder) were compared on medical/behavioral health and health care utilization variables. Since data collection consisted of a retrospective database review, conclusions drawn from associations between PTSD/depression and outcomes may have been due to background confounding characteristics such as age, substance use and other psychiatric disorders. Controlling for these confounding variables using standard multi-way analysis of variance is problematic because much of the shared variance between PTSD/depression and the outcomes is removed leading to higher type II errors.

Propensity scores have been observed to reduce the potential bias inherent in observational studies where non-random group assignment often occurs (Rosenbaum and Rubin, 1983). The propensity scores for each subject are equal to the probability of group membership. This probability was calculated constructing a multinomial logistic regression model that included age, substance use, number of primary care visits and other psychiatric disorders and their appropriate interaction coefficients. Thus, four covariates were condensed into a single scalar variable, named the propensity score, designated as the covariate in a second statistical model.

Outcomes defined on a continuous scale were analyzed using analysis of co-variance, whereas binomial logistic regression was utilized for dichotomous outcomes defined with respect to pre-determined threshhold or cut-off points (e.g. HbA1c $>7\%$). These analyses assessed each outcome variable by including a factor for group and a covariate defined as the propensity score. For each statistically significant outcome variable, a post-hoc analysis was conducted using Tukey's (ANCOVA) or Bonferroni's (logistic regression) multiple comparison procedure to preserve the experiment-wise type I error rate. All statistical tests of hypotheses were two tailed and assessed at the 0.05 level of significance.

RESULTS

Diagnostic Groups

Of the participants defined as having diabetes, 8% ($n = 1129$) had PTSD diagnoses. Of these patients, over half (57%; $n = 649$) had comorbid depression diagnoses. Also, 12% ($n = 1,696$) had depression-only diagnoses and 80% ($n = 11,613$) had neither PTSD nor depression diagnoses. Of the group with no PTSD or depression, 6% ($n = 736$) had another psychiatric diagnosis (all ICD-9 psychiatric codes were included).

Demographic and Clinical Characteristics

Table I presents unadjusted demographic and clinical characteristics of the sample. Groups differed significantly on age, the presence of a substance use diagnosis, the presence of another psychiatric diagnosis (excluding substance use disorders), and the number of primary care visits during the study period. PTSD patients were younger than patients without PTSD; patients with depression-only were younger than those without depression. While patients with any psychiatric diagnosis were more likely to have a substance use disorder diagnosis than those without a psychiatric diagnosis, those patients with comorbid PTSD and depression diagnoses had higher rates of substance use disorder diagnoses than all other patient groups.

Patients with depression diagnoses were more likely to be on insulin than those without depression. Patients with psychiatric diagnoses were more likely to be on psychotropic medications, and those patients with comorbid PTSD and depression diagnoses had the highest proportion of psychotropic medication use. Because of these significant differences, the variables of age, presence of a substance use disorder or psychiatric diagnosis other than PTSD or depression, and number of primary care visits, were then used to create the propensity score to control for their influence on the outcomes of interest.

Medical/Behavioral Factors

Table II presents information on the relationship between diabetes outcomes (HbA1c, cholesterol, weight, BMI) and PTSD/depression diagnostic groupings, controlling for potential confounding variables. Each outcome was analyzed as a continuous variable (e.g., range of HbA1c) and as a dichotomous variable (e.g., HbA1c >7% as cut-off for poor glycemic control). Note that superscripts identify the significant differences that were found. Adjusted means and standard errors are presented for continuous variables, and unadjusted frequency and proportions for dichotomous variables. For ease of presentation, adjusted odds ratios are presented, using the no PTSD/no depression group as the comparison group.

