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Depressive symptoms predict incident chronic disease burden 10 years later: Findings from the English Longitudinal Study of Ageing (ELSA)

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ABSTRACT

Objective: To assess the association between depressive symptoms and incident chronic illness burden in prospective longitudinal analyses.

Methods: We analysed data from 2472 participants (62.88 ± 8.49 years old; 50.8% female) from the English Longitudinal Study of Ageing (ELSA). Depressive symptoms were measured using the Centre for Epidemiological Studies Depression (CES-D) scale at baseline in 2004, and participants were followed up for 10 years. Participants with prevalent illness at baseline (coronary heart disease [CHD], other cardiac illness, stroke, cancer, diabetes/high blood glucose, arthritis, lung disease, osteoporosis and Parkinson's disease) were excluded from models predicting illness burden (the sum of illnesses reported) over follow-up. Linear regression was used controlling for a wide range of covariates.

Results: The mean chronic illness burden was 0.57, with 43.1% experiencing at least one incident physical illness. Baseline continuous CES-D score was a significant predictor of incident chronic illness burden up to 10 years later (incident rate ratio = 1.05, 95% confidence intervals = 0.05–0.21, $p = .003$), independent of sociodemographic, behavioural, cognitive and clinical covariates. Sensitivity analyses excluding participants who developed a chronic illness within the 2 years following baseline corroborated the main results.

Conclusion: Depressive symptoms were associated with greater incident chronic illness burden 10 years later. These findings have clinical implications for the treatment of depression in physically healthy older adults.

1. Introduction

Chronic physical illnesses are on the increase, and this is in part explained by an ageing population [1]. Furthermore, older adults often find themselves with comorbidities in addition to their initial diagnosis; the co-occurrence of cardio-respiratory and metabolic diseases being two notable examples of this [2]. In the UK, data from over 1.75 million patients from Scottish primary care practices revealed an overall multimorbidity prevalence of 23.2%, rising to 67.0% in those aged 65 years or older [3]. Data from the US suggest a similar prevalence [4]. While the consequences of multimorbidity are thought to include increased mortality, reduced quality of life and increased health care utilisation [5–8], the causes are yet to be fully elucidated.

Depression in physical illness is highly prevalent. The WHO World Health Survey [9] estimated the one year prevalence of a depressive episode in conjunction with diabetes to be 9.3% (95% CI 7.3–11.3), 10.7% for arthritis (95% CI 9.1–12.3), 15.0% for angina (95% CI 12.9–17.2), and 18.1% for asthma (95% CI 15.9–20.3). In comparison, the healthy control group had a one-year prevalence of a depressive episode at just 3.2% (95% CI 3.0–3.5). Moreover, these authors demonstrated a dose-response relationship, whereby those participants with two or more physical illnesses had even greater risk, with nearly a quarter (23.0%) also having co-morbid depression [9]. Smith and colleagues [10] conducted cross-sectional analyses of just over 140 thousand depressed participants and 128 thousand non-depressed controls recruited from primary care practices across Scotland and showed that those with depression were more likely to have a single physical illness and more likely to have multiple physical illnesses than controls. Similar results have also emerged using Australian data [11]. However, these cross-sectional analyses do not allow for the direction of the effect to be examined. UK data have also highlighted the increased prevalence of depressive symptoms and negative affect, not just clinical depression, among the physically ill [12], and it is not yet clear whether such symptoms that do not meet a diagnostic threshold also show the same relationship with physical illness burden.

The relationship between depression and physical illness is thought to be bidirectional [13] and several good quality reviews have been published showing that depression can lead to physical illness onset.

