## Levels and Risks of Depression and Anxiety Symptomatology Among Diabetic Adults

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**OBJECTIVE** — To determine levels of depression and anxiety symptoms among adults with diabetes and identify factors associated with increased risk.

**RESEARCH DESIGN AND METHODS** — This study administered self-report symptom inventories to patients at the beginning (n = 634) and end (n = 578) of an outpatient diabetes education program. Subjects (n = 246) contacted by mail 6 months later completed the same instruments.

**RESULTS** — Rates of disturbance for depression (41.3%; 95% CI: 37.4–45.2%) and anxiety (49.2%; 95% CI: 45.3–53.1%) were higher than those typical in the general population (10–20%). Probability of disturbance ranged from 5–7% for those with the lowest risk profile to 82–92% for those with the highest risk profile. Diabetes-related complications were the only disease factor associated with significantly increased risk of disturbance. Women and those with less education were at much higher risk. Only 13% of those followed for 6 months were disturbed at all three time-points.

**CONCLUSIONS** — Diabetes is associated with increased risk of psychological disturbance, especially for those with more diabetes-related complications. Sociodemographic factors account for much of the risk differential among people with diabetes.

ubstantial research has suggested that people with diabetes have higher levels of psychological disturbance, especially depression, than exist in the general population (1). However, the recent comprehensive review by Gavard et al. (1) indicated that published research generally has been based on small samples of diabetic subjects (n < 200) with rather large CIs. Thus, it has not been possible to obtain reliable prevalence estimates for subsamples, including those known to have different levels of psychological disturbance in the general population, e.g., sex, age, race, marital status, and socioeconomic status (2-5), or for subgroups defined by diabetes-related factors such as presence of complications, level of metabolic derangement, duration of disease, and type of diabetes.

There is a clear need for studies that provide accurate estimates of psychological disturbance rates for subsamples since several hypotheses concerning higher rates of psychological disturbance among people with diabetes identify disease factors as mediating or causal factors. For example, type of diabetes is associated with the burden of illness because some forms of the disease require more intrusive treatment regimens (6). Duration of diabetes is an indicator of chronic stress and is a risk factor for medical complications and psychological disturbance (7,8). Complications decrease physical quality of life, which is associated with higher risk of psychological disturbance (9-11). Finally, it has been proposed that diabetes and depression are expressions of a common metabolic derangement (12,13). The latter

mechanisms have been the basis for suggesting that diabetes carries a special risk for psychological disturbance, beyond that attributable to chronic disease in general. A number of these hypotheses can be addressed by examining variations in rates of psychological disturbance in defined subgroups.

## RESEARCH DESIGN AND

**METHODS** — Subjects were adults with diabetes who enrolled in the 1-week comprehensive outpatient diabetes education program at Johns Hopkins Hospital in Baltimore, MD (14-16). Patients were referred by community physicians or selfreferred. Consecutive admissions from October 1985 through November 1990 were approached to participate in the study. Only a small number (<5%) refused to participate, and a similarly small number were not included in the study because a member of the research staff was not able to obtain data before the education program had been initiated, leaving a total of 682 who agreed to participate in the study. Complete information from the preprogram data collection protocol was not available for 48 patients, leaving a total of

Of those who completed the preprogram data collection protocol, 578 (91%) completed the program and participated in the postprogram data collection protocol 4 days later. During part of the study resources were available to conduct a 6-month follow-up. Attempts to contact 413 subjects who completed the postprogram protocol were successful in obtaining follow-up data from 246 (60%).

To determine whether nonresponse biased the results of the follow-up data we performed an analysis of potential differences between responders and nonresponders among those for whom contact was attempted. Student's t tests and  $\chi^2$  statistics were used to examine whether people who responded at follow-up were different at program entry from nonresponders. Comparisons were made for all variables used in the analysis. The only difference that was significant at the P = 0.10 level was marital

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Table 1—Sample characteristics