Analyzing HbA1c as a continuous variable, results show that the depression-only group had higher HbA1c than the PTSD-only and the neither PTSD-nor depression groups ($p = 0.001$). Using an HbA1c of 7% as the cut-off for good versus poor glycemic control, we found that, in subjects with PTSD, those who were also depressed were 48% more likely to be in poor glycemic control than those who were not diagnosed with depression ($p = 0.023$,

Table I. Characteristics According to PTSD and Depression

	No Depression and no PTSD					
($n = 14,438$)	PTSD with depression ($n = 649$)	PTSD without depression ($n = 480$)	Depression without PTSD ($n = 1696$)	With other psychiatric diagnosis ($n = 736$)	Without other psychiatric diagnosis ($n = 10,877$)	*P*-value
Age	59.6 ± 9.3	61.1 ± 9.7	64.3 ± 11.8	66.6 ± 12.2	69.5 ± 10.3	<0.001
Substance use	140 (22)	61 (13)	178 (11)	75 (10)	155 (1)	<0.001
Other Psychiatric Diagnoses	294 (45)	133 (27)	532 (31)	n/a	n/a	<0.001
# of primary care encounters	3.7 ± 2.6	3.4 ± 2.2	3.5 ± 2.5	3.3 ± 2.5	2.9 ± 2.9	<0.001
Medications						
Insulin	155 (24)	85 (18)	371 (22	109 (15)	1712 (16)	<0.001
Psychotropic	393 (61)	203 (42)	851 (50)	275 (37)	932 (9)	<0.001

Note. Continuous characteristics are expressed as the mean plus or minus the standard deviation, whereas categorical variables as the sum followed by the percentage of patients without missing values.

Table II. Relationship between diabetes outcomes and PTSD/depression diagnoses-Analysis of Covariance (ANCOVA) and logistic regressions adjusted with propensity scores comparing four groups on diabetes-related outcomes (1 = PTSD with Depression, 2 = PTSD without Depression, 3 = Depression without PTSD, 4 = No PTSD and no Depression)

(n = 14,438)	PTSD with depression[1] (n = 649)	PTSD without depression[2] (n = 480)	Depression without PTSD[3] (n = 1696)	No PTSD and No depression[4] (n = 11,613)	P-value*
Medical/Behavioral					
HbA1c (n = 10,639)	7.5 ± 0.06	7.3 ± 0.07	7.5 ± 0.04[2,4]	7.4 ± 0.02	0.001
> 7%	305 (58)	187 (50)	761 (57)	4442 (53)	
	1.2 (0.9–1.5)[2]	0.81 (0.61–1.08)	1.1 (0.93–1.29)	Reference	0.023
Cholesterol (n = 11,581)	175 ± 1.7[3,4]	172 ± 1.9	169 ± 1.0	168 ± 0.4	0.001
>135	506 (89)	375 (88)	1194 (83)	7505 (82)	
	1.45 (0.99–2.11)[3]	1.39 (0.92–2.08)	0.93 (0.75–1.15)	Reference	0.005
LDL mg/dl (n = 11,226)	99.7 ± 1.3[3,4]	98.1 ± 1.5	95.1 ± 0.8	95.7 ± 0.3	0.006
>110	200 (37)	133 (33)	393 (29)	2259 (25)	
	1.41 (1.09–1.82)[3,4]	1.23 (0.92–1.65)	1.02 (0.85–1.22)	Reference	0.002
HDL mg/dl (n = 11,536)	38.2 ± 0.5	38.2 ± 0.6	37.5 ± 0.3[4]	38.9 ± 0.1	<0.001
≤ 50	75 (13)	55 (13)	171 (12)	1188 (13)	0.445
Triglycerides mg/dl (n = 11,529)	214 ± 7.4[4]	194 ± 8.3	203 ± 4.6[4]	186 ± 1.8	<0.001
>150	350 (62)	240 (56)	820 (57)	4385 (48)	
	1.45 (1.13–1.86)[4]	1.22 (0.93–1.59)	1.29 (1.08–1.49)[4]	Reference	<0.001
Weight in lbs (n = 13,650)	222 ± 1.8[4]	217 ± 2.1	218 ± 1.1	215 ± 0.4	0.001
BMI (n = 13,640)	33.3 ± 0.3[4]	32.5 ± 0.3	32.7 ± 0.2[4]	32.2 ± 0.1	<0.001

Note. Continuous outcomes are presented as the adjusted mean plus or minus the standard error. Dichotomous response variables are represented by the frequency and proportion of patients experiencing a particular characteristic. The adjusted odds ratios with Bonferroni adjusted 95% confidence intervals are shown for statistically significant ($p < 0.05$) outcome variables. Odds ratios compare each group to the reference group defined as no PTSD/no Depression. P-values represent the probability of incorrectly rejecting the null hypothesis of no difference in adjusted outcomes among the four groups.

