International Journal of Obesity (1998) 22, 688–694
 0307–0565/98 $12.00
http://www.stockton-press.co.uk/ijo

Symptoms of depression and changes in body weight from adolescence to mid-life

JC Barefoot[1,2], BL Heitmann[2], MJ Helms[1], RB Williams[1], RS Surwit[1] and IC Siegler[1]

[1]*Behavioral Medicine Research Center, Duke University Medical Center, Durham, NC 27710, USA; and* [2]*Institute of Preventive Medicine, Copenhagen Municipal Hospital, Copenhagen, Denmark*

OBJECTIVE: To investigate the relationship of symptoms of depression to weight changes in healthy individuals of normal weight across a follow-up of over 20 y.
PARTICIPANTS AND DESIGN: College students (3885 men and 841 women) were administered a self-report depression measure in the mid-1960s. Their baseline body mass index (BMI) was calculated from their college medical records. Participants were contacted by mail in the late 1980s and asked to report their current height and weight as well as their smoking and exercise habits. Another measure of depressive symptoms was obtained from 3560 individuals at follow-up. Multiple regression models were used to relate changes in weight to depression scores while controlling for background (gender, baseline BMI and the gender by BMI interaction) and behavioral (exercise and smoking) predictors.
RESULTS: The relationship between depressive symptoms and body weight change took the form of an interaction with baseline BMI ($P < 0.001$). Those with high baseline depression scores gained less weight than their nondepressed counterparts if they were initially lean, but more if they were initially heavy. This trend was especially strong in those with high depression scores at both baseline and follow-up.
CONCLUSIONS: The findings support the hypothesis that depression exaggerates pre-existing weight change tendencies. This pattern would not have been detected by an examination of main effects alone, illustrating the need to move toward more complicated interactive models in the study of psychological factors and weight.

Keywords: depression; longitudinal studies; body weight

Introduction

Symptoms of depression have a number of physiological and behavioral concomitants, including altered neuroendocrine functioning,[1] smoking,[2] alcohol consumption[3] and physical inactivity.[4] A number of studies have examined the associations of depression and other forms of psychological distress with body weight, but their results do not present a consistent or clear picture. Some of these investigations have examined the prevalence of clinical depression and other psychopathologies in obese individuals and have found little evidence to suggest that they have unusually high rates of these disorders.[5] Studies that have investigated associations of milder forms of psychological distress to body mass have had mixed results (for example, see Refs 6–10). Some of the inconsistencies may be the result of the use of cross-sectional designs that create interpretive ambiguities, because it is plausible that psychological distress could be a consequence of being overweight,[6] as well as a precursor of body weight change.[11] Prospective studies are necessary to address questions regarding causal directionality.

Correspondence: Dr John C. Barefoot, Box 2969, Duke Medical Center, Durham, NC 27710, USA.
Received 10 November 1997; revised 26 February 1998; accepted 2 March 1998

One of the most promising lines of investigation deals with depression and depressive symptomatology as precursors of body weight change. Noppa and Hällström[12] found that depression in middle-aged women was associated with subsequent weight gain. DiPietro *et al*[11] examined depressive symptoms as predictors of weight change over approximately eight years and observed a more complex pattern of relationships. Among persons aged < 55 y, who generally gain weight over time, the effect of depression was modified by both gender and education. Young men with high depression scores gained more than the nondepressed, especially if they had a low level of education. Young depressed women gained less than nondepressed women, but this trend was confined to those with less than a high school education. Among older participants, where weight maintenance or loss are the norms, those who were depressed lost more than the nondepressed regardless of gender. The authors summarized the bulk of these results by suggesting that depression may amplify age-dependent patterns of weight change,[11] especially in less educated groups. This description can also be applied to findings from another study that examined weight change among psychiatric patients who were subject to recurrent depression.[13] Depression led to weight gain in some patients and weight loss in others. The direction of the change tended to be consistent within individuals across episodes. Moreover, those who

were initially lean were more likely to lose weight and those who were heavy were more likely to gain weight. This pattern can be interpreted as another illustration of the tendency for depression to magnify the effects of existing predispositions.

