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# Unraveling the link between physical activity and cognitive function: the mediating impact of depressive symptoms

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## Abstract

**Background** This study investigates the association between physical activity and cognitive function in adults aged 50 and older, with a focus on the mediating role of depressive symptoms.

**Methods** Data were drawn from Waves 7 to 9 of the English Longitudinal Study of Ageing (ELSA), including 5,499 participants aged 50 years and older. Cognitive function was assessed through measures of episodic memory, numeracy, and orientation. Physical activity was quantified using a composite measure combining the frequency of vigorous, moderate, and light exercise with their respective average metabolic equivalents. Depressive symptoms were evaluated using the CES-D-8 scale. Generalized estimating equations (GEE) were employed to examine the association between physical activity and cognitive function, while the Bootstrap method was utilized to evaluate the mediating role of depressive symptoms.

**Results** GEE analyses revealed that higher levels of physical activity were significantly associated with improved global cognitive function ( $\beta = 0.151$ , 95%CI: 0.118–0.183), episodic memory ( $\beta = 0.074$ , 95%CI: 0.058–0.090), numeracy ( $\beta = 0.033$ , 95%CI: 0.015–0.051), and orientation ( $\beta = 0.039$ , 95%CI: 0.022–0.057) (all  $P < 0.001$ ). Physical activity also demonstrated a significant negative association with depressive symptoms ( $\beta = -0.402$ , 95%CI: -0.452 to -0.352) ( $P < 0.001$ ), which, in turn, were linked to lower cognitive function. Notably, depressive symptoms mediated 8.46% of the association between physical activity and global cognitive function.

**Conclusions** Greater engagement in physical activity is associated with better cognitive function, and this association is partially mediated by depressive symptoms. These findings underscore the importance of promoting physical activity to support cognitive health in older adults, with particular attention to its potential role in mitigating depressive symptoms.

**Keywords** Physical activity, Cognitive function, Depressive symptoms, Cohort study

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## Background

Cognitive function naturally declines with age, often progressing through stages from subjective cognitive impairment to mild cognitive impairment, and eventually leading to dementia. This decline can impair complex attention, executive function, learning and memory, language, perceptual motor abilities, and social cognition, significantly affecting an individual's quality of life [1]. Multiple risk factors for cognitive decline have been identified, including low physical activity levels, poor sleep patterns, reduced social engagement, depressive symptoms, and limited educational attainment [2, 3]. Among these, physical activity has received particular attention due to its potential protective effects. Lower levels of physical activity are consistently associated with an increased risk of cognitive decline and dementia, whereas individuals engaging in higher levels of physical activity tend to exhibit better cognitive performance [4, 5]. In addition to its cognitive benefits, physical activity has been linked to lower rates of depression and fewer depressive symptoms [6–8]. Depression, in turn, is strongly associated with accelerated cognitive decline and a increased risk of developing dementia [9, 10].