Means and odds ratios in each group are adjusted for the propensity score that was calculated from a multi-nomial logistic regression model (c-stat = 0.747) including age, substance use, other psychiatric disorders and the number of primary care encounters as covariates and group as the dependent variable. Significant pair-wise differences are denoted by a superscripted integer ranging from 1 to 4.

The error degrees of freedom used in the calculation of the F-statistic varies from 10,632 to 14,431. The chi-square statistic used to test the group effect in the logistic regression analysis has 3 degrees of freedom. Sample sizes are dependent on outcome and range from 10,369 to 14,438.

CI = 0.93–1.29). Thus, we conclude that patients with depression diagnoses had poorer blood glucose control than patients without depression diagnoses.

Analyzing lipids as continuous variables, we find that the PTSD and depression group had higher total cholesterol ($p < 0.001$) and LDL ($p = 0.006$) values than patients with depression-only, and than those with neither diagnosis. Using 135 as the cut-off for total cholesterol, we found that, in subjects with depression, those who were also diagnosed with PTSD were 56% more likely to have high cholesterol than those without PTSD ($p = 0.005$, CI = 0.99–2.11). Using 110 as the cut-off for LDL values, in subjects with depression, those who were diagnosed with PTSD were 38% more likely to have high LDL ($p = 0.002$, CI = 1.09–1.82). Findings for HDL were less convincing as patients with depression-only had lower HDL values than patients with neither diagnosis ($p < 0.001$) and, using 50 as the cut-off for HDL, there were no significant differences between the groups ($p = 0.445$). Patients with PTSD and depression diagnoses, and those with depression-only, had higher triglycerides than patients with neither diagnosis ($p < 0.001$). Using 150 as the cut-off for triglycerides, patients with PTSD and depression were 69% ($p < 0.001$, CI = 1.13–1.86), and those with depression-only were 27% ($p < 0.001$, CI = 1.08–1.49), more likely to have high triglycerides than those with no diagnoses.

In addition, patients with PTSD and depression diagnoses weighed more than patients with neither diagnosis ($p = 0.001$). Lastly, patients with PTSD and depression diagnoses and those with depression-only had higher BMI values than patients with neither diagnosis ($p < 0.001$).

Effects of Insulin and Psychiatric Medications

We examined the effects of insulin and psychiatric medications on the association between group and medical variables. No significant interactions were found. Thus, patients on insulin did not differ from those not on insulin, and patients taking psychotropic medications did not differ from those not taking psychotropic medications in these analyses.

CONCLUSIONS

This study was designed to take a first step towards better understanding diabetes patients who have PTSD and identifying potential relationships between PTSD and diabetes outcomes for a large sample of male VA patients. We found that a large number, and significant percentage, of diabetes patients have comorbid PTSD, suggesting that this is an important group for further study. Because PTSD is so often comorbid with depressive disorders (in this study 57%), it was important to compare groups with both diagnoses to those with only one to try to shed light on potentially important relationships.

The findings that the PTSD and depression group were most likely to have a comorbid substance abuse disorder, to have another psychiatric diagnosis (other than substance abuse, PTSD or depression) and to have the highest rate of psychotropic medication use, suggests that PTSD adds a significant extra burden to the mental health challenges of diabetes patients and may specifically increase their risk of substance abuse. We recognize that patients with PTSD without comorbid diabetes are at increased risk of substance abuse and other psychiatric disorders. However, the health implications for diabetes patients make this a particularly important vulnerability.