The present study examines changes in body weight in a large sample of students who were administered a measure of depressive symptoms at college entry and contacted again more than 20 years later. We first addressed the hypothesis that high levels of depressive symptoms lead to an exaggeration of weight change predispositions. The homogeneity of the sample made it impractical to investigate the effects of depression on weight change tendencies associated with age and education. However, it was possible to investigate the potential interaction of depressive symptoms with personal predispositions as represented by initial body mass. We hypothesized that high levels of depression in adolescence would lead to larger than expected weight gains among the initially heavy participants and less than expected weight gains among the leaner participants.

A second question was the relative impact of stable *vs* transient depressive symptoms. Several studies (for example, see Refs 4, 14, 15) have reported large test–retest correlations of depression scales across extended time periods, indicating considerable stability in this psychological characteristic. However, depressive symptoms are undoubtedly temporary in many individuals. One would expect the impact of depression on weight change to be greater among those for whom the symptoms are relatively stable rather than transient. In the present study, it was possible to get a rough estimate of the stability of the symptoms by comparing the baseline depression scores to another measure of depression obtained at approximately the same time as the follow-up measure of body weight. It was predicted that the effects of depressive symptoms would be especially strong among those with high scores both at baseline and at follow-up.

Finally, the study examined the extent to which the effects of depression were modified by gender. DiPietro *et al*[11] observed that the effects of depression differed between men and women in the younger age group. We evaluated that interactions, as well as other potential gender differences.

Methods

Participants

The University of North Carolina Alumni Heart Study[16] is an ongoing longitudinal study of the role of psychosocial factors in the development of coronary heart disease. The target population is a group of approximately 7000 students who enrolled at the University of North Carolina in the years 1964–1966. At enrollment these students were administered the Minnesota Multiphasic Personality Inventory (MMPI),[17] which serves as a source of baseline psychosocial indicators. Follow-up of this sample was initiated in 1987 with the first mail questionnaire and has continued through seven waves of data collection.

The present study is based on those participants who were aged <25 y at baseline. Follow-up height and weight were obtained from 4860 individuals, 80% of the living members of the target sample who were located. Data of 134 participants were omitted from analyses due to missing values on key variables, leaving 841 women and 3885 men in the final sample. The sample is highly educated and almost completely of European-American origin. The ethnic and gender composition of the sample reflects the composition of the University of North Carolina in the mid-1960's. Information on participant characteristics is presented in Table 1.

The possibility of selection bias was evaluated by comparing participants in the present analyses to those not in the study on the prevalence of depressive symptoms (see below for definition). Nonparticipants were more likely to be classified as having a high level of symptoms (23.5% *vs* 18.5%, X^2 1 df $= 15.4$, $P < 0.001$). It was not possible to investigate the possibility of selection bias due to the other critical variable, body weight, because of lack of access to medical records of nonparticipants to obtain baseline height and weight.

Measures

Baseline depression. The most commonly used MMPI measure of depression is the D scale, which has its origins in work with psychiatric samples. Items on the D scale are heterogeneous and many of them are not face valid reflections of depression.[17] However, the scale has been divided on the basis of face validity into obvious and subtle subscales.[18] The 40-item Obvious Depression subscale (OBD) is a more

Table 1 Participant characteristics

	Men (s.d.)	*Women (s.d.)*
Baseline		
Mean age (y)	18.6 (1.0)	18.9 (1.5)
Mean height (m)	1.8 (0.1)	1.6 (0.1)
Mean weight (kg)	71.9 (10.6)	56.5 (8.3)
% BMI >30 kg/m^2	1.6	1.2
% In depressed category	18.2	20.1
Follow-up		
Mean age (y)	40.5 (1.5)	40.4 (1.8)
Mean weight (kg)	82.8 (12.8)	62.8 (13.3)
% BMI >30 kg/m^2	7.8	6.4
% Smokers	17.5	15.9
Mean exercise (h/week)	3.7 (3.3)	2.7 (2.5)
% In depressed category	18.2	20.6

BMI = body mass index.

straightforward measure of depressive symptoms as experienced outside the psychiatric context. It has been found to be more appropriate than the D scale for nonhospitalized samples[19] and to correlate more highly with other depression criteria.[20] It was therefore chosen as the measure for the present study to increase the relevance of the findings for nonpsychiatric populations.