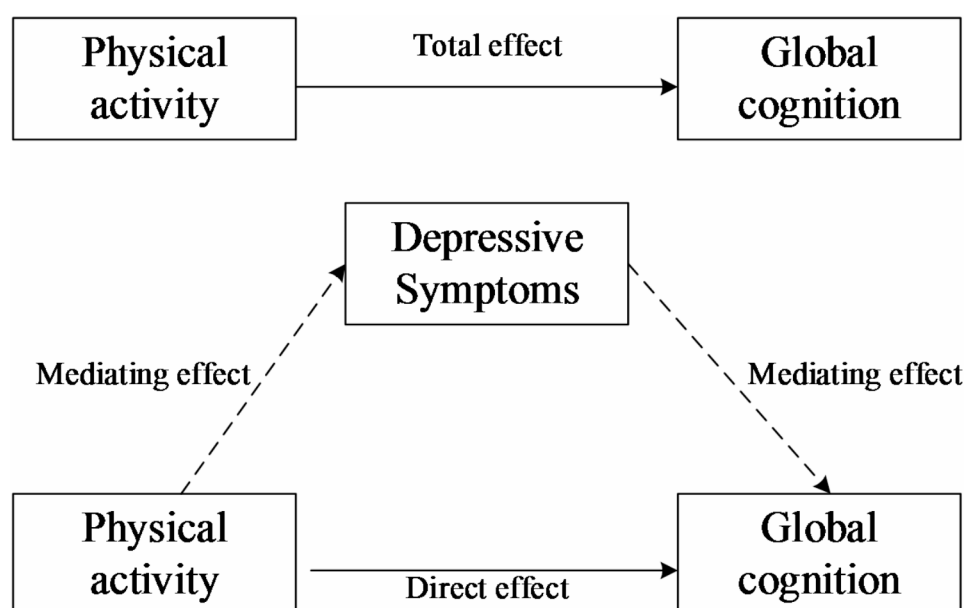
Despite this, the mechanisms underlying these associations remain poorly understood. This study hypothesizes that physical activity influences cognitive function both directly and indirectly, with depressive symptoms acting as a mediator (Fig. 1). Specifically, reduced physical activity may increase the risk of developing depressive symptoms, which in turn could exacerbate cognitive impairment. While prior studies have explored the interplay between physical activity, depressive symptoms, and

cognitive function, most have relied on cross-sectional designs, limiting their ability to infer causal relationships [11, 12]. To address this gap, the present research leverages longitudinal data from a large aging cohort to investigate these relationships more rigorously. Using a mediation analysis framework, this study aims to quantify the mediating role of depressive symptoms in the association between physical activity and cognitive function, shedding light on the pathways through which physical activity influences cognitive health. Ultimately, this study seeks to clarify the association between physical activity and cognitive function while elucidating the role of depressive symptoms in this relationship.

## Methods

### Study population

This study utilized data from the English Longitudinal Study of Ageing (ELSA), a national longitudinal survey of adults aged 50 and older in the United Kingdom. The initial cohort was recruited from the Health Survey for England (HSE) in 1998, 1999, and 2001, using eligibility criteria designed to ensure broad representativeness of the UK population, as confirmed by comparisons with national census data [13]. Since 2002, follow-up surveys have been conducted biennially, with additional nurse visits every four years to collect biomarker data. Regular updates to the sample ensure ongoing representativeness of the 50+ population. The original ELSA cohort included 11,391 participants, with data collected through computer-assisted personal interviews and self-completed questionnaires [14]. For this study, data from Waves 7 to 9 were used, given the availability of comprehensive



**Fig. 1** Hypothesized mediation model

cognitive function measures. Participants diagnosed with Alzheimer's Disease (AD) or dementia and those lost to follow-up were excluded. Attrition analyses indicated no significant impact on the representativeness of the remaining sample. Ultimately, 5,499 participants with complete data on physical activity, depressive symptoms, and cognitive function across three waves were included (Fig. 2).

## Measurement

### Demographic data and covariates

Demographic variables included age, gender, ethnicity, marital status, and educational attainment. Education levels were categorized as below O-level (no formal qualifications), O-level (equivalent to GCSEs, typically taken at age 16), or A-level and above (typically taken at ages 16–18 and used for university entry). Marital status was grouped into married/cohabiting and separated/divorced/widowed/never married. Smoking status was classified into current, former, and never smokers based on participants' responses to smoking history. Alcohol consumption over the past year was categorized as daily, frequent (1–2 times per week or more), or rare (1–2 times per month or less). Chronic disease history (hypertension, diabetes, stroke, hypercholesterolemia, arthritis, osteoporosis, cancer, chronic lung diseases, and heart disease) was self-reported based on physician diagnoses, adhering to international diagnostic standards.

In all analyses, age, marital status, education, alcohol consumption, smoking, and chronic conditions were included as time-varying covariates (2014/2016/2018).

