Psychological Medicine (2011), **41**, 1665–1676. © Cambridge University Press 2011
doi:10.1017/S0033291710002473

ORIGINAL ARTICLE

Depression trajectories and obesity among the elderly in Taiwan

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Background. The present study aimed to (*a*) characterize 10-year trajectory patterns of depressive symptoms and (*b*) investigate the association between depressive trajectory and subsequent obesity, metabolic function and cortisol level.

Method. In a prospective study of Taiwanese adults aged ⩾60 years ($n=3922$) between 1989 and 1999, depression was assessed using a 10-item short-form of the Center for Epidemiologic Studies Depression Scale and information on body mass index (BMI) was collected by self-report. A subsample ($n=445$) of the original cohort in 1989 was drawn to assess metabolic variables and cortisol levels in a 2000 follow-up. After trajectory analyses were performed, multinomial logistic regression analyses were used to estimate the association estimates.

Results. We identified four distinctive trajectories of depressive symptoms: class 1 (persistent low, 41.8%); class 2 (persistent mild, 46.8%); class 3 (late peak, 4.2%); and class 4 (high-chronic, 7.2%). The results from both complete cases and multiple imputation analyses indicated that the odds of obesity were lower in the class 2, 3 or 4 elderly, as compared with those in class 1, while the odds of underweight were higher. The classes of older adults with more and persistent depressive symptoms showed a trend toward having both a lower BMI ($p=0.01$) and a higher cortisol level ($p=0.04$) compared with those with low depressive symptoms.

Conclusions. Incremental increases in depressive symptoms over time were associated with reduced risk of obesity and higher cortisol levels.

Received 22 February 2010; Revised 18 October 2010; Accepted 23 November 2010; First published online 5 January 2011

Key words: Body mass index, cortisol levels, depressive symptoms, longitudinal studies, trajectory analysis.

Introduction

Both depression (Blazer, 2003; Djernes, 2006) and obesity (Arterburn *et al*. 2004; Zamboni *et al*. 2005; McTigue *et al*. 2006) are increasingly prevalent in older adults and linked to adverse health consequences. A reciprocal relationship between depression and obesity has gradually drawn investigators' attention to its complex manifestation and underlying mechanisms (Stunkard *et al*. 2003; Markowitz *et al*. 2008). To this point, the evidence concerning depression and obesity seems conflicting; for example, several cross-sectional studies have reported a positive association between depression and obesity in elderly with a mean age of 63 years (Roberts *et al*. 2000, 2003), some reported lack of such an association in older adults aged >65 years (Heo *et al*. 2006), and others even reported an inverse relationship in elderly aged >65 years (Li *et al*. 2004; Kuriyama *et al*. 2006; Ho *et al*. 2008). A recent meta-analysis of 15 longitudinal studies conducted in the USA or Europe found a modest and positive association between depression and obesity (Luppino *et al*. 2010). Whether such an association exists in non-Western populations remains to be investigated.

Studies on the severity and duration of depression in late life have shown that the chronic nature of depression and levels of depressive symptoms may evolve over time (Beekman *et al*. 2002; Mojtabai & Olfson, 2004). Yet, prior studies on the connection between depression and obesity largely relied on

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depressive symptoms or diagnosis at a single period that usually failed to take time-varying depressive manifestation into account; they may therefore not adequately reflect how change and stability of depressive symptoms have an impact on obesity (Faith *et al*. 2002). Furthermore, a diagnostic approach may include patients of different courses or neglect people with subsyndromal depression (Amore *et al*. 2007; Hybels *et al*. 2009). To overcome these limitations, trajectory analysis has been proposed to identify the source of heterogeneity within a sample of individuals (Nagin & Tremblay, 2001). In such analysis, subjects with a similar degree of depression are assigned to groups based on their initial level and depressive symptom changes over time. These group memberships therefore can be adopted to examine the subsequent outcome of obesity.

Depression has been linked to a variety of metabolic abnormalities, including alterations in levels of blood glucose, lipid profile and blood pressure (Skilton *et al*. 2007). In the wake of concerns for depression and obesity, researchers have begun to place greater emphases on possible mechanisms accounting for depression–obesity links; one frequently explored is the roles that cortisol and metabolic factors play in the association (Vogelzangs *et al*. 2009). A recent review on molecular and clinical evidence further pointed out that dysregulation of the hypothalmic–pituitary–adrenal axis, often measured by means of cortisol levels, may be one of the key factors for the shared susceptibility to depression and obesity (Bornstein *et al*. 2006).

In the present study, we turned to a 10-year study on a representative sample of community-dwelling elders in Taiwan, which offers the opportunity to assess temporal variation in depressive symptoms and body weight in a non-clinical setting. The specific aims of this research were (*a*) to identify distinct trajectories of depressive symptoms among older adults; and (*b*) to examine the association between depression trajectory patterns with subsequent obesity, metabolic indexes and cortisol levels.

Method

Subjects

The study subjects were participants in a prospective follow-up of the Survey of Health and Living Status of the Elderly (SHLSE) in Taiwan, which was established in 1989 with a nationally representative sample of 4049 persons aged $\geqslant$60 years with a response rate of 92% (Hermalin *et al*. 1989). Three follow-up in-person interviews were conducted in 1993, 1996 and 1999, respectively. The mortality rates of participants were 15% (n=590) in 1993, 14% (n=471) in 1996 and 14% (n=426) in 1999 (Glei *et al*. 2005). At each wave, efforts were made to reduce the attrition due to loss of follow-up, with a response rate of 91% (n=3155) in 1993, 89% (n=2669) in 1996 and 90% (n=2310) in 1999 among the survivors from the original 1989 cohort.