Analyses were performed with OBD treated both as a continuous and dichotomous variable. No substantial differences were observed in the results, so only analyses using the dichotomous variable will be reported. A high level of depressive symptoms was defined as OBD scores one or more standard deviations above the mean of the gender specific United States norms.[17] The cut-points were scores of 11 for men and 13 for women. It is important to note that this depression categorization does not reflect clinical depression according to DSM-IV criteria, but only identifies those with a relatively large number of depressive symptoms.

Body mass. Baseline height and weight were obtained from records of the medical examination required of participants upon entry into university. This examination took place at approximately the same time as the administration of the MMPI. Body mass index (BMI) at baseline was calculated as weight (kg) divided by height squared (m^2).

Follow-up height and weight were obtained from self-reports on the questionnaire completed by the participants in the late 1980's. Self-reports of body mass are not as desirable as physical measurements, but discrepancies between the two types of measures tend to be small and occur mainly at the extremes of the BMI distributions.[21,22] The difference of body weight at follow-up minus weight at baseline, was the primary dependent variable.

Behavioral covariables. Smoking and exercise, variables known to influence body weight, were used as covariates to control for potential confounding. Information regarding these behaviors was available from the follow-up questionnaire, but not at baseline. Smoking at follow-up was defined as a dichotomous variable (yes/no). The exercise indicator was continuous and based on the response to the question, 'How many hours a week, on average, do you exercise or play sports for fun or to keep in shape, not counting job or housework activities?'

Follow-up depression. The NEO Personality Inventory[23] was administered to participants in 1988. This test is designed to be a comprehensive inventory of normal personality, based on the five-factor taxonomy of traits.[24] The instrument contains an eight-item depression subscale, that comprises our measure of follow-up depression status. Our unpublished data showed that the correlation of the NEO depression score with the OBD measure was 0.73 in a sample of 48 community volunteers.

As with the baseline depression measure, we used gender specific norms to classify participants into dichotomous categories of depressive symptom levels. The critical value for this classification was defined as one or more standard deviations above the mean of these norms.[23]

Analysis strategy

Initial analyses examined the associations of the background (gender, baseline BMI) and behavioral (smoking, exercise) variables with weight change. Those that were found to be significant predictors were included as covariables in subsequent models.

In the course of the initial analyses, it was observed that the variability of weight change was strongly related to the baseline BMI, with more variability in weight change among those who had higher initial body mass (*see* Results). This pattern of variability creates conditions of heteroscedasticity that violate the assumptions of multiple linear regression. To correct this problem, the dependent variable, weight change, was converted into a ratio by dividing it by baseline BMI and the inverse of baseline BMI was used on the independent variable side of the equation. The regression coefficients from the resulting models were converted to estimators of the original untransformed variables. This is the strategy recommended by Neter and Wasserman[25] for dealing with this pattern of heteroscedasticity.

Tests of significance for the depression effects were conducted in a series of multiple regression models with the weight change score as the dependent variable. The first model included the main effect for the dichotomous depression variable and controls for background variables found to be significant predictors of weight change (*See* Results). The second added the term for the predicted interaction between depression and baseline BMI. The third added the term for the predicted gender by depression interaction and the fourth model added the three-way (gender $\times$ BMI $\times$ depression) interaction. Finally, these steps were repeated with additional controls for behavioral variables associated with weight change: exercise and smoking. The added controls for behavioral variables resulted in the loss of data for 103 participants due to missing values.