When we looked at the relationship of PTSD to diabetes-related outcomes, some interesting findings emerged. In terms of glycemic control, depression appears to be the most relevant variable. PTSD alone did not relate to poorer glycemic control, nor did PTSD comorbid with depression significantly change results. However, in terms of lipids, patients with comorbid PTSD and depression had poorer values for total cholesterol and LDL than patients with depression alone or neither diagnosis, and higher triglycerides than patients with neither diagnosis. Since the PTSD-depression group also had higher weight and BMI, these findings may reflect the impact of this comorbidity on obesity and eating behavior. As no differences were found between subjects who took, or did not take, insulin or psychotropic medications, there is no evidence that these medications affected our results.

As a retrospective database review, the study has limitations. Most significantly, we had to rely on the diagnostic input and data provided by providers. Thus, patients were classified as having PTSD because their providers entered this diagnosis into the database as the purpose of the visit. In many cases, these providers were mental health providers, but in some cases diagnoses were made by primary care providers. While we believe this is a significant improvement over the patient self-reports of diagnoses used in most previous studies, future research will need to establish diagnostic accuracy at a higher level (e.g., use of structured clinical interviews). Also, while statistically significant differences did emerge, it is not clear that these differences are clinically significant. We assume that our patients had type 2 diabetes, as it is unlikely that men with type 1 diabetes would serve in the military (although type 1 diabetes might develop in adulthood). However, this is not a certainty. Also, it is likely that there were individuals with undiagnosed diabetes, or individuals who had diabetes that was not being treated with medications, who were missed. Thus, studies that include direct assessments of diabetes outcomes and comparisons of type 1 and type 2 diabetic patients who have comorbid PTSD would be useful. Finally, our sample was limited to male veterans who use the VA healthcare system. Data suggests that these veterans are more likely to be unemployed and nonwhite, as well as in poorer health, more functionally limited and have a lower income than male veterans not using the VA (Haas and Watts, 2005). Thus, results may not generalize to other male veterans, to female veterans, or to PTSD patients whose symptoms relate to noncombat traumatic incidents. Studies of other patient groups would also be helpful.

In summary, our data suggests that male diabetes patients who have both PTSD and depression may be especially vulnerable to substance abuse disorders and to weight and lipid problems that could affect their overall health. Depression emerges, again, as an important correlate of poor glycemic control. Future research should include more direct and stringent assessments of both diabetes (e.g., diabetes type and HbA1c through lab measurements) and psychiatric comorbidities using structured clinical interviews, as well as assessments of the generalizability of these findings to other patient groups (e.g.,

women, non-war-trauma patients). In addition, it will be important to begin to identify the mechanisms by which PTSD, and other psychiatric comorbidities, might affect diabetes outcomes. We do not know if the pathway is biological, hormonal, adherence-related or through some other yet unidentified path.

Recent evidence suggests that patients with mental health conditions may receive less intensive medical care (e.g., no HbA1c tests, no LDL test, no eye exams) for their diabetes (Frayne *et al.*, 2005). Our data supports the conclusion that careful screening of mental health comorbidities is important, and suggests that these individuals are in greatest need of high quality care. If these findings are supported in future research, they might lead to the recommendation that diabetes quality improvement programs pay particular attention to patients with PTSD and/or depression, and especially to those dealing with the medical and behavioral effects of their comorbidity, as these patients may need enhanced interventions to improve diabetes outcomes.

REFERENCES

Anderson, R. J., Freedland, K. E., Clouse, R. E., and Lustman, P. J. (2001). The prevalence of comorbid depression in adults with diabetes: A meta-analysis. *Diabetes Care* 24: 1069–1078.

Anderson, R. J., Grigsby, A. B., Freedland, K. E., de Groot, M., McGill, J. B., Clouse, R. E., and Lustman, P. J. (2002). Anxiety and poor glycemic control: A meta-analytic review of the literature. *Int. J. Psychiatry Med.* 32: 235–247.

Baker, D. G., Mendenhall, C. L., Simbartl, L. A., Magan, L. K., and Steinberg, J. L. (1997). Relationship between posttraumatic stress disorder and self-reported physical symptoms in Persian Gulf War veterans. *Arch. Intern. Med.* 157: 2076–2078.

Beckham, J. C., Moore, S. D., Feldman, M. E., Hertzberg, M. A., Kirby, A. C., and Fairbank, J. A. (1998). Health status, somatization and severity of posttraumatic stress disorder in Vietnam combat veterans with posttraumatic stress disorder. *Am. J. Psychiatry* 155: 1565–1569.