In 2000, a national representative sample (n=1713) was randomly selected for the Social Environment and Biomarkers of Aging Study (SEBAS; Goldman *et al*. 2003), including a subsample (n=840) from the SHLSE's 1989 cohort with oversampling of those with ages $\geqslant$71 years or with residence in an urban area, in which 57 had died and 728 (93%) out of 783 survivors completed the interview. Among the 1713 cohort members selected for the SEBAS, 1497 (92% among survivors) were interviewed at their homes and, from these, a subset of 1023 (68% among those interviewed) underwent physical examinations in a nearby hospital, with items covering anthropometric measures and blood and urine specimens. Comparing those interviewed for the SEBAS (n=1497) with those who underwent the physical examination (n=1023), no significant differences were found in gender, socioeconomic status and average score of self-assessed health. A total of 445 subjects ascertained from the 1989 SHLEE cohort completed the physical examination and were included in the present study. The study protocol was approved by the institutional review boards of the National Institute of Family Planning-Taiwan, Princeton University, Georgetown University, and the University of California-Los Angeles. Information obtained from the participants in the longitudinal surveys (i.e. 1989, 1993, 1996 and 1999 survey) and the SEBAS (i.e. 2000 survey) was used for this study.

Measures

Center for Epidemiologic Studies Depression Scale (*CES-D*)

Depressive symptoms were measured using a 10-item short-form of the CES-D, which is a modified version of the original 20-item version (Radloff, 1977). The reliability and factor structures are similar between the short version and the original one (Kohout *et al*. 1993). The Chinese version of the 10-item CES-D has a score range of 0 to 30 and has been shown to perform well in Chinese older adults (Krause & Liang, 1992; Boey, 1999). Cronbach's α estimates for the CES-D index were 0.76 and 0.79 in 1989 and 1993, respectively (Weinstein *et al*. 2004). The subjects completed the CES-D according to their depressive symptoms in the past week on a four-point scale (ranging from 0 'no' to 3 'often or always'). The symptom score was the sum of the total 10 items and was treated as a continuous

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variable at each time point (1989, 1993, 1996 and 1999). Participants with more than two items missing on the CES-D were excluded from the analyses, with 157 (4%) in 1989, 190 (5%) in 1993, 263 (6%) in 1996 and 236 (6%) in 1999. Subjects with a CES-D score [mean age = 67.5 (S.D. = 6.1) years] were younger than those without a CES-D score [mean age = 71.4 (S.D. = 7.4) years], but were not different in their gender distribution ($p > 0.05$). For the participants with only one CES-D item missing, the item was substituted by the mean of their gender- and age-specific subgroup, which is a common practice to compensate for attrition within the scale. The number of subjects with mean substitution was 27 (0.7%) in 1989, 16 (0.4%) in 1993, 17 (0.4%) in 1996 and 15 (0.4%) in 1999. Among the 4049 subjects recruited, the number of subjects with CES-D scores in each wave and the corresponding distributions are listed in Table 1. A total of 3922 had at least one CES-D score (including those with mean substitution) out of four times of assessment, in which 1766 had CES-D scores at four time points, 740 at three time points, 637 at two time points and 779 at only one time point.

Demographic characteristics and health status

In addition to the CES-D, each participant was interviewed with the use of a structured questionnaire containing other questions on demographic features (age, gender and education attainment), ever use of tobacco or alcohol, exercise, history of chronic diseases, self-rated health status and cognitive impairment, as shown in Table 1. Age was originally assessed as a continuous variable and was trichotomized into 60–69, 70–79, and ⩾80 years for subsequent analyses. The number of reported chronic diseases was counted on the history of hypertension, heart disease, stroke, asthma, arthritis, peptic ulcer, diabetes mellitus or liver disease. Self-assessed health, which has been shown to be a valuable predictor for physical health outcomes (Idler & Benyamini, 1997), was rated using a five-point scale and was regrouped into three categories, good (5 or 4), average (3) and not good (2 or 1), for subsequent analyses. Cognitive impairment was assessed using five questions from the Short Portable Mental Status Questionnaires in 1993, including current address, age in years, the date (month, day, and year), the weekday, and subtraction of the number three from twenty in four consecutive times (Glei *et al*. 2005).

Body weight and height

Self-reported data on body weight and height were collected in 1989, 1993 and 1999, but not in 1996, whereas for the participants of the SEBAS in 2000 their body weight and height were measured in hospital. Body mass index (BMI) was then calculated as the weight in kilograms divided by the height in metres

Table 1. *Baseline characteristics of the participants from the SHLSE, who had at least one CES-D score in any wave and were included for a depression trajectory analysis* ($n = 3922$)