Analyses examining depression at both baseline and follow-up were limited to the 3560 participants who completed both depression measures. Those who did not complete the follow-up depression measure did not differ on baseline depression from those who did. They did have significantly lower BMI's at both baseline ($t = 4.2$, $P < 0.001$) and follow-up ($t = 6.8$, $P < 0.001$). Participants were categorized into three groups: those not classed as depressed at either baseline or follow-up ('Neither'; $n = 2473$), those whose

scores indicated high symptom levels at both time points ('Both'; $n=255$), and those who had high depression scores at one time point, but not the other ('Once'; $n=832$). The Once category contains two types of participants whose depression status was not stable: those classed as depressed only at baseline and those who were depressed only at follow-up. These two groups had similar weight change patterns. The same modeling strategy described in the previous section was repeated, with this three-level categorical value substituted for the dichotomous baseline depression indicator.

Results

Background and behavioral predictors of weight change

Univariate comparisons found that men gained a mean of 10.9 kg compared to only 6.3 kg for women ($P<0.001$). Weight changes were negatively correlated with baseline BMI and this relationship was stronger for men ($r=-0.28$, $P<0.001$) than for women ($r=-0.04$). Not only was baseline BMI related to the mean weight change, it was also related to the variability of weight change scores. For example, the variance of weight changes in the highest tertile of baseline BMI was more than twice as great as that in the lowest tertile (733 *vs* 330).

Self-reports of smoking and exercise at follow-up were also related to weight change. On average, women who were smokers at follow-up had gained only 4.2 kg, compared to 6.6 kg for nonsmoking women ($P<0.02$). This association was not significant for men (10.4 *vs* 11.0 kg). Weight changes of ex-smokers were not different from those of never smokers. Number of hours of reported exercise was negatively correlated with weight change in both men ($r=-0.15$, $P<0.001$) for women ($r=-0.20$, $P<0.001$).

Baseline depression scores as predictors of weight change

The first set of multiple regression models controlled for gender, baseline BMI, and their interaction. They confirmed the absence of an overall depression main effect, but revealed that the interaction between depression status and baseline BMI was highly significant ($F(1, 4719)=12.77$, $P<0.001$). These effects can be seen in Figure 1, which presents the unadjusted associations of baseline depression categories with subsequent median weight change stratified by gender and quintile of baseline BMI. It is apparent that there is no simple linear association between depression status and weight change. There is little difference between depression groups in the middle of the baseline BMI distribution, but there is in the extremes. Depressed participants who were initially lean appear to have gained less weight than lean participants who were not depressed. Depressed participants who were initially heavy appear to have gained more weight than heavy participants who were not depressed. This interaction pattern is especially striking in the data of women. However, the tests of the gender by depression interaction and the three-way interaction were not significant. The estimates for depressed women are less stable than the others, presumably because of the smaller number of individuals in that group.

While there is no main effect of depression, it is clear that it did have sizeable consequences for some parts of the sample. In the highest quintile of baseline BMI the odds of a woman being among the 34% who gained >10 kg were 2.2-times higher if she was classified as depressed than if she was not. The comparable odds ratio was 1.3 for men, who also had a 34% prevalence of that weight gain.

The addition of controls for exercise and smoking did not substantially reduce the size of the predicted depression by baseline BMI interaction ($F(1, 4615)=12.0$, $P<0.001$). Tests of other interactions involving depression were not significant. This model is presented in Table 2.