### Physical activity measurement

Physical activity levels were assessed based on the frequency of light, moderate, and vigorous activities. Participants reported engagement frequency as “more than once a week,” “once a week,” “1–3 times a month,” or “rarely/never,” with examples provided for each intensity level. To quantify activity levels, frequencies were converted into “times per month” and combined with metabolic equivalent estimates to calculate a total physical activity score (MET×times/month), which was then standardized [15, 16]. (Table A.1). This score represents the total monthly activity level, with higher values indicating greater physical activity.

### Cognitive function measurement

Cognitive function was evaluated across three domains: episodic memory, numeracy, and orientation. Episodic memory was assessed using a word recall test, with scores ranging from 0 to 20. Numeracy was measured through a serial subtraction task (scores: 0–5), and orientation involved questions on date and time (scores: 0–4). Standardized domain scores were summed to create a

global cognitive function score, with higher scores indicating better cognitive function.

### Depressive symptoms measurement

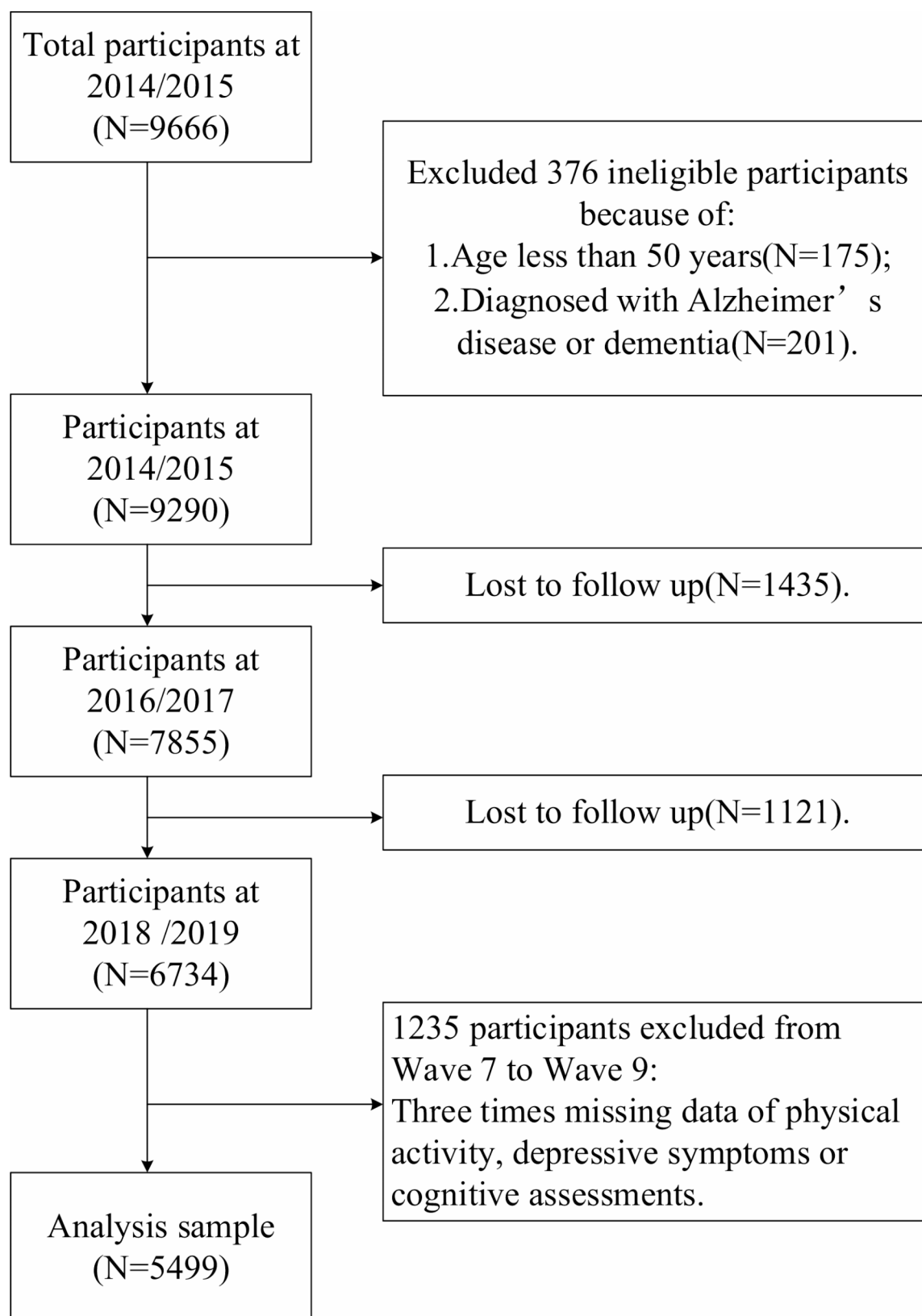
Depressive symptoms were measured using the eight items of the Center for Epidemiologic Studies Depression Scale (CES-D-8 scale), where participants answered “yes” or “no” to items based on their feelings in the past week. Scores ranged from 0 to 8, with a threshold of  $\geq 4$  indicating significant depressive symptoms [17, 18].