Variables	*n* (%)	Mean (S.D.)
Characteristics in 1989		
Male	2241 (57.1)	
Education		
Illiterate	1592 (40.6)	
Uneducated but literate	345 (8.8)	
Primary	1226 (31.3)	
Secondary or above	748 (19.1)	
Missing	11 (0.3)	
Ever use of tobacco	1374 (35.0)	
Missing	1 (0.03)	
Ever use of alcohol	843 (21.5)	
Exercise in the past 6 months		
Never	2751 (70.1)	
Seldom	299 (7.6)	
Sometimes	247 (6.3)	
Often	624 (15.9)	
Missing	1 (0.03)	
Number of chronic disease		
0	1090 (27.8)	
1	1256 (32.0)	
2–3	1257 (32.1)	
4 and above	258 (6.6)	
Missing	61 (1.6)	
Self-assessed health status		
Good	1526 (38.9)	
Average	1492 (38.0)	
Not good	878 (22.4)	
Missing	26 (0.7)	
Cognitive impairment in 1993		
0	1897 (48.4)	
1	517 (13.2)	
2	291 (7.4)	
3–5	253 (6.5)	
Missing	964 (24.6)	
Characteristics in different years		
Age in 1989, years	3922	67.9 (6.3)
10-item CES-D score in 1989	3892	6.6 (5.3)
10-item CES-D score in 1993	2964	6.8 (6.0)
10-item CES-D score in 1996	2406	6.6 (6.5)
10-item CES-D score in 1999	2704	6.4 (6.6)
Reported BMI in 1989, kg/m²	2177	22.9 (3.6)
Reported BMI in 1993, kg/m²	1771	22.9 (3.6)
Reported BMI in 1999, kg/m²	1998	23.0 (3.4)
Measured BMI in 2000, kg/m²	445	23.8 (3.7)

SHLSE, Survey of Health and Living Status of the Elderly; CES-D, Center for Epidemiologic Studies Depression Scale; S.D., standard deviation; BMI, body mass index.

https://doi.org/10.1017/S0033291710002473 Published online by Cambridge University Press

squared (kg/m²). Subjects were initially categorized into five BMI groups using the World Health Organization guideline for Asians (2000): underweight (<18.5 kg/m²), normal weight (18.5 to 22.9 kg/m²), overweight (23 to 24.9 kg/m²), obesity I (25 to 29.9 kg/m²) and obesity II (≥30.0 kg/m²). Because the number of participants belonging to obesity II was small (2.9% of subjects), it was combined with obesity I as a single category of obesity for subsequent analyses. The number of subjects with information on BMI and their distributions are listed in Table 1. Subjects without BMI data were older, had higher depression scores, and were more likely to be female than those with BMI data in each wave except for gender ($p=0.1$) and depression score ($p=0.1$) in the wave of 1999.

Anthropometric measures, and blood and urine specimens

For subjects included in the SEBAS in 2000, their blood pressure, blood specimens and urine specimens were collected. After subjects had arrived at the hospital for at least 20 min, three blood pressure indexes were measured using a mercury sphygmomanometer in a seated position. The second and third measurements were averaged for data analysis. After an overnight fast, fasting blood samples were drawn for the measurement of fasting blood glucose levels and cholesterol, high-density lipoprotein (HDL)-cholesterol and triglycerides. Fasting glucose was measured using the Beckman glucose oxidase method, with a sensitivity of 3 mg/dl and a coefficient of variation (CV) of 1.6%. Levels of total cholesterol and triglycerides were measured using a Beckman CX7 (sensitivity = 5 mg/dl and CV = 1.3% for total cholesterol; sensitivity = 10 mg/dl and CV = 1.9% for triglycerides), while levels of HDL-cholesterol were determined using a Ciba Corning Express 560 (Siemens Medical Solutions, Germany; sensitivity = 7 mg/dl and CV = 4.7%). Cortisol levels were measured using a 12-h overnight urine sample and assayed using high-performance liquid chromatography with ultraviolet absorbance detection and a lower detection limit of 4 μg/l and inter-assay CV of 10.8% (Goldman *et al*. 2005).

Statistical analysis

We started by determining groups of depressive symptoms over the follow-up period by means of trajectory analyses, using semi-parametric group-based modelling (Nagin & Tremblay, 2001) as implemented in PROC TRAJ of SAS software (Jones *et al*. 2001), which optimally uses the available data by allowing for missing data at different time points. The model with the smallest absolute Bayesian information criterion (BIC) value was selected as the best-fitting one. Subjects were classified into different depression trajectory classes based on the highest posterior probabilities of belonging to each group. Trajectory group membership for each subject was then used for subsequent analyses.

Initial trajectory analyses included those subjects with at least three waves of data ($n=2506$) and then repeated for all subjects ($n=3922$). Since the trajectory structures were remarkably similar (κ coefficient = 0.98, $p<0.001$) in identifying four distinctive classes of depressive symptoms over the 10-year span, only the results of the latter one were used for further analyses.

We then tested whether baseline characteristics were associated with the depression trajectories using multinomial logistic regression analysis. Similar methods were used to determine the relationships of depression trajectories to reported BMI categories in 1989, 1993 and 1999 as well as measured BMI in 2000 with adjustment for potential covariates. Potential confounding variables were identified based on association estimates linking with depression trajectories or reported BMI. In examining the relationships between depression trajectories and biomarkers, analysis of covariance and the trend test were conducted with adjustment for covariates. The effect size was calculated as the standardized difference between two means divided by the pooled standard deviation of each group (Cohen, 1987).

To evaluate the influence of missing information on reported BMI (Allison, 2000), we imputed the missing value for those who had CES-D but lacked BMI data in 1989, 1993 and 1999, respectively, according to an individual's gender, age and group membership of depression trajectory using SAS PROC MI. We imputed five different datasets and analysed each dataset using SAS PROC LOGISTIC to generate the multinomial logistic regression parameter estimates and associated standard errors. The final estimates from the multiple imputations were based on the combined results using SAS PROC MIANALYZE (Yuan, 2000). All analyses were conducted using SAS software, version 9.1.3 (SAS Institute Inc., USA).