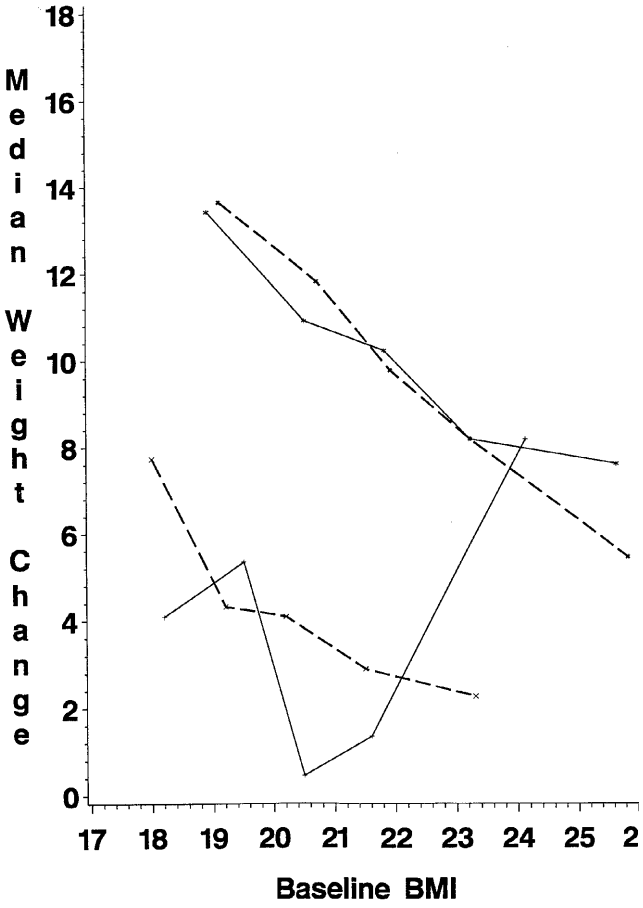

Figure 1 Median weight gain by gender, depression category, and quintile of baseline body mass index (BMI). Upper two lines are for men. Solid lines are for those with high levels of depressive symptoms.

Table 2 Model of weight change as a function of baseline depression ($R^2 = 0.191$, $n = 4673$)

Effect	*b*	*P*
Intercept	81.61	
Exercise	−0.89	0.0001
Smoking	−2.00	0.0092
Female gender	−50.10	0.0001
Baseline BMI	−2.44	0.0001
Gender × BMI	1.70	0.0001
Depression	−19.68	0.0008
Depression × BMI	0.95	0.0006

BMI = body mass index.

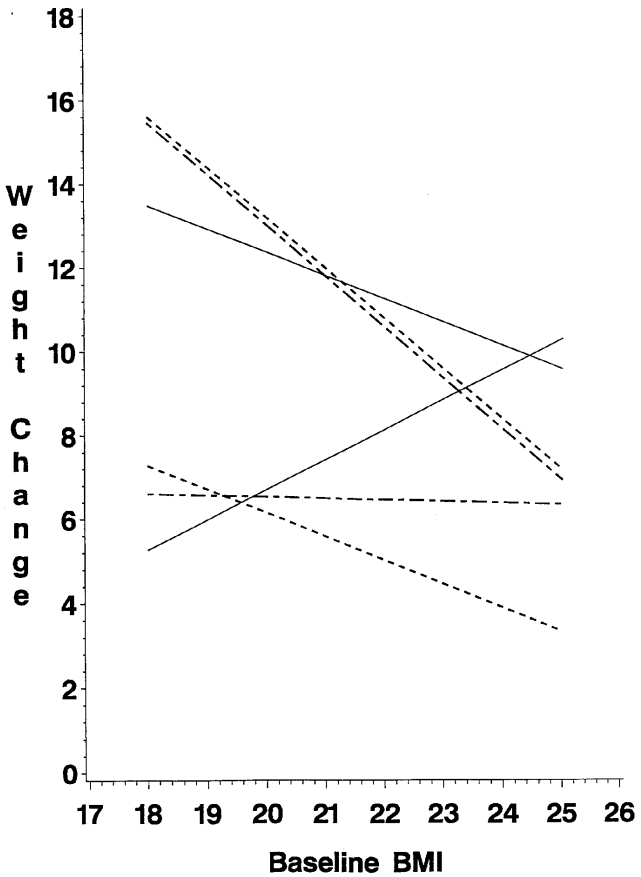

Figure 2 Predicted weight gain by gender, depression category, and baseline body mass index (BMI). – – – – – Neither; - - - - - - Once; ———— Both. Upper three lines are for men.