Sex	TO 0 (2T4)
Female	59.0 (374)
Male	40.5 (257)
Race	(0.0 (200)
White (and other)	60.3 (382)
Black	34.5 (219)
Marital status	· · · · · · · · · · · · · · · · · ·
Married	51.9 (329)
Not married	44.3 (281)
Age (years)	
<30	15.9 (98)
30–39	13.1 (83)
40–49	18.6 (118)
50–59	17.7 (112)
60–69	23.8 (151)
70+	10.9 (69)
Education	
Less than high school	19.5 (124)
High school graduate	50.9 (323)
College graduate	26.0 (165)
Disease type	
IDDM	32.0 (203)
NIDDM (insulin)	30.9 (196)
NIDDM (no insulin)	35.2 (235)
Duration of diabetes (years)	
<1	10.3 (65)
1–5	28.8 (179)
6–10	20.0 (127)
11–19	20.0 (127)
20–29	11.2 (71)
≥30	8.8 (55)
Glycemic control (HbA <sub>1c</sub> )	
<9.5	28.5 (181)
9.5–12.0	29.5 (187)
≥12.0	28.2 (179)
Complications	
Type	
Retinopathy	25.7 (163)
Nephropathy	7.1 (45)
Peripheral neuropathy	46.5 (295)
Autonomic neuropathy	14.2 (90)
Impotence	17.5 (111)
Peripheral vascular	28.5 (181)
Coronary artery	18.6 (118)
Skin	18.9 (120)
Infections	22.2 (141)
Number	
0	25.9 (164)
1	21.0 (133)
2	20.5 (130)
3–4	21.9 (139)
5–9	10.7 (68)
Total	100.0 (634)

Data are % (n). Figures do not sum to 100 because missing data is not shown.

status; nonresponders were more likely to be unmarried (42.5 vs. 52.4%, P = 0.06). To eliminate these differences when examining persistence of disturbance over the 6-month study period, only subjects who completed the follow-up are included at each time-point.

Subjects were approached on the 1st day of the program, before any educational activities had taken place. Subjects filled out the Center for Epidemiological Studies Depression (CESD) (17) and Zung Self-Rating Anxiety (ZSRA) questionnaires (18). Medical and sociodemographic characteristics (see Table 1) were obtained by chart audit from medical histories taken on the 1st day of the program. HbA<sub>1c</sub> was measured by gel electrophoresis (upper limit for the normal range of the nondiabetic population = 7.7%).

Participants completing the program were approached at the end of the program to fill out a packet of questionnaires containing the depression and anxiety instruments. Six months later a packet of questionnaires containing the depression and anxiety instruments was mailed to subjects. Telephone calls were made to encourage nonrespondents to reply.

The criteria for depression and anxiety are the conventional cutoff points for the self-report symptom inventories. Because these measures are not diagnoses by trained medical professionals the term "psychological disturbance" (rather than "psychopathology") is used to refer to scores above the cutoff points. Presence of disturbance for depression was assessed by a CESD score of 16 or higher (17). Presence of disturbance for anxiety was assessed by a ZSRA score of over 36 (19).

A supplementary analysis was conducted to determine whether prevalence rates were influenced by the fact that some psychological symptoms are also symptoms of metabolic disregulation. Because hypoglycemia and hyperglycemia can produce symptoms such as sweating, dizziness, and lethargy, we recalculated prevalence after eliminating all items that might represent symptom confounding (questions 7 and 11 on the CESD and questions 3, 6–8, 10, 11, 14–17, 19, and 20 on the ZSRA). Cutoff levels were determined using mean item scores for the recalculated totals that were equal to those that produced the cutoff points on the original scales.

Logistic regression was used to estimate odds ratios and 95% CIs (20). If the 95% CI does not include zero, the category is

significantly different from the reference category. Where there were missing data for a particular variable, this category was included in the estimates as a dummy variable, but is not presented in Tables 1–3 (21). For multicategory variables, an extreme category of risk (highest or lowest rate of disturbance) was chosen as the reference category; the same reference category was used for both types of disturbance.

**RESULTS** — Subjects entering the program had a high prevalence of depressive (41.3%) and anxious (49.2%) symptomatology (see Table 2). These levels are considerably higher than the estimates of depression (16.7%) and anxiety (9%) in the general population, using similar measurement techniques (1,19). Most subjects (56.8%) manifested at least one type of disturbance, and 38.0% had both.