Results

Trajectory classes of depression

A cubic four-class growth mixture model (Fig. 1), i.e. having four depression trajectory classes, was selected as the best-fitting model since it had the lowest BIC (BIC = −32641.49), as compared with a quadratic four-class growth model (BIC = −32647.64). The average posterior probability ranged from 0.68 (S.E. = 0.16) to 0.79 (S.E. = 0.18), indicating adequate model fitting for

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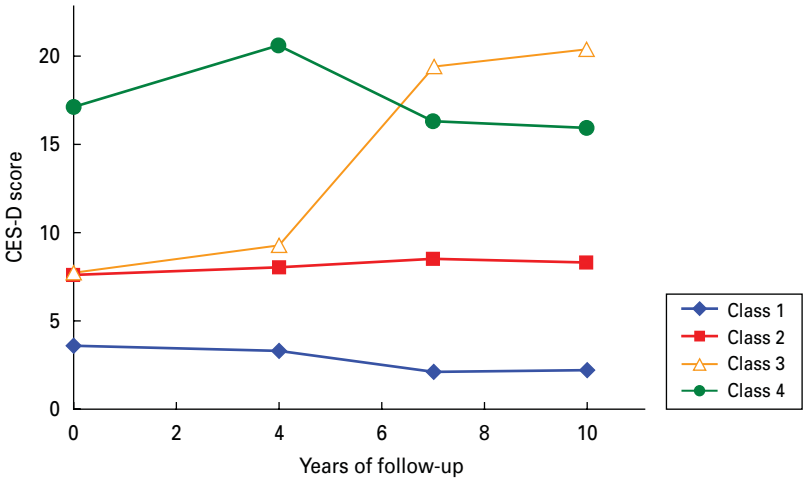

Fig. 1. Four classes of depression trajectories among Chinese elderly ($n = 3922$), 1989–1999, measured using the Center for Epidemiologic Studies Depression (CES-D) Scale. The proportion of subjects in each class was as follows: class 1 'persistent low' (41.8%); class 2 'persistent mild' (46.8%); class 3 'late peak' (4.2%); and class 4 'high-chronic' (7.2%).

the cubic four-class structure according to a criterion of average posterior probability being $\geqslant 0.70$ (Cote *et al*. 2002). On the basis of the pattern in depression scores across the four waves, these four classes were named as persistent low (class 1), persistent mild (class 2), late peak (class 3) and high-chronic (class 4), respectively.

The class of persistent low (class 1) included 1638 (41.8% of 3922) subjects who were characterized by low depression scores at waves 1 and 2 (mean score about 3) and even lower ones at waves 3 and 4 (mean score about 2). Similarly, the class of persistent mild (class 2), including 1836 (46.8%) subjects, started with a moderate level of depression scores (mean score about 7.6 at wave 1) and stayed relatively flat throughout the follow-up (mean score = 8.3 at wave 4). In contrast, the class of late peak (class 3), including 165 (4.2%) subjects, started with a moderate level of depression scores (mean score about 7.8 at wave 1) but increased rapidly after wave 2 and peaked at wave 4 (mean score = 20.4). For the class of high-chronic (class 4), including 283 (7.2%) subjects, they started with the highest depression score (score of 17.1) at baseline among the four classes. Their depressive symptoms peaked at wave 2 (mean score = 20.6), and then gradually decreased but remained consistently high (mean score = 15.9 at wave 4).

Baseline characteristics and depression trajectories

Table 2 shows the baseline characteristics for the four trajectory classes and how these were related to trajectory class in the multinomial logistic regression analysis using 'persistent low' as the reference category. The average age at baseline was 67.2 (S.D. = 6.0) years for class 1, 68.4 (S.D. = 6.5) years for class 2, 67.3 (S.D. = 5.2) years for class 3, and 69.1 (S.D. = 6.7) years for class 4. The results of univariate analyses showed that older age, female gender, being illiterate, tobacco smoking, no alcohol consumption, no regular exercise, presence of chronic disease and poor self-assessed health status were significantly associated with higher levels of depressive symptoms, whereas the severity of cognitive impairment was not. The significance level of crude odds ratios is denoted in the corresponding column of *n* (%) in Table 2, if significant. In the multivariable analysis containing all the variables listed in Table 2, the adjusted odds ratios (aORs) of these variables remained significant for depression trajectory classes 2, 3 and 4, except for age and tobacco smoking (all the associations becoming non-significant). The association estimates were generally greater for classes 3 and 4 than those for the other trajectory classes. For instance, with all the listed covariates adjusted, elders who reported regular exercise had 18% reduced risk to become persistent mild depressive, and the corresponding estimate was as great as 79% for becoming high-chronic depressive.