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For example, a meta-analysis of longitudinal studies showed depressed individuals to be at 60% greater risk of developing type II diabetes than those without depression [14]. Depression has also been implicated as a risk factor in coronary heart disease (CHD) [15], while mixed evidence exists for cancer [16, 17]. Therefore, it seems plausible that depressive symptoms could also be linked to incident chronic illness burden. Due to the higher prevalence of multimorbidities among the elderly and females [2] and that there is a social gradient to health [18], particularly cardiac disease [19], demographic factors such as age, sex and socioeconomic status may also be pertinent to understanding the relationship between depressive symptoms and physical illness burden. Moreover, evidence for the toxicity of somatic depressive symptoms for CHD patients also suggests that the type of depressive symptoms experienced warrants further investigation [20]. We aimed to test these hypotheses using prospective data from the English Longitudinal Study of Ageing (ELSA) [21]. A secondary aim was to explore the association between depressive symptoms and individual illnesses contributing to this combined effect.

2. Methods

2.1. Sample and study design

Data were taken from ELSA, a nationally representative general population study of adults aged 50 years and older living in England. Further details can be found elsewhere [21]. Briefly, ELSA participants were drawn from the Health Survey for England cohorts in 1998, 1999 and 2001, who were born prior to March 1952. The sample is followed up every two years from 2002 onwards, with wave 7 being the most recently completed phase of data collection. At every wave participants complete a computer-assisted personal interview plus a self-completion questionnaire. On alternate waves, a nurse visit is conducted to allow for the collection of blood samples and objective assessments of physical function, such as body mass index (BMI).

This current paper reports data spanning a decade, from wave 2 (2004/5) through to wave 7 (2014/2015), of initial core members who completed the nurse assessment, were free from certain physical illnesses at baseline, and who provided details of their physical health at at least one follow-up wave. The physical illnesses were selected based on the consistency of their measurement in subsequent ELSA waves and included CHD (comprising angina and myocardial infarction [MI]), other cardiac illnesses (comprising heart failure, cardiac arrhythmia and heart murmur), diabetes/high blood glucose, arthritis, osteoporosis, lung disease (e.g. chronic bronchitis and emphysema), cancer and Parkinson's disease. Analyses were performed on a total sample of 2472 participants out of a possible 8780 (see Fig. 1 for full details).

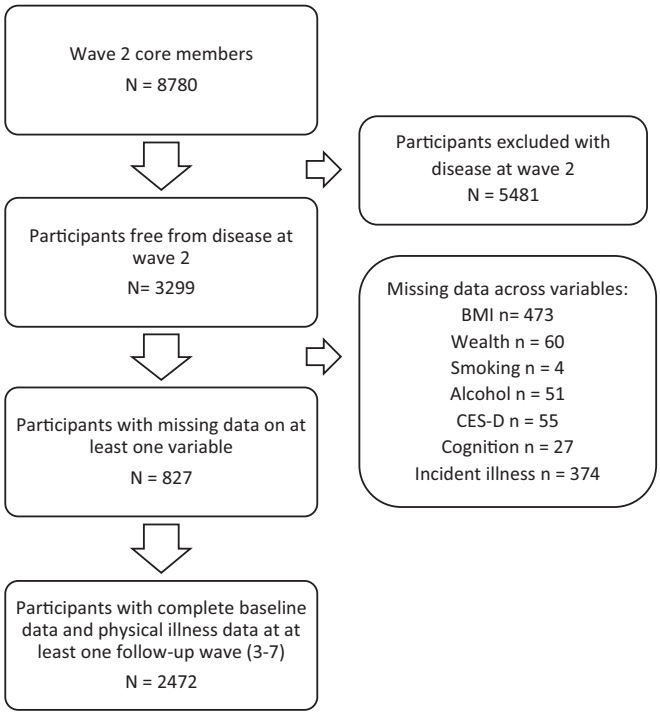

Fig. 1. Flow diagram of sample size.

2.2. Measures

2.2.1. Depressive symptoms

Depressive symptoms were measured at wave 2 using the eight-item Centre for Epidemiological Studies Depression scale (CES-D). The CES-D measures those symptoms that can be used to identify people at risk of depression, rather than clinical depression per se [22]. The psychometric properties of the eight-item version have been shown to be comparable to the original twenty-item version [23]. Five of the eight CES-D items (i.e. felt depressed, was happy, felt lonely, enjoyed life, felt sad) relate to cognitive/affective symptoms, while the remaining three (i.e. everything was an effort, restless sleep, and could not get going) relate to somatic symptoms [23]. We computed a summary score by adding responses to all eight dichotomous questions (possible range: 0–8). The Cronbach's alpha for the CES-D in this study was 0.76. The Cronbach's alpha for the affective scale was 0.75 and for the somatic scale was 0.52. For secondary analyses we also used a binary CES-D score using the standard cut-off ≥ 3 [24, 25].