Weight change as a function of depression status at baseline and follow-up

Figure 2 displays the predicted weight changes calculated from the model containing gender, baseline BMI, the three depression categories (Both, Once and Neither time) and all their interactions. The basic pattern for those with high depression scores, a less than expected gain if initially lean and a more than expected gain if initially heavy, is most apparent among those who were classed as depressed at both time points.

A model with nonsignificant terms deleted, revealed a significant interaction between baseline BMI and the three depression categories ($F\ (2,\ 3552) = 6.54$, $P = 0.002$). Comparisons of the gradients revealed that the slope of the Both group differed from that of the Once group ($P = 0.003$) as well as from that of the Neither group ($P < 0.001$). The comparison between the slopes for Once and Neither groups was not significant. Addition of the behavioral covariates did not appreciably alter the magnitude of the interaction or its significance ($F\ (2,\ 3533) = 6.93$, $P = 0.001$). This model is presented in Table 3.

Table 3 Model of weight change as a function of baseline and follow-up depression ($R^2 = 0.203$, $n = 3543$)

Effect	*b*	*P*
Intercept	85.78	
Exercise	−0.88	0.0001
Smoking	−2.05	0.0193
Female gender	−53.43	0.0001
Baseline BMI	−2.67	0.0001
Gender × BMI	1.88	0.0001
Depression		
Neither	(referent)	
Once	−2.89	0.65
Both	−33.87	0.0003
Depression × Baseline BMI		
Neither	(referent)	
Once	0.135	0.65
Both	1.629	0.0002

BMI = body mass index.

Discussion

The primary hypothesis of the study, the enhancement of existing predispositions by the presence of depressive symptoms, was supported. Those who reported high levels of depressive symptoms gained less weight than their nondepressed counterparts if they were initially lean, but more if they were initially heavy. These patterns would not have been detected by an examination of the simple depression main effect. The findings of this and other studies (for example, see Ref. 11) illustrate the need to move toward more complicated interactive models in the study of psychological factors and weight.

The combination of baseline depression with a second depression measure near the time of follow-up improved our ability to account for body weight changes. Those depressed at both time points showed exaggerated weight change tendencies compared to those depressed at only one or neither test administration. The interpretation of this finding is complicated by the fact that it is not (and cannot be) a truly prospective analysis. Levels of depression at follow-up may have been influenced by the prior history of weight change. In addition, the method for estimating the temporal stability of depressive symptoms was far from ideal. Nonetheless, the pattern of results that emerged tends to support the proposition that chronic depressive symptoms are particularly important in their associations with body weight changes.

Findings regarding gender differences were less clear. The gender differences among those with lower education reported by DiPietro *et al*[11] were not observed, perhaps because our sample was highly educated. There were trends for the effects of depressive symptoms to be stronger in the data of women, but those trends were not statistically significant. It is difficult to make any conclusive statements about the relative impact of depression on weight change in men and women on the basis of the present study, partially because of the relatively small number of women in the sample. However, it should be noted that many of the observed relationships between psychological factors and body weight have been obtained in samples of women.[6,7,12,14]

One can only speculate regarding the mechanisms that might underlie these phenomena. Central serotonergic metabolism has been implicated in both depression and obesity. Dysphoric mood and hyperphagia can both be modified by altering brain serotonin reuptake and/or release. However, typically, agents that have a therapeutic effect of mood have little or no effect on appetite and *vice versa*.[26] Chronic depressive symptoms may serve to alter metabolism.[13] It is also possible some heavy individuals use eating behavior as a means of coping with psychological distress or dysthymia. Carbohydrate ingestion can increase brain serotonin levels by increasing the transport of tryptophan through the blood brain barrier.[27,28] This is thought to influence mood.