Univariate odds ratios (see Table 2) show that being a woman, unmarried, less educated, and aged 40–49 years was associated with a higher likelihood of being depressed. Being a woman, black, less educated, aged 40–49, and having more complications was associated with higher likelihood of being anxious.

Multivariate odds ratios (see Table 3) for being depressed were similar to the univariate results, except that higher complications (3 or more) were significantly associated with higher depression. Multivariate results for being anxious were similar to the univariate results, except that being black was no longer a risk factor and the presence of multiple complications (more than 1) was a consistent risk factor.

Using the multivariate odds ratios, synthetic probability estimates for the highest and lowest risk subjects were computed (22). The estimates represent the probability that a subject with a given risk profile would be depressed or anxious. Subjects with no risk factors had an expected prevalence rate of 5% for depression and 6% for anxiety; subjects with all risk factors had an expected prevalence rate of 92% for depression and 91% for anxiety. No subjects in our sample had a profile that included either no risk factors or all risk factors, so actual values for highest and lowest risk cases in our sample were 8 and 86% for depression and 10 and 90% for anxiety. When only statistically significant (P < 0.05) risk factors were used in computing the synthetic estimates, the low and high estimates were 7 and 83% for depression and 7 and 82% for anxiety (actual values for the highest and lowest risk cases in our sample were 10 and 81% for depression and 11 and 86% for anxiety, respectively).

A separate analysis was conducted on the measures that eliminated hyperglycemic and hypoglycemic symptom confounding. At program admission 38.2% met the recomputed criterion for being depressed compared with 41.3% using the total set of items. Rates for being anxious using the two measures also were similar; 52.5% met the recomputed criterion for anxiety compared with 49.2% using the total set of items. Rates for both types of disturbance using the recomputed measures fell within the 95% CIs for the original estimates.

Subjects who completed the education program (n = 578) had a significantly lower prevalence at the end of the education program than those same subjects had at the beginning of the program for both being depressed (24.9 vs. 41.7%; P < 0.001) and anxious (30.3 vs. 48.1%; P < 0.001). For those who completed the 6-month followup (n = 246) prevalence at both follow-up (T3) and post-program (T2) was lower than those same subjects' scores at pre-program (T1) for depression (T1 = 38.0%, T2 = 21.6%, T3 = 27.8%; P < 0.005) and anxiety (T1 = 42.0%, T2 = 27.8%, T3 = 26.9%; P <0.001). For subjects present at all three times, only 13.1% were above the depression cutoff at all three times and only 13.5% were above the anxiety cutoff at all three times; thus only one-third of those with a psychological disturbance at admission to the program remained disturbed for 6 months.

**CONCLUSIONS** — The results of this study regarding nondisease factors are consistent with other studies of psychological disturbance. Sex is a risk factor for disturbance (12,23,24), with women twice as likely to be disturbed as men. Race is a risk factor in the univariate analysis of anxiety, but when other factors are controlled the risk is not significant. Education is strongly associated with psychological disturbance (7,9), with college graduates experiencing less than half the risk of those who did not graduate from high school. Being married is also associated with lower disturbance for depression (7,25). Highest rates of disturbance were found in the middle-aged groups (40-49 for depression and 30-39 for anxiety), consistent with studies that have found lower rates of disturbance among older age-groups (7,9).