2.2.2. Incident chronic disease

Incident chronic disease burden was calculated as the sum of chronic illnesses reported by participants at waves 3, 4, 5, 6 and 7, excluding participants who reported a chronic illness at wave 2 baseline; therefore our dependent variable is an aggregate measure of the total number of illnesses reported by each participant over follow-up. As mentioned, the specific illnesses considered included CHD, other cardiac illnesses, diabetes/high blood glucose, arthritis, osteoporosis, lung disease, cancer and Parkinson's disease. Incident chronic illness burden ranged from 0 to 4. We also considered the incidence of each illness separately (yes/no) in secondary analyses.

2.2.3. Covariates

Covariates were all measured at baseline. Sociodemographic variables included in models were age, sex, ethnicity (White/Non-white) and whether participants were cohabiting with a partner. Socioeconomic status was included in models as quintiles of net financial wealth, which refers to participants' gross financial wealth with financial debt subtracted. Height and weight were collected during the wave 2 nurse visit and body mass index was derived using the standard formula (kg/m^2). Whether or not participants reported being a current smoker (yes/no) or a regular consumer of alcohol (>3 days per week over the past 12 months) were also included. Participants reported the frequency in which they engaged in vigorous, moderate and mild physical activity and we used this data to derive three possible categories reflecting regularity of physical activity: none/mild activity only per week, moderate/vigorous activity once a week, moderate /vigorous activity more than once a week. Doctor diagnosis of hypertension and use of anti-hypertensive medication was self-reported and these responses were combined with objective assessments taken at the nurse visit (hypertension defined as systolic blood pressure >140 and diastolic blood pressure >90) to generate a binary variable (yes/no). Cognitive capacity at baseline was measured by aggregating performance on five objective tests administered in face to face interviews. These were immediate recall, delayed recall, verbal fluency, and speed and accuracy on a letter cancellation task. We z transformed scores on the five tests and averaged these to generate an index of cognitive

function [26].

2.3. Statistical analysis

Model parameters were assessed and since our dependent variable was a count variable with no evidence of over-dispersion following assessment of conditional means and variances and Log Likelihood estimates, Poisson regression was used. Poisson regression models examined the relationship between baseline CES-D scores and incident chronic illness burden over follow-up, controlling for covariates. Covariates were selected on an a priori basis and variables were entered into models simultaneously. Covariates were: age, sex, ethnicity, cohabitation, wealth, smoking, BMI, regular alcohol consumption, regular physical activity, cognitive function, and hypertension. In a separate model, we also examined the contribution of depression symptom subtypes (somatic/affective) on incident chronic illness burden, controlling for covariates. Sensitivity analyses were performed to assess whether the findings were upheld with younger and older adults ($<65/\geq 65$ years old), for both men and women, across wealth quintiles, and after excluding participants who developed an incident illness within the two years after baseline (i.e. by wave 3). This latter analysis was performed to test whether the relationship could be due to undiagnosed diseases confounding the association. Results for these models are presented as adjusted incident rate ratios (IRR) with 95% confidence intervals (CI). We also performed secondary analyses to examine the association between CES-D scores at baseline on individual incident illnesses over follow-up. In these fully adjusted logistic regression analyses, only participants with each specific illness were excluded from the sample at baseline; therefore separate Ns are reported for these models. Results for these models are presented as adjusted odds ratios (OR) with 95% CI. All analyses were conducted using SPSS version 21. Two-tailed tests were used throughout and the significance level was set at $p < .05$.