Interpretation of these findings must take into account several limitations of the study. Due to the study design, potential confounders could only be assessed at follow-up and not at baseline or at intervening time points. We were also unable to obtain baseline BMI values for nonparticipants, so selection bias due to that variable could not be evaluated. There was evidence of possible selection bias on the depression variable, with those reporting more symptoms at baseline less likely to later enroll in the longitudinal study. This is not surprising, but it could bias the findings. However, this bias could not account for the pattern of interactions that were observed and would tend to work against finding differences. Finally, we do not have data on the use of anti-depressants or other drugs that could have influenced weight change, although the absence of severe depression in the sample makes it unlikely that anti-depressants were widely used. To account for the observed findings anti-depressants would have to be differentially prescribed to heavy and lean depressed participants, an improbable pattern.

It is important to note that few individuals in the sample had depression scores that would suggest psychopathology and few had BMI's indicative of serious obesity. These phenomena primarily occurred in individuals within the normal range of both variables. Even so, the consequence of depressive symptoms were clear and important for some subgroups in the sample, especially those in the highest quintile of baseline BMI. The present study probably underestimates the magnitude of the effects in the general population since the sample was highly educated and the impact of depression appears to be stronger in lower socioeconomic groups,[11] where it is also more prevalent.[29]

It is becoming increasingly recognized that subclinical levels of depressive symptoms can have important consequences for health (for example, see Refs 14, 30). The same is true for variations in weight within the normal range.[31,32] The body weight changes associated with depressive symptoms may further enhance the impact of depression on physical health.

Conclusion

These data support the notion that depression tends to exaggerate preexisting weight change tendencies, particularly among those with high depression scores at both baseline and follow-up. These findings were observed only with interactive modeling and no main effects of psychological factors on weight were present. This underscores the importance of applying a more comprehensive modeling approach when studying psychosocial influences on weight changes.

Acknowledgements

This study was supported in part by grants 5F06 TW01952 from the Fogarty International Center; P01 HL36587 and R01 HL55356 from the National Heart, Lung and Blood Institute; R01 AG09276, R01 AG12458, P20 AG12058, and P60 AG11268 from the National Institute on Aging and R05 MH70482 from the National Institute of Mental Health.

References

1 Carney RM, Freedland KE, Rich MW, Jaffe AS. Depression as a risk factor for cardiac events: A review of possible mechanisms. *Ann Behav Med* 1995; **17**: 142–149.
2 Anda RF, Williamson DF, Escobedo LG, Mast EE, Giovino GA, Remington PL. Depression and the dynamics of smoking. *JAMA* 1990; **264**: 1541–1545.
3 Frederick T, Frerichs RR, Clark VA. Personal health habits and symptoms of depression at the community level. *Prev Med* 1988; **17**: 173–182.
4 Farmer ME, Locke BZ, Moscicki EK, Dannenberg AL, Larson DB, Radloff LS. Physical activity and depressive symptoms: the NHANES I Epidemiologic Follow-up Study. *Am J Epidemiol* 1988; **128**: 1340–1351.
5 Stunkard AJ, Wadden TA. Psychological aspects of human obesity. In: *Obesity*, Bjorntorp P, Brodoff BN (eds). Lippincott: Philadelphia, 1992.
6 Rumpel C, Ingram DD, Harris TB, Madans J. The association between weight change and psychological well-being in women. *Int J Obes* 1994; **18**: 179–183.
7 Wing RA, Matthews KA, Kuller LH, Meilahn EN, Plantinga P. Waist to hip ratio in middle-aged women: Associations with behavioral and psychosocial factors and with changes in cardiovascular risk factors. *Arteriosclerosis and Thrombosis* 1991; **11**: 1250–1257.