Overall levels of psychological disturbance were higher than in studies of gen-

Table 2—Rates and univariate risk of depression and anxiety

	Depression		Anxiety	
	Rate	OR (95% CI)	Rate	OR (95% CI)
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Sex	40 4 (101)	2.00 (1.40. 2.00)	50.0 (210)	2 44 (1 75 2 40)
Female Male	48.4 (181)	2.08 (1.49–2.90)	58.0 (218)	2.44 (1.75–3.40)
	31.1 (80)	1.00 ()	36.2 (93)	1.00 (—)
Race	24 5 (210)	1.00()	20 7 (140)	1.00 ( )
White (and other) Black	34.5 (219)	1.00 (—)	38.7 (148)	1.00 (—)
Marital status	38.7 (148)	1.32 (0.95–1.85)	46.3 (177)	1.43 (1.03–2.00)
	26.2 (110)	1.00()	49.0 (150)	1.00()
Married	36.2 (119) 48.0 (135)	1.00 (—)	48.0 (158) 50.9 (143)	1.00 (—)
Not married	48.0 (133)	1.63 (1.17–2.28)	30.9 (143)	1.12 (0.82–1.53)
Age (years) <30	44.9 (44)	1 55 (0 01 2 64)	45 0 (45)	1 00 (0 66 1 92)
30–39	42.2 (35)	1.55 (0.91–2.64) 1.40 (0.81–2.43)	45.9 (45) 54.2 (45)	1.09 (0.66–1.82)
40–49	53.4 (63)	2.18 (1.34–3.56)	55.9 (66)	1.52 (0.90–2.58) 1.63 (1.00–2.66)
50-59	35.7 (40)	1.06 (0.64–1.77)	49.1 (55)	1.25 (0.76–2.03)
60–69	34.4 (52)	1.00 (0.04–1.77)	43.7 (66)	1.00 (—)
70+	39.1 (27)	1.22 (0.68–2.20)	49.3 (34)	1.25 (0.71–2.20)
Education	39.1 (21)	1.22 (0.00-2.20)	T9.5 (JT)	1.25 (0.71-2.20)
Less than high school	51.6 (64)	2.77 (1.70-4.53)	62.1 (77)	3.56 (2.18–5.81)
High school graduate	44.0 (142)	2.03 (1.35–3.07)	53.3 (172)	2.46 (1.66–3.64)
College graduate	27.9 (46)	1.00 ()	31.5 (52)	1.00 (—)
Disease type	21.9 (10)	1.00 (—)	31.3 (32)	1.00 ()
IDDM	42.4 (86)	1.16 (0.79–1.72)	46.8 (95)	.79 (0.54–1.18)
NIDDM (insulin)	38.8 (76)	1.00 (—)	52.6 (103)	1.00 (—)
NIDDM (no insulin)	42.6 (100)	1.17 (0.79–1.74)	48.5 (114)	.85 (0.59–1.24)
Duration of diabetes (years)	12.0 (100)	1.17 (0.79-1.74)	10.5 (114)	.03 (0.39-1.24)
<1	36.9 (24)	0.76 (0.37–1.56)	46.2 (30)	1.03 (0.50–2.13)
1–5	47.5 (85)	1.17 (0.64–2.15)	53.6 (96)	1.39 (0.76–2.55)
6–10	37.0 (47)	0.76 (0.40–1.46)	42.5 (54)	.89 (0.47–1.66)
11–19	41.7 (53)	0.92 (0.48–1.76)	52.8 (67)	1.34 (0.71–2.50)
20–29	35.2 (25)	0.70 (0.34–1.46)	50.7 (36)	1.23 (0.61–2.50)
30+	43.6 (24)	1.00 (—)	45.5 (25)	1.00 ()
Glycosylated hemoglobin (		1.00 ( )	15.5 (25)	1.00 ( -)
<9.5	37.6 (68)	1.00 (—)	47.0 (85)	1.00 (—)
9.5–12.0	40.6 (76)	1.14 (0.75–1.72)	47.6 (89)	1.03 (0.68–1.56)
>12.0	43.6 (78)	1.28 (0.83–1.98)	51.4 (92)	1.20 (0.79–1.81)
Complications	13.0 (10)	1.20 (0.03 1.70)	31.7 (32)	1.20 (0.1)
0	37.8 (62)	1.00 (—)	41.5 (68)	1.00 ()
1	38.3 (51)	1.02 (0.64–1.63)	45.9 (61)	1.20 (0.75–1.92)
2	44.6 (58)	1.32 (0.83–2.12)	56.9 (74)	1.86 (1.16–2.98)
3–4	42.4 (59)	1.21 (0.77–1.90)	51.1 (71)	1.48 (0.94–2.32)
5–9	47.1 (32)	1.46 (0.83–2.58)	55.9 (38)	1.79 (1.01–3.15)
Total	41.3 (262)	•	49.2 (312)	•

Data are % (n) or odds ratio (OR) (95% CI).

eral populations not composed of people with chronic health conditions (17% for depression and 9% for anxiety). This replicates earlier research documenting higher rates of psychological disturbance among people with diabetes. The rate of depressive disturbance observed in this study is lower than only one study reported by Gavard et al. (1); that study, which also used a CESD

score of 16 as the criterion with a clinic sample, found a current prevalence of 60% (26). The other study reported by Gavard et al. (1) that used a CESD score of 16 as the criterion was based on a community sample of people with diabetes and reported a rate of 26.1%, a figure close to that observed in this study after participation in the diabetes education program.