Depression trajectories and BMI-based obesity status

Before the relationship between depression trajectories and obesity was examined, potential confounders were evaluated by means of examining baseline characteristics in relation to BMI-based obesity status at the latest wave of the SHLSE in 1999 among subjects in trajectory class 1 (i.e. individuals without depression). The results revealed that older age, secondary or above education, tobacco smoking and less alcohol consumption were associated with underweight, whereas female gender, higher education and

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Table 2. *Relationships of baseline characteristics with depression trajectories in the multinomial logistic regression analysis: the 1989–1999 SHLSE (n = 3922)*

	Class 1 (*n* = 1638): persistent low	Class 2 (*n* = 1836): persistent mild		Class 3 (*n* = 165): late peak		Class 4 (*n* = 283): high-chronic	
Variables	*n* (%)	*n*(%)[a]	aOR (95% CI)[b]	*n* (%)[a]	aOR (95% CI)[b]	*n* (%)[a]	aOR (95% CI)[b]
Age, years							
60–69	1174 (71.7)	1143 (62.3)	1.00	111 (67.3)	1.00	169 (59.7)	1.00
70–79	397 (24.2)	573 (31.2)***	1.26 (1.1–1.5)**	50 (30.3)	1.12 (0.7–1.7)	89 (31.1)**	1.10 (0.8–1.6)
⩾80	67 (4.1)	120 (6.5)***	1.40 (0.9–2.1)	4 (2.4)	0.18 (0.02–1.3)	25 (8.8)***	1.39 (0.7–2.7)
Gender							
Male	1117 (68.2)	953 (51.9)	1.00	71 (43.0)	1.00	100 (28.5)	1.00
Female	521 (31.8)	883 (48.1)***	1.41 (1.1–1.8)**	94 (57.0)***	2.40 (1.4–4.2)**	183 (64.7)***	1.56 (1.0–2.4)
Education							
Illiterate	484 (29.6)	854 (46.5)	1.00	83 (50.3)	1.00	171 (60.4)	1.00
Uneducated but literate	135 (8.2)	168 (9.2)*	0.81 (0.6–1.1)	22 (13.3)	0.92 (0.5–1.8)	20 (7.1)**	0.48 (0.2–0.9)*
Primary	596 (36.4)	524 (28.5)***	0.65 (0.5–0.8)***	41 (24.9)***	0.66 (0.4–1.1)	65 (23.0)***	0.54 (0.3–0.8)***
Secondary or above	417 (25.5)	287 (15.7)***	0.67 (0.5–0.9)***	18 (10.9)***	0.56 (0.3–1.1)	26 (9.2)***	0.57 (0.3–1.0)
Tobacco smoking							
No	981 (59.9)	1239 (67.5)	1.00	119 (72.1)	1.00	208 (73.5)	1.00
Yes	656 (40.0)	597 (32.5)***	1.03 (0.8–1.3)	46 (27.9)**	1.28 (0.7–2.2)	75 (26.5)***	1.04 (0.7–1.6)
Alcohol consumption							
No	1204 (73.5)	1475 (80.3)	1.00	140 (84.9)	1.00	260 (91.9)	1.00
Yes	434 (26.5)	361 (19.7)***	1.07 (0.9–1.3)	25 (15.2)***	0.91 (0.5–1.6)	23 (8.1)***	0.43 (0.3–0.8)*
Exercise							
Never	1048 (64.0)	1344 (73.2)	1.00	119 (72.1)	1.00	240 (84.8)	1.00
Seldom	136 (8.3)	135 (7.4)*	1.09 (0.8–1.5)	10 (6.1)	0.89 (0.4–2.0)	18 (6.4)*	0.98 (0.5–1.9)
Sometimes	110 (6.7)	108 (5.9)	1.13 (0.8–1.6)	12 (7.3)	1.57 (0.8–3.2)	17 (6.0)	0.97 (0.5–2.0)
Often	344 (21.0)	249 (13.6)***	0.82 (0.7–1.0)	24 (14.6)*	1.17 (0.7–2.0)	7 (2.5)***	0.21 (0.1–0.5)***
No. of chronic diseases							
0	586 (35.8)	439 (23.9)	1.00	28 (17.0)	1.00	37 (13.1)	1.00
1	573 (35.0)	553 (39.1)***	1.11 (0.9–1.4)	62 (37.6)***	1.79 (1.1–3.2)**	68 (24.0)***	1.17 (0.7–2.0)
2–3	415 (25.3)	670 (36.5)***	1.49 (1.2–1.9)***	54 (32.7)***	1.92 (1.1–3.5)*	118 (41.7)***	1.46 (0.9–2.4)
4 and above	42 (2.6)	141 (7.7)***	2.67 (1.7–4.3)***	19 (11.5)***	4.86 (2.1–11.4)***	56 (19.8)***	4.50 (2.3–8.9)***

Self-assessed health status							
Good	874 (53.4)	596 (32.5)***	0.73 (0.6–0.8)***	39 (23.6)***	0.44 (0.3–0.7)***	17 (6.0)***	0.30 (0.2–0.6)***
Average	604 (36.9)	740 (40.3)	1.00	76 (46.1)	1.00	72 (25.4)	1.00
Not good	155 (9.5)	483 (26.3)***	2.30 (1.8–3.0)***	48 (29.1)***	1.72 (1.0–2.9)	192 (67.8)***	7.92 (5.3–11.8)***
Cognitive impairment							
0	791 (48.3)	892 (48.6)	1.00	84 (50.9)	1.00	130 (45.9)	1.00
1	208 (12.7)	253 (13.8)	1.00 (0.8–1.3)	14 (8.5)	0.62 (0.3–1.1)	42 (14.8)	1.14 (0.7–1.7)
2	118 (7.2)	141 (7.7)	0.98 (0.7–1.3)	14 (8.5)	0.98 (0.5–1.9)	18 (6.4)	0.70 (0.4–1.3)
3–5	97 (5.9)	124 (6.8)	1.05 (0.8–1.4)	9 (5.5)	0.86 (0.4–1.8)	23 (8.1)	1.20 (0.7–2.0)

SHLSE, Survey of Health and Living Status of the Elderly; aOR, adjusted odds ratio; CI, confidence interval.