3. Results

Table 1 displays the demographic, clinical and mood characteristics of the sample. Participants were on average 62.88 years old (standard deviation: 8.49) at baseline, with 1571 (63.6%) of participants being younger than 65 years. The large majority were cohabiting with a partner and of White ethnic origin. The distribution of men and women was almost equal. The majority of the sample were overweight, with 69.3% of participants having a BMI > 25. Most were non-smokers and reported at least weekly moderate or vigorous physical activity. Depression scores were on average low with a range of 0 to 8. Incident chronic illness was common in the sample with 772 (31.2%) of participants reporting one chronic illness, 244 (9.9%) reporting two chronic illnesses, 42 (1.7%) reporting three chronic illnesses, and 7 (0.3%) participants reporting 4 chronic illnesses. 1407 (56.9%) participants did not develop any of the chronic illnesses over the follow-up period. 39.5% of those < 65 years old developed at least one chronic illness over follow-up, compared to 49.3% of those ≥65 years. The most frequently reported illness was arthritis, followed by cancer and diabetes/high blood glucose.

Table 1
Demographic, clinical and biological characteristics of the sample ($N = 2472$).

Characteristic	Mean ± SD or N(%)
Baseline sociodemographics	
Age	62.88 ± 8.49
Female	1256 (50.8)
Ethnicity – White	2429 (98.3)
Cohabiting	1869 (75.6)
Net financial wealth – quintiles	
1	333 (13.5)
2	366 (14.8)
3	493 (19.9)
4	571 (23.1)
5	709 (28.7)
Baseline health and health behaviour	
Current smoker	340 (13.8)
Regular physical activity	
None/only light	291(11.8)
Moderate/vigorous sessions ≤1 a week	618 (25.0)
Moderate/vigorous sessions > 1 a week	1563 (63.2)
Regular alcohol drinker (≥3 days/week)	969 (39.2)
Body mass index (kg/m^2)	27.40 ± 4.52
Hypertension	1234 (49.9)
Cognitive function	0.16 ± 0.56
Baseline mood	
CES-D total score	1.09 ± 1.63
CES-D somatic symptom score	0.57 ± 0.82
CES-D affective symptom score	0.52 ± 1.07
Follow-up morbidity (incident cases)	
Burden (n chronic illnesses)	0.57 ± 0.76
Coronary heart disease	105 (4.2)
Other cardiac illness	111 (4.5)
Stroke	80 (3.2)
Diabetes/high blood glucose	160 (6.5)
Cancer	211 (8.5)
Lung disease	77 (3.1)
Osteoporosis	119 (4.8)
Arthritis	530 (21.4)
Parkinson's disease	21 (0.8)

3.1. The association between baseline depressive symptoms and incident chronic illness burden

In an unadjusted model, baseline CES-D was a significant predictor of incident illness burden (IRR = 1.05, $p = .001$). Baseline CES-D score remained a significant predictor of incident chronic illness burden up to 10 years later (IRR = 1.05, $p = .003$) independent of covariates in fully adjusted models (see Table 2). The association was positive such that for each 1-point increase in depressive symptoms there was a 5.0% increase in the incidence of chronic illness at follow-up. The only other significant predictors in the final model were age (IRR = 1.02, $p < .001$), BMI (IRR = 1.03, $p < .001$) and hypertension (IRR = 1.18, $p = .003$). The analyses were replicated in models using binary CES-D as the predictor, with elevated scores on the CES-D associated with a 19% greater incidence of chronic illness burden compared to those with low CES-D scores at baseline, in fully adjusted models (IRR = 1.19, $p = .02$).

Next, analyses to examine the role of somatic and affective depressive symptoms for chronic illness burden were performed, with both symptom subtypes being entered into models simultaneously. Results showed a significant positive association between somatic depressive symptoms (IRR = 1.09, $p = .01$) and higher chronic illness burden at follow-up after controlling for covariates. However, affective depressive symptoms were non-significant in this model (IRR = 1.02, $p = .42$). The only other significant predictors in the final model were age (IRR = 1.02, $p < .001$), BMI (IRR = 1.03, $p < .001$) and hypertension (IRR = 1.18, $p = .003$).