Table 3—Multivariate odds of depression and anxiety

	Depression	Anxiety
Sex		
Female	2.29 (1.58-3.33)	2.66 (1.84–3.87)
Male	1.00 (—)	1.00 ()
Race		
White (and other)	1.00 (—)	1.00 ()
Black	0.87 (0.58–1.31)	0.94 (0.62-1.42)
Marital status		
Married	1.00 (—)	1.00 (—)
Not married	1.55 (1.07–2.25)	1.01 (0.70-1.47)
Age		
<30	1.46 (0.69-3.08)	1.43 (0.68–3.02)
30–39	1.52 (0.81–2.85)	1.92 (1.02-3.59)
40–49	2.16 (1.27–3.67)	1.72 (1.01-2.91)
50–59	1.04 (0.60–1.80)	1.22 (0.72-2.07)
60–69	1.00 (—)	1.00 (—)
70+	1.30 (0.67–2.53)	1.57 (0.81–3.05)
Education		
Less than high school	2.64 (1.52–4.57)	3.56 (2.06–6.16)
High school graduate	1.90 (1.23–2.92)	2.39 (1.55–3.67)
College graduate	1.00 (—)	1.00 (—)
Disease type		
IDDM	1.27 (0.72–2.24)	0.89 (.50–1.57)
NIDDM (insulin)	1.00 (—)	1.00 (—)
NIDDM (no insulin)	1.36 (0.89–2.10)	0.97 (0.63–1.49)
Duration of diabetes (years)		
<1	0.87 (0.35–2.14)	1.54 (0.62–3.79)
1–5	1.51 (0.69–3.30)	2.08 (0.95–4.54)
6–10	0.93 (0.43–2.04)	1.23 (0.56–2.70)
11–19	0.97 (0.46–2.04)	1.60 (0.76–3.37)
20–29	0.76 (0.34–1.74)	1.54 (0.67–3.50)
30+	1.00 (—)	1.00 (—)
Glycosylated hemoglobin (%)		( )
<9.5	1.00 (—)	1.00 (—)
9.5–12.0	1.06 (0.68–1.67)	0.97 (0.62–1.52)
>12.0	1.13 (0.70–1.80)	1.03 (0.66–1.62)
Complications		/ >
0	1.00 ()	1.00 (—)
1	1.30 (0.78–2.16)	1.54 (0.92–2.56)
2	1.67 (0.98–2.83)	2.39 (1.41–4.05)
3–4	1.90 (1.10–3.28)	2.20 (1.27–3.81)
5–9	3.29 (1.62–6.66)	3.49 (1.72–7.07)

Data are odds ratio (95% CI).

The rate of depressive disturbance in our study at entrance to the program fell roughly halfway between the high and low extremes of these two studies.

Although rates of psychological disturbance in this study were high, caution must be used in interpreting this study as support for the hypothesis that psychological disturbance is higher among people with diabetes. First, the measurement technique used may have contributed to the high rate

of psychological disturbance in our sample. Self-reported symptomatology is a crude criterion for "caseness," which may have low positive-predictive power for psychiatric diagnosis (27). Symptom-based rates generally are higher than those obtained by clinical diagnosis (28).