8 Kittel F, Rustin RM, Dramaix M, deBacker G, Kornitzer M. Psycho-socio-biological correlates of moderate overweight in an industrial population. *Psychsom Res* 1978; **22**: 145–158.
9 Cohen S, Schwartz JE, Bromet EJ, Parkinson DK. Mental health, stress, and poor health behaviors in two community samples. *Prev Med* 1991; **20**: 306–315.
10 Klesges RC, Klem ML, Klesges LM. The relationship between changes in body weight and changes in psychosocial functioning. *Appetite* 1992; **19**: 145–153.
11 DiPietro L, Anda RF, Williamson DF, Stunkard AJ. Depressive symptoms and weight change in a national cohort of adults. *Int J Obes* 1992; **16**: 745–753.
12 Noppa H, Hällström T. Validity of the MMPI depression scale for outpatients. *Psychological Assessment* 1981; **3**: 55–59.
13 Stunkard AJ, Fernatrom MH, Price RA, Frank E, Kupfer DJ. Direction of weight change in recurrent depression. Consistency across episodes. *Arch Gen Psychiatry* 1990; **47**: 857–860.
14 Barefoot JC, Schroll M. Symptoms of depression, acute myocardial infarction, and total mortality in a community sample. *Circulation* 1996; **93**: 1976–1980.
15 Costa PT, McCrae RR. Depression as an enduring disposition. In: *Diagnosis and treatment of depression in late life: Results of the NIH Consensus Development Conference*. Schneider LS, Reynolds CF, Lebowitz BD, Friedhoff AJ (eds). American Psychiatric Press: Washington DC, 1991.
16 Siegler IC, Peterson BL, Barefoot JC, Williams RB. Hostility during late adolescence predicts coronary risk factors at midlife. *Am J Epidemiol* 1992; **136**: 146–154.
17 Greene RL. *The MMPI-2/MMPI: An interpretive manual*. Allyn and Bacon: Boston, 1991.
18 Weiner DN. Subtle and obvious keys for the MMPI. *J Consul Psychol* 1948; **12**: 164–170.
19 Nelson LD, Chichetti D. Validity of the MMPI depression scale for outpatients. *Psychological Assessment* 1991; **3**: 55–59.
20 Burgess PM, Campbell IM, Zylberberg A. Face validity *vs* Item subtlety in the MMPI D Scale. *J Clin Psychol* 1984; **40**: 499–504
21 Kuskowskia-Wolk A, Berström R, Boström G. Relationship between questionnaire data and medical records of height, weight, and body mass. *Int J Obes* 1998; **16**: 1–9.
22 Sørensen TIA, Stunkard AJ, Teasdale TW, Higgins MW. The accuracy of reports of weight: Children's recall of their parents' weights 15 years earlier. *Int J Obes* 1983; **7**: 115–122.
23 Costa PT Jr., McCrae RB. *The NEO personality inventory manual*. Psychological Assessment Resources: Odessa, 1985.
24 Digman JM. Personality structure: Emergence of the five-factor model. *Annual Review of Psychology* 1990; **41**: 417–440.
25 Netter J, Wasserman W. *Applied linear statistical models. Regression, analysis of variance, and experimental designs*. Richard D. Irwin, Inc.: Homewood, 1974.
26 National Task Force on the Prevention and Treatment of Obesity. Long-term pharmacolotherapy in the management of obesity. *JAMA* 1996; **18**: 1907–1915.
27 Fernstrom JD, Wurtman RM. Brain serotonin content: Increase following ingestion of carbohydrate diet. *Science* 1971; **174**: 1023–1025.
28 Lyons PM, Truswell S. Serotonin precursor influenced by type of carbohydrate meal in healthy adults. *Am J Clin Nutr* 1988; **47**: 433–439.
29 Murphy JM, Olivier DC, Monson RR, Sobol AM, Federman EB, Leighton AH. Depression and anxiety in relation to social status. *Arch Gen Psychiatry* 1991; **48**: 223–229.
30 Anda R, Williamson D, Jones D, Macera C, Eaker E, Glassman A, Marks J. Depressed affect, hopelessness, and the risk of ischemic heart disease in a cohort of US adults. *Epidemiology* 1993; **4**: 285–294.
31 Willet WC, Manson JE, Stampfer MJ, Colditz GA, Rosner B, Speizer FE, Hennekens CH. Weight, weight change, and coronary heart disease in women. Risk within the 'normal' weight range. *JAMA* 1995; **273**: 461–465.
32 Epstein FH, Higgins M. Epidemiology of obesity. In: *Obesity* Bjorntorp P, Brodoff BN (eds). Lippincott: Philadelphia, 1992.