Boscarino, J. A. (2004). Posttraumatic stress disorder and physical illness: Results from clinical and epidemiologic studies. In Yehuda, R., and McEwen, B., (Eds.), (*Biobehavioral Stress Response: Protective and Damaging Effects. Annals of the New York Academy of Sciences* (vol. 1032, pp. 141–153). New York, NY.

Carnethon, M. R., Kinder, L. S., Fair, J. M., Stafford, R. S., and Fortmann, S. P. (2003). Symptoms of depression as a risk factor of incident diabetes: Findings from the National Health and Nutrition Examination Epidemiologic Follow-up Study, 1971–1992. *Am. J. Epidemiol.* 158: 416–423.

Centers for Disease Control Vietnam Experience Study (1988). Health status of Vietnam veterans: I. Psychosocial characteristics. *JAMA* 259: 2701–2707.

Clouse, R. E., Lustman, P. J., Freedland, K. E., Griffith, L. S., McGill, J. B., and Carney, R. M. (2003). Depression and coronary heart disease in women with diabetes. *Psychosom. Med.* 65: 376–383.

Davidson, J. R. T., Hughes, D., Blazer, D. G., and George, L. K. (1991). Posttraumatic stress disorder in the community: An epidemiological study. *Psychol. Med.* 21: 713–721.

DeGroot, M., Anderson, R. J., Freedland, K. E., Clouse, R. E., and Lustman, P. J. (2001). Association of depression and diabetes complications: A meta-analysis. *Psychosom. Med.* 63: 619–630.

Egede, L. E., Nietert, P. J., and Zheng, D. (2005). Depression and all-cause and coronary heart disease mortality among adults with and without diabetes. *Diabetes Care* 28: 1339–1345.

Fairbank, J. A., Schlenger, W. E., Saigh, P. A., and Davidson, J. R. T. (1995). An epidemiologic profile of post-traumatic stress disorder: Prevalence, comorbidity, and risk factors. In Friedman, M. J., Charney, D. S., and Deutch, A. Y. (Eds.), *Neurobiological and Clinical Consequences of Stress; From Normal Adaptation to PTSD* (pp. 415–427). Lippincott-Raven, Philadelphia, PA.

Frayne, S. M., Halanych, J. H., Miller, D. R., Wang, F., Lin, H., Pogach, L., Sharansky, E. J., Keane, T. M., Skinner, K. M., Rosen, C. S., and Berkowitz, D. R. (2005). Disparities in diabetes care: Impact of mental illness. *Arch. Intern. Med.* 165: 2631–2638.

Frayne, S. M., Seaver, M. R., Loveland, S., Christiansen, C. L., Spiro, A. 3rd, Parker, V. A., and Skinner, K. M. (2004). Burden of medical illness in women with depression and posttraumatic stress disorder. *Arch. Intern. Med.* 164: 1306–1312.

Friedman, M. J., and Schnurr, P. P. (1995). The relationship between trauma, post-traumatic stress disorder, and physical health. In Friedman, M. J., Charney, D. S., and Deutsch, A. Y. (Eds.), *Neurobiological and Clinical Consequences of Stress: From Normal Adaptation to PTSD* (pp. 507–524). Lippincott-Raven Pub, Philadelphia, PA.

Goodwin, R. D., and Davidson, J. R. (2005). Self-reported diabetes and posttraumatic stress disorder among adults in the community. *Prev. Med.* 40: 570–574.

Haas, L. B. and Watts, S. A. (2005). Diabetes care in the Veterans Health Administration. *Diabetes Spectr* 18: 159–162.

Hoge, C. W., Castro, C. A., Messer, S. C., McGurk, D., Cotting, D. I., and Koffman, R. L. (2004). Combat duty in Iraq and Afghanistan, mental health problems, and barriers to care. *N. Engl. J. Med.* 351: 13–22.