[a] The significance level of a univariate odds ratio is denoted in this column if significant.

[b] aORs and their 95% CIs were obtained with statistical adjustment for all the variables listed in this table.

* $p<0.05$, ** $p<0.01$, *** $p<0.001$.

presence of chronic disease were associated with either overweight or obesity (see Supplementary Table, online). These analyses indicated that, with the exception of regular exercise, self-assessed health status and cognitive impairment, all the other variables listed in Table 2 were considered as potential confounders in subsequent analyses given that they were associated with both depression trajectories and BMI-based obesity status. However, we also included exercise and cognitive impairment in our analyses since these were reported as potential confounders in previous studies.

Table 3 shows the results of multivariable multinomial logistical regression analyses of BMI-based obesity status on depression trajectories with controlling for age, gender, level of education, use of alcohol, smoking, presence of chronic disease, exercise and cognitive impairment. For the analysis with complete cases, the subjects of depression trajectory class 2 (aOR 0.79) in 1989, and class 2 (aOR 0.67) or 4 (aOR 0.30) in 1999 were less likely to be obese compared with those in class 1. Meanwhile, the subjects in classes 2 and 4 in 1989, classes 2, 3 and 4 in 1993, class 3 in 1999, as well as class 2 in 2000 were more likely to be underweight. When individuals with missing information on BMI were taken into account by the multiple imputation analysis, the magnitude of aORs remained similar despite the variation in the level of significance.

Depression trajectories and metabolic functions and cortisol levels

To further explore the possible mechanisms underlying the associations observed between depression trajectories and BMI-based obesity status, we examined the possible roles of metabolic functions as well as levels of cortisol that might be played in the relationship. The analyses were limited to the 445 subjects who were part of the original SHLSE cohort in 1989 and were later selected for the SEBAS in 2000 to undergo physical examination and blood drawing. As expected, the measured BMI in this subsample was correlated with levels of cholesterol ($r=0.17$), HDL ($r=-0.18$), triglycerides ($r=0.24$), fasting blood glucose ($r=0.17$), systolic blood pressure ($r=0.14$) and diastolic blood pressure ($r=0.15$), but not with that of cortisol ($r=-0.01$). Their membership in the depression trajectory class was 229 (51.5%) in class 1, 184 (41.3%) in class 2, 15 (3.3%) in class 3 and 17 (3.8%) in class 4, respectively. Since classes 3 and 4 had small number of subjects, they were combined for the trend analyses on metabolic functions and cortisol levels by depression trajectories (Table 4). The classes of older adults with more and persistent depressive symptoms showed a trend toward having both a lower BMI

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Table 3. *Relationships of depression trajectories to BMI-based obesity status in the multinomial logistic regression analysis: the 1989–1999 SHLSE and 2000 SEBAS*

	Underweight (BMI <18.5 kg/m²)		Overweight (BMI 23.0–24.9 kg/m²)		Obese (BMI $\geqslant 25.0$ kg/m²)	
Source of BMI and depression trajectories	Complete case analysis aOR[a] (95% CI)	Multiple imputation analysis aOR (95% CI)	Complete case analysis aOR (95% CI)	Multiple imputation analysis aOR (95% CI)	Complete case analysis aOR (95% CI)	Multiple imputation analysis aOR (95% CI)
Reported BMI in 1989	$n=187$	$n=372$	$n=485$	$n=836$	$n=525$	$n=1022$
Class 1 (persistent low)	1.00	1.00	1.00	1.00	1.00	1.00
Class 2 (persistent mild)	1.60 (1.1–2.3)**	1.50 (1.0–2.3)	0.83 (0.7–1.1)	0.89 (0.7–1.1)	0.79 (0.6–0.99)*	0.91 (0.7–1.2)
Class 3 (late peak)	1.43 (0.6–3.3)	1.23 (0.5–3.0)	0.88 (0.5–1.6)	0.90 (0.5–1.7)	0.83 (0.5–1.5)	0.81 (0.5–1.4)
Class 4 (high chronic)	2.39 (1.2–4.6)**	1.92 (0.9–4.3)	0.42 (0.2–0.8)	0.73 (0.4–1.5)	0.64 (0.4–1.1)	0.78 (0.4–1.4)
Reported BMI in 1993	$n=140$	$n=276$	$n=428$	$n=684$	$n=395$	$n=744$
Class 1 (persistent low)	1.00	1.00	1.00	1.00	1.00	1.00
Class 2 (persistent mild)	1.70 (1.1–2.6)*	1.42 (0.9–2.2)	0.90 (0.7–1.2)	0.88 (0.7–1.2)	0.79 (0.6–1.0)	0.85 (0.7–1.1)
Class 3 (late peak)	2.54 (1.2–5.4)*	1.77 (0.8–3.7)	1.00 (0.6–1.8)	1.05 (0.6–2.0)	0.77 (0.4–1.4)	0.74 (0.3–2.0)
Class 4 (high chronic)	2.43 (1.1–5.2)*	1.86 (0.8–4.1)	0.86 (0.5–1.6)	0.83 (0.5–1.5)	0.65 (0.3–1.3)	0.62 (0.3–1.2)
Reported BMI in 1999	$n=181$	$n=187$	$n=470$	$n=489$	$n=497$	$n=517$
Class 1 (persistent low)	1.00	1.00	1.00	1.00	1.00	1.00
Class 2 (persistent mild)	1.40 (1.0–2.0)	1.25 (0.8–1.9)	0.80 (0.6–1.0)	0.86 (0.6–1.1)	0.67 (0.5–0.9)**	0.72 (0.5–0.9)*
Class 3 (late peak)	2.09 (1.1–4.0)*	1.52 (0.7–3.3)	0.68 (0.4–1.2)	0.57 (0.3–1.1)	0.70 (0.4–1.2)	0.70 (0.4–1.3)
Class 4 (high chronic)	1.41 (0.7–3.0)	1.20 (0.5–2.9)	0.81 (0.5–1.4)	0.85 (0.4–1.6)	0.30 (0.2–0.6)***	0.35 (0.2–0.7)**
Measured BMI in 2000	$n=29$	–	$n=110$	–	$n=149$	–
Class 1 (persistent low)	1.00	–	1.00	–	1.00	–
Class 2 (persistent mild)	2.81 (1.1–7.1)*	–	0.59 (0.3–1.0)	–	0.67 (0.5–1.1)	–
Classes 3 and 4 (late peak/ high chronic)	0.46 (0.1–4.1)	–	0.33 (0.1–0.97)*	–	0.47 (0.2–1.2)	–