3.2. Age, sex, wealth and short-latency disease onset sensitivity analyses

We examined age, sex and wealth interactions in our data using both mean-centred interaction terms and by examining 95% confidence intervals of the IRRs in Poisson regression models and found no significant results (for results please see the supplementary file). Sensitivity analyses were also performed to remove participants who developed a chronic illness within the 2 years following baseline. This reduced the sample size to 2068. Baseline depressive symptoms (IRR = 1.05, $p = .006$) continued to predict follow-up chronic illness burden in fully adjusted analyses.

Table 2
Baseline depressive symptoms predicting incident chronic illness burden at follow-up (N = 2472).

Model	IRR	95% CI		p
		Lower	Upper	
Age	1.02	1.01	1.02	< 0.001
Sex				
Male	Reference			
Female	1.04	0.93	1.16	0.47
Ethnicity				
White	Reference			
Non-White	1.04	0.72	1.50	0.84
Cohabitation				
Cohabiting	Reference			
Non-cohabiting	0.95	0.84	1.08	0.43
Wealth				
1 (Poorest)	Reference			
2	0.94	0.77	1.14	0.50
3	0.90	0.74	1.08	0.25
4	0.96	0.79	1.15	0.65
5 (Wealthiest)	0.98	0.82	1.18	0.84
BMI	1.03	1.02	1.05	< 0.001
Smoking				
Non-smoker	Reference			
Smoker	1.05	0.89	1.23	0.56
Alcohol consumption				
< 3 days a week	Reference			
≥3 days a week	0.97	0.87	1.09	0.63
Regular physical activity				
Light/none weekly	Reference			
Moderate/vigorous ≤1 a week	0.91	0.76	1.10	0.33
Moderate/vigorous > 1 a week	0.92	0.78	1.08	0.29
Hypertension				
Not hypertensive	Reference			
Hypertensive	1.18	1.06	1.32	0.003
Cognitive function	0.98	0.87	1.10	0.68
Baseline CES-D	1.05	1.02	1.08	0.003

3.3. The association between baseline depression and individual incident illnesses

Logistic regression models were performed to predict each incident illness, excluding those with that same illness at baseline. Full details of these analyses are displayed in Table 3. In fully adjusted models, baseline depressive symptoms were a significant predictor of incident CHD (OR 1.08, 95% CI 1.02–1.15, $p = .01$), other cardiac illnesses (OR 1.10, 95% CI 1.04–1.17, $p = .001$), lung disease (OR 1.13, 95% CI 1.07–1.20, $p < .001$), arthritis (OR 1.09, 95% CI 1.04–1.13, $p < .001$), and osteoporosis (OR 1.12, 95% CI 1.06–1.18, $p < .001$), but not diabetes/high blood glucose, stroke, cancer or Parkinson's disease.

Table 3
Prospective associations between baseline CES-D and individual incident chronic illnesses over follow-up.

Incident disease	Cases/no incident disease	Adjusted odds ratio[a] (95% C.I.)	p
CHD	343/5030	1.08 (1.02–1.15)	0.01
Other cardiac illnesses	361/5031	1.10 (1.04–1.17)	0.001
Diabetes/high blood glucose	426/5207	1.03 (0.98–1.09)	0.24
Stroke	272/5623	0.98 (0.91–1.05)	0.55
Lung disease	258/5464	1.13 (1.07–1.20)	< 0.001
Arthritis	879/3001	1.09 (1.04–1.13)	< 0.001
Osteoporosis	419/5320	1.12 (1.06–1.18)	< 0.001
Cancer	485/5204	1.01 (0.96–1.07)	0.64
Parkinson's disease	55/6055	1.08 (0.93–1.25)	0.35

[a] Age, sex, ethnicity, cohabitation, wealth, smoking, BMI, regular alcohol consumption, regular physical activity, cognitive function and hypertension.