Ironson, G., Wynings, C., Schneiderman, N., Baum, A., Rodriguez, M., Greenwood, D., Benight, C., Antoni, M., LaPerriere, A., Huang, H. S., Klimas, H. S., and Fletcher, M. A. (1997). Posttraumatic stress symptoms, intrusive thoughts, loss, and immune function after Hurricane Andrew. *Psychosom Med.* 59: 128–141.

Kang, H. K., Natelson, B. H., Mahan, C. M., Lee, K.Y., and Murphy, F. M. (2003). Post-traumatic stress disorder and chronic fatigue syndrome-like illness among Gulf War veterans: A population-base survey of 30,000 veterans. *Am. J. Epidemiol.* 157: 141–148.

Kang, H. K. and Hyams, K. C. (2005). Mental health care needs among recent war veterans. *N. Engl. J. Med.* 352: 1289.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., and Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Arch. Gen. Psychiatry* 52: 1048–1060.

Kulka, R. A., Schlenger, W. E., Fairbank, J. A., and Weiss, D. (1990). *Trauma and the Vietnam War Generation: Report of Findings from the National Vietnam Veterans Readjustment Study*. Brunner/Mazel, New York.

Lin, E. H. B., Katon, W., VonKorff, M., Rutter, C., Simon, G. E., Oliver, M.. Ciechanowski, P., Ludman, E.J., Bush, T., and Young, B. (2004). Relationship of depression and diabetes self-care, medication adherence and preventive care. *Diabetes Care* 27: 2154–2160.

Lustman, P. J., Anderson, R. J., Freedland, K. E., DeGroot, M. K., Carney, R. M., and Clouse, R. E.(2000). Depression and poor

glycemic control: A meta-analytic review of the literature. *Diabetes Care* 23: 934–942.
Musselman, D. L., Betan, E., Larsen, H., and Phillips, L. S. (2003). Relationship of depression to diabetes types 1 and 2: Epidemiology, biology, and treatment. *Biol. Psychiatry* 54: 317–329.
Ouimette, P., Cronkite, R., Henson, B. R., Prins, A., Gima, K., and Moos, R. H. (2004). Posttraumatic stress disorder and health status among female and male medical patients. *J. Trauma. Stress* 17: 1–9.
Peyrot, M. and Rubin, R. R. (1997). Levels and risks of depression and anxiety symptomatology among diabetic adults. *Diabetes Care* 20: 585–590.
Resnick, H. S., Kilpatrick, D. G., Dansky, B. S., Saunders, B. E., and Bedst, C. L. (1993). Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *J. Consult. Clin. Psychol.* 61: 984–991.
Rosenbaum, P. R. and Rubin, D. B. (1983). The central role of the propensity score in observational studies for causal effects. *Biometrica* 70: 41–55.
Schnurr, P. P., Ford, J. D., Friedman, M. J., Green, B. L., Dain, B. J., and Sengupta, A. (2000). Predictors and outcomes of posttraumatic stress disorder in World War II veterans exposed to mustard gas. *J. Consult. Clin. Psychol.* 68: 258–268.
Schnurr, P. P. and Jankowski, M. K. (1999). Physical health and post-traumatic stress disorder: Review and synthesis. *Semin. Clin. Neuropsychiatry* 4: 295–304.
Solomon, Z. (1988). Somatic complaints, stress reaction, and posttraumatic stress disorder: A three-year follow-up study. *Behav. Med.* 14: 179–185.
Sullivan, G. M. and Gorman, J. M. (2002). Finding a home for posttraumatic stress disorder in biological psychiatry. Is it a disorder of anxiety, mood, stress, or memory? *Psychiatr. Clin. N. Am.* 25: 463–468.
Thomas, J., Jones, G., Scarinci, I., and Brantley, P. (2003). A descriptive and comparative study of the prevalence of depressive and anxiety disorders in low-income adults with type 2 diabetes and other chronic illnesses. *Diabetes Care* 26: 2311–2317.
Weisberg, R. B., Bruce, S. E., Machan, J. T., Kessler, R. C., Culpepper, L., and Keller, M. B. (2002). Nonpsychiatric illness among primary care patients with trauma histories and posttraumatic stress disorder. *Psychiatr. Serv.* 53: 848–854.