BMI, Body mass index; SHLSE, Survey of Health and Living Status of the Elderly; SEBAS, Social Environment and Biomarkers of Aging Study; aOR, adjusted odds ratio; CI, confidence interval.

[a] aORs were calculated using those with normal weight (BMI = 18.5–22.9 kg/m²) as the referent and with adjustment for age, gender, education, alcohol drinking, smoking, exercise, cognitive impairment and chronic disease at baseline.

* $p<0.05$, ** $p<0.01$, *** $p<0.001$.

Table 4. *BMI, metabolic functions and cortisol levels by depression trajectories class: the 1989–1999 SHLSE and 2000 SEBAS (n = 445)*

	Class 1: persistent low (*n* = 229)	Class 2: persistent mild (*n* = 184)		Classes 3 and 4: late peak/high-chronic (*n* = 32)		Group comparison by ANCOVA[b]		
Measures in 2000	Mean (S.D.)	Mean (S.D.)	ES[a]	Mean (S.D.)	ES[a]	*F* (df)	*p*	Trend test[c]: *p*
BMI, kg/m²	24.18 (3.46)	23.46 (3.89)	0.20	23.53 (3.86)	0.18	3.91 (2, 442)	0.02	0.01
Cholesterol, mg/dl	198.17 (38.04)	200.20 (42.23)	0.05	208.06 (60.86)	0.19	0.60 (2, 441)	0.55	0.80
HDL-cholesterol, mg/dl	48.52 (12.72)	50.57 (15.33)	0.16	48.56 (12.47)	0.003	0.83 (2, 441)	0.44	0.56
Triglycerides, mg/dl	116.93 (74.96)	119.81 (91.65)	0.03	121.59 (80.31)	0.06	0.05 (2, 441)	0.95	0.78
Glucose, mg/dl	105.29 (27.94)	105.08 (29.51)	0.001	106.25 (43.20)	0.03	0.33 (2, 441)	0.72	0.43
Systolic BP, mmHg	142.84 (19.13)	141.64 (22.12)	0.06	144.80 (21.06)	0.09	0.46 (2, 442)	0.63	0.75
Diastolic BP, mmHg	80.33 (10.20)	80.21 (10.79)	0.01	81.42 (7.38)	0.12	0.36 (2, 442)	0.72	0.66
Cortisol, μg/l	18.54 (21.22)	23.83 (52.38)	0.13	36.03 (66.77)	0.35	2.21 (2, 435)	0.11	0.04

BMI, Body mass index; SHLSE, Survey of Health and Living Status of the Elderly; SEBAS, Social Environment and Biomarkers of Aging Study; ANCOVA, analysis of covariance; S.D., standard deviation; ES, effect size; df, degrees of freedom; HDL, high-density lipoprotein; BP, blood pressure.

[a] ES was derived from comparison with the persistent low group. [b] All analyses were adjusted for age, gender, smoking, alcohol consumption and chronic disease at baseline. [c] Linear regression conducted by means of recoding class 1 as 0, class 2 as 1, and classes 3 and 4 as 2.

($p = 0.01$) and a higher cortisol level ($p = 0.04$) compared with those with low depressive symptoms. In contrast, no significant associations were found between trajectory classes and blood glucose, blood pressure and lipid profiles.

Discussion

By prospectively examining the long-term course of depressive symptoms over a 10-year span in a nationally representative sample of community-dwelling older adults in Taiwan, we identified four distinctive trajectory classes of depressive symptoms. As compared with those with persistent-low depressive symptoms, the odds of obesity were lower, while the odds of underweight were higher in the elderly with persistent mild, late peak or high-chronic depressive symptoms. Furthermore, after adjustment for potential confounders, long-term levels of depressive symptoms were positively associated with cortisol levels.

Although we did not explicitly dichotomize individuals' depressive symptoms in our analyses, comparability in terms of the prevalence of depression can be made by adopting a cut-off score suggested in a previous cross-validation study (i.e. $\geqslant 10$ on the 10-item CES-D; Boey, 1999). Since the mean scores of classes 3 and 4 were above 10 at least some time during the four-wave follow-ups, the prevalence of depression in this study could be estimated as 11.4% (i.e. 4.2% for class 3 and 7.2% for class 4). This is not much different from the estimates derived from a study in Hong-Kong: 11.0% for males and 14.5% for females (Chi *et al*. 2005). By using the trajectories analysis, we demonstrated heterogeneity in the depressive symptoms and their changes over time in older adults, which is consistent with previous longitudinal studies in Western populations (Beekman *et al*. 2002; Mojtabai & Olfson, 2004).