4. Discussion

This study sought to examine the effects of depressive symptoms on incident chronic disease burden over a 10 year follow-up period using data from a national representative cohort of men and women aged 52 and above. We also aimed to explore the association between depressive symptoms and individual incident illnesses that might shed light on the cumulative model. Our results showed that greater depressive symptoms, particularly those somatic in nature, predicted incident chronic disease burden over time and these results were robust to adjustment for multiple covariates and after excluding illnesses with an onset in the two years after baseline. Our findings revealed that the associations between baseline depressive symptoms and future morbidity burden were not dependent on age, sex or wealth. In secondary analyses we showed that greater depressive symptoms at baseline significantly predicted increased odds of incident CHD, other cardiac illnesses, lung disease, osteoporosis and arthritis, but not incident diabetes/high blood glucose, stroke, cancer or Parkinson's disease.

Our findings showed physical illness to be prevalent in ELSA. At wave 2 5481 (62.43%) ELSA core participants were excluded from our analyses because of an underlying physical illness (see Fig. 1). These figures are congruent with those reported by Barnett et al. [3] who found 42.2% of all patients registered with primary care practices in Scotland had one or more physical illnesses. Moreover, pertinent to interpreting our results, these authors also found in favour of an age effect, with over half of those aged 50 years having at least one physical illness and by the age of 65 years most were multimorbid. Our results reinforce the need to understand the epidemiology of multimorbidity in order to tackle this widespread health challenge.

The importance of depression for physical health has been shown in previous research. Depression is known to be highly prevalent in chronic physical illnesses [9] and is not only associated with having a chronic illness diagnosis but also symptom burden above and beyond disease severity [27]. While longitudinal research is sparse, there is some evidence that depression is important in multimorbidity at least cross-sectionally. For example, Pruchno et al. [28] studied the cross-sectional associations between depressive symptoms and combinations of chronic physical illnesses in a sample of community-dwelling 50–74 year olds taking part in the ORANJ BOWL study. Results showed that physical illness was associated with depression in a dose-response manner and more specifically that those participants who had arthritis and pulmonary disease and those who had arthritis and pulmonary disease and heart disease were most likely to have the highest depression symptomatology. In another cross-sectional study, Smith and colleagues [10] showed that those with depression were more likely to have multiple physical illnesses than controls. To the best of our knowledge, our study is the first to demonstrate the prospective association between depressive symptoms and incident chronic disease burden. These findings have clear clinical implications, corroborating the need for early detection and treatment of depression for future health benefits [29], with recent work on the efficacy of antidepressants [30] paving the way for future investment into depression treatments, specifically among the physically ill.

The somatic dimension of depression symptomatology may be particularly important in understanding this association between depression and physical illness [20, 31, 32], which in part might be explained by overlapping symptom profiles between mental and physical health, such as appetitive changes, sleep disturbances and changes to sexual functioning. Our results corroborated the importance of somatic symptoms for chronic illness burden, but due to the brief nature of our depression measure further work is needed to understand these effects in more detail. Age, sex and wealth were not shown to interact with depressive symptoms in our analyses suggesting these factors were not moderators of the association between depression and physical illness burden in ELSA.

Not only did we test the association between depressive symptoms

and the accumulation of physical illnesses over time, but also the relationship depressive symptoms had with individual illnesses that might help explain this effect. Several good quality studies and meta-analyses have found depression to be implicated in the aetiology of several different chronic illnesses including CHD [33–35], stroke [36], diabetes [14], cancer [16] and hormonally-mediated cancer more specifically [37], and dementia [38]. Previously published work using ELSA has supported the link between depression and risk of diabetes [39]; however this association was not supported here perhaps due to methodological differences. For example, we had 10 years of follow-up data available in our analyses compared to the Demakakos study which used 4 years of follow-up; in conjunction with differences in coding and covariates these differences may have contributed to the discrepancy between the effect estimates. Another ELSA study has looked at the association between positive wellbeing and chronic disease incidence showing an inverse association with arthritis onset and, in those under 65 years old, an inverse association with diabetes and lung disease risk [40]. In line with these previous findings we showed depressive symptoms to predict incident CHD, other cardiac illnesses (heart murmur, heart failure and cardiac arrhythmia), lung disease, arthritis and osteoporosis. However, we were unable to support evidence for a link between depressive symptoms and stroke, cancer, diabetes/high blood glucose or Parkinson's disease onset. It is unclear why our findings we unable to corroborate previous literature in the field, but it is possible these exploratory analyses were underpowered. For example, we only had 55 incident cases of Parkinson's disease, which could have reduced our ability to detect an effect; using G*Power (version 3.1) it was estimated that a sample size of 14,713 participants would have been needed to detect an effect for Parkinson's disease. With regards to the cancer findings, it has been suggested that publication biases exist and that results for depression may be strongest for certain types of cancer such as lung cancers; an association which may be mediated by tobacco use [16]. Greater work to tease out the individual effects taking into the account the heterogeneity of cancer diagnoses is warranted.