Second, it may be inappropriate to generalize from this study to all people with diabetes because this clinical population may be a biased sample of all people with

diabetes. Specifically, the clinical sample may be biased on the dependent variable itself. That is, psychological disturbance is associated with increased help-seeking (29,30). People in this study may have sought treatment (diabetes education) because of pessimism about their current state of health and prognosis, along with a sense of helplessness in managing their disease. The high rate of disturbance identified in the current study may be partly a result of this bias. Such bias is one limitation of a clinical sample, but even a case-control design in which controls are spouses (31) or relatives (32) of diabetic patients is not adequate because it contains bias in the selection of diabetic subjects into the sample. Eliminating this bias requires the use of a community sample to identify both diabetic and nondiabetic subjects.

Sample bias might also be implicated in the substantial lack of persistence over time in depression and anxiety symptoms; roughly one-third of those disturbed at program entry were not disturbed 4 days later after participating in the diabetes education program. This finding is consistent with the possibility noted above that subjects may have entered the diabetes education program because they were distressed, especially in regard to their diabetes. As they became more empowered in regard to their diabetes, they may have experienced fewer symptoms of anxiety and depression. Of course, it also is possible that people would have become less disturbed without treatment, e.g., because of regression to the mean.

This study provides no direct test of the hypothesis that diabetes has unique effects on levels of psychological disturbance because people with diabetes were not compared with people with other chronic medical conditions. However, a variety of studies have found that while medical conditions in general are a risk factor for psychological disturbance, diabetes carries no special risk (33-36). The comprehensive review by Gavard et al. (1) contained two studies of adults that compared rates of depression between people with diabetes and those with other medical conditions; neither found a significant difference (26,37). The cutoff used in this study has produced rates of anxiety disturbance of 32% for family practice patients and 50% for cardiac service patients (19); the rate of anxiety in the present study fell halfway between those two rates for medical patients.

Although this study did not compare people with and without diabetes, it can

provide evidence on the risk associated with two factors that have been hypothesized to account for the increased risk of psychological disturbance in diabetes—disease severity and complications. There was no support for the hypothesis that diabetes is a special risk for depression because they are associated at a biochemical level; glycemic control and disease type were not related to level of depression (nor anxiety) symptoms. Moreover, the rate of anxiety was also high, indicating that the risk is not specific to depression, but rather to global psychological distress. Furthermore, the supplementary analysis showed that eliminating the items from the psychological symptom inventories that represented symptoms of glycemic control did not substantially alter the rates of disturbance for depression and anxiety. Although this study provides no evidence of a biological mechanism for psychological disturbance, this finding should be viewed with caution because the criterion variables were symptom levels rather than psychiatric diagnoses. It is possible that diagnosed psychopathology would be more strongly associated with biological factors (38).

A second hypothesis about why diabetes might be associated with higher rates of psychological disturbance than other diseases is because, unlike other diseases, diabetes is a significant risk factor for a number of other debilitating and potentially fatal conditions. There was some support for this hypothesis because a greater number of complications was associated with higher levels of depression and anxiety symptoms. However, psychological disturbance was higher among diabetic patients with no complications than among the general population. Moreover, diseases that are complications of diabetes are also present in the general population, although at a lower rate than among people with diabetes. Therefore, although complications may have a major impact on psychological disturbance, they cannot entirely account for the higher rates of depression and anxiety symptoms among people with diabetes. Also, because the onset of disturbance and complications could not be correlated, we cannot rule out the alternative possibility that disturbance might cause complications (39).

One methodological note should be made regarding the analysis of complications in this study. In a preliminary analysis (results not shown) each type of complication was entered as a separate risk factor. Using that analysis approach, only peripheral vascular disease was significantly (P < 0.05) related to psychological disturbance (both depression and anxiety). That analysis approach, often used in research on the relationship between depression and complications of diabetes (12,23,32,40), substantially underestimated their relationship in our study.

While rates of psychological disturbance in this study were high, diabetes itself does not automatically result in a high risk of disturbance. Prevalence for risk subgroups ranged from 5 to 92%. People without risk factors other than diabetes are at relatively low risk for psychological disturbance. At the other end of the risk continuum, the question remains of how diabetes might combine with other risk factors to influence psychological disturbance whether diabetes has a simple additive effect or its effect is potentiated by other risk factors. Answering this question will require comparison of the effect of other risk factors among a community sample of healthy, diabetic, and chronically ill nondiabetic people, preferably in a longitudinal study.

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