The baseline correlates for depression were similar across trajectory classes 2, 3 and 4, indicating the stability of these features in predicting higher levels of depression. Among these correlates, many are quite congruous with the profile of risk and protective factors reported in Western elderly, including older age, female gender, lower educational attainment, chronic disease, regular exercise and poor self-assessed health (Djernes, 2006). In particular, regular exercise, as a modifiable factor, has not only been consistently associated with reduced risk for subsequent depression in older adults (Strawbridge *et al*. 2002) but also been promoted to have many other health benefits as well (Nelson *et al*. 2007). However, unlike the association of alcohol consumption with depression found in Western populations (Graham & Schmidt, 1999; Perreira & Sloan, 2002), the older adults with alcohol consumption experiences in this study had lower odds of being in the high chronic depression trajectory. One possibility is that, given alcoholic beverages are commonly taken when one is in social settings rather than being solitary, those who drank alcohol may represent a subgroup of elderly who either had a close crowd to hang around (e.g. social network) or engaged in social or leisure

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activities; both have been related to reduced mortality and more preserved cognitive functions (Cornman *et al*. 2003; Glei *et al*. 2005). Further investigation on the possible role of alcohol drinking on older adults' depressive symptoms is warranted.

Our finding that trajectory classes of more or persistent depressive symptoms were associated with lower odds of being obese and higher odds of being underweight does not support prior research conducted in Western populations (Luppino *et al*. 2010), yet is congruent with existing evidence in Chinese (Li *et al*. 2004; Ho *et al*. 2008) and Japanese populations (Kuriyama *et al*. 2006). Intriguingly, this association pattern echoes with the so-called 'jolly fat' observation, suggesting that obese people are less anxious and less depressed than the rest of the population (Crisp & McGuiness, 1976).

Although the mechanisms for the inverse associations between depression and obesity remain to be further explored, some explanations may be suggested. First, previous studies on community-dwelling older adults showed that a decrease in body weight is common in older people (Thompson Martin *et al*. 2006). Depression, often associated with poor appetite, can lead to weight loss (Forman-Hoffman *et al*. 2007) and become a common cause of low body weight in the elderly (Chapman, 2007). Second, the Chinese idiom of 'happy mind and fat body' – literally meaning that people who are less worried are prone to have a larger body weight – reflects a traditional Chinese viewpoint on the relationship between affect and body weight (Li *et al*. 2004). Hence, older adults with obesity might not face negative pressure as younger people do. Furthermore, gaining weight during late life in Chinese culture also indicates a good fortune for the elderly since more food eating is affordable only for wealthy people.

The link between long-term depressive symptoms and less obesity in older adults, as suggested in this study, might be accounted for in part by the dysregulation of the hypothalamic–pituitary–adrenal axis that was characterized by high cortisol levels. The elevated cortisol levels may reflect the host's response to stress mediated by some shared biological factors between depression and obesity (Bornstein *et al*. 2006). A recent longitudinal study on European elderly also showed that depression was associated with both low and high levels of cortisol (Bremmer *et al*. 2007). Nevertheless, we did not find that measures of nutritional markers and blood pressure differed significantly among the depression trajectories, suggesting the possibility that heterogeneous pathways may exist in the link between long-term depression and body weight in elderly populations (Weber-Hamann *et al*. 2002; McCaffery *et al*. 2003).

Several limitations of our study need to be considered when interpreting the findings. First, a large number of subjects (1487 out of 4049 originally recruited individuals) died during the follow-up, which is a common limitation in a longitudinal study of older adults. Since subjects who survived into later life were likely to have fewer health complications than those that died, this might weaken the link between depression and obesity as a result of healthy selection. Second, there was considerable number of older adults who failed to provide self-reported information on BMI. Nevertheless, no appreciable differences were found between the results of complete case analyses and multiple imputation analyses. Third, the information on BMI was based on self-report for the majority of subjects. Nevertheless, the correlation between reported BMI and measured BMI in a subsample was high ($r = 0.88$). Moreover, despite two different methods of collected BMI adopted in this study, the results of inverse association between depression and obesity were similar (see Table 3), suggesting that the effects of collection method on the observed association may be reasonably limited.

In conclusion, this study identified four distinctive trajectories of depressive symptoms over time in community-dwelling older adults in Taiwan. Baseline sociodemographic and life-style attributes, such as gender, educational level, regular exercise, chronic disease and self-assessed health, were associated with depressive trajectory pattern. The results further indicated that older adults with incremental increases in depressive symptoms over time experienced lower odds of being obese, but had higher cortisol levels. These findings may help shed light on the relationship between long-term profiles in depressive symptoms and obesity in later life.

Note

Supplementary material accompanies this paper on the Journal's website (http://journals.cambridge.org/psm).

Acknowledgements

The study was supported by the Taiwan National Science Council (grants NSC 93-2320-B-002-069 and NSC 94-2312-B-002-099). We thank those who contributed to the Survey of Health and Living Status of the Near Elderly and Elderly in Taiwan, including staff in the Population and Health Research Center, Bureau of Health Promotion, and all interviewers, and Mr Po-Chang Hsiao for preparing Fig. 1.

Declaration of Interest

None.

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