There is a large literature investigating the mechanisms by which depression may affect physical health including behavioural, cognitive and biological pathways. For example, depression has long been associated with health negative behaviours such as smoking [41, 42], obesity [43], physical inactivity [44] and medication non-adherence [45]. Cognitive function and depression are also known to be closely related, although the direction of the causal pathway is not yet established [46]. We attempted to address these factors by controlling for a wide range of covariates. However, depression is also known to be associated with important biological pathways that have clear implications for disease risk, which we were unable to account for in these analyses. For example, cortisol has been implicated in the pathophysiology of depression [47] and hypothalamic pituitary adrenal axis abnormalities have been observed in patients with major depression, including increased secretion and reactivity of cortisol [48], elevated corticotrophin releasing hormone [49, 50], and increased size and activity of the pituitary and adrenal glands [51, 52]. Depression is also associated with an innate inflammatory response [53], and a cumulative meta-analysis by Haapakoski and colleagues has shown depressive symptoms to be positively associated with C-reactive protein and interleukin (IL)-6 [54]. Interestingly, depression is associated with both immune up-regulation, characterised by increasing pro-inflammatory cytokines, as well as immune down-regulation such as reduced proliferative responses of immune cells [55]. Future work is needed to assess how these biological changes in those with depression vary across different physical illnesses in order to establish common mechanistic pathways.

Our study has a number of strengths. Firstly, the data were drawn from ELSA which is a large, nationally representative cohort of adults living in the UK. The prospective nature of our analyses has allowed us to assess the temporal relationship between depressive symptoms and future disease onset. We were able to control for a large number of covariates to take into account the influence of potential confounders of the relationship. While these covariates are useful to retain in analyses to circumvent residual confounding, it must be acknowledged that some of these variables are also possible mediators, though we did not find evidence for this in our analyses. Moreover, our findings were upheld in sensitivity analyses removing illnesses with an onset two years after baseline. We also took into account two different symptom profiles of depression, namely both affective and somatic depressive symptoms. However, we must also acknowledge some weaknesses. We relied on self-report measures of both depressive symptoms and physical illness, but this may not negate our findings since there is evidence for high agreement between self-report and medical record validation in population studies [56–58]. In addition, we were limited to the number of physical illnesses we could include in models by the need for consistent measures to have been taken across all waves of data collection. We computed morbidity as a count measure of chronic illness burden and did not take into account the severity of the illnesses or the impact on functional decline. Further replication of our findings in studies which utilise a multimorbidity instrument such as the Charlson Index [59] would therefore be beneficial. Another limitation is that our outcome variable did not take into account the time at which new diseases were first reported so we are unable to comment on the rate of illness burden. Finally, we must acknowledge that we did not take into account the time-varying course of depression, and our results are restricted to depression symptoms measured at wave 2 only.

In conclusion, we found that depressive symptoms were associated with greater incident chronic illness burden 10 years later, in a large, nationally representative sample of adults aged 52 years or older living in England. These findings have clear clinical implications for the screening and treatment of depression in physically healthy adults.

Ethical approval

Ethical approval for all the ELSA waves was granted from the National Research and Ethics Committee (http://www.nres.npsa.nhs.uk/).

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Declaration of interest

Both authors have completed the ICMJE Form for Disclosure of Potential Conflicts of Interest and have no conflicts to declare.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychores.2018.07.009.

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