### Follow-up survey

Wave 7 (2014–2015) served as the baseline for this study. Cognitive function, depressive symptoms, and physical activity were reassessed at Waves 8 and 9, following consistent protocols.

### Statistical analysis

Data analysis was conducted using SPSS and Stata. Continuous variables were presented as means  $\pm$  standard deviations, with comparisons made using ANOVA or Kruskal-Wallis tests as appropriate. Categorical variables were summarized as frequencies and percentages, and chi-square tests were used for group comparisons. Cognitive function scores were grouped into quartiles ( $Q_1 \leq -1.223$ ,  $Q_2$  -1.223 to 0.312,  $Q_3$  0.312 to 1.500,  $Q_4 \geq 1.500$ ) for descriptive analyses and group comparisons. Multiple imputation addressed missing covariates [19] (Table A.2). Generalized Estimating Equations (GEE) were used to assess correlations and potential mediating effects, three models were constructed to adjust for relevant covariates. For cognitive function and physical activity (a continuous variable), we specified a Gaussian distribution with an identity link (linear regression). For depressive symptoms (a binary variable), we specified a binomial distribution with a logit link (logistic regression). Continuous cognitive function scores were used in the GEE and mediation analyses. Meanwhile, the interaction effects between the exposure variables and the mediating variables were assessed separately. To address potential concerns about temporality and reverse causation, we conducted sensitivity analyses using lagged variables. Specifically, physical activity and depressive symptoms from Wave  $t-1$  were used to predict cognitive function at Wave  $t$ . The mediation significance was assessed with the Hayes and Preacher method by bootstrap [20], with simulation samples used to generate 95% CIs for the indirect effect and the direct effect. Gender-stratified sensitivity analyses were conducted. Statistical significance was set at  $P < 0.05$ .

**Fig. 2** The flow diagram of study participants

**Table 1** Characteristics of the study sample at baseline

Variable	Total n = 5499	Q <sub>1</sub> n = 1445	Q <sub>2</sub> n = 1298	Q <sub>3</sub> n = 1750	Q <sub>4</sub> n = 1006	PValues
<b>Age mean (SD), years</b>	66.28(8.59)	68.59(9.30)	67.29(8.74)	65.50(8.05)	63.03(6.90)	< 0.001
<b>Gender, N (%)</b>						
Male	2511(45.66)	663(45.88)	602(46.38)	874(49.94)	372(36.98)	< 0.001
Female	2988(54.34)	782(54.12)	696(53.62)	876(50.06)	634(63.02)	
<b>Education, N (%)</b>						
O level or below	1894(34.44)	670(46.37)	485(37.37)	522(29.83)	217(21.57)	< 0.001
O level	1694(30.81)	406(28.10)	403(31.05)	568(32.46)	317(31.51)	
A level or higher	1911(34.75)	369(25.54)	410(31.59)	660(37.71)	472(46.92)	
<b>Ethnicity, N (%)</b>						
White	5343(97.16)	1382(95.64)	1257(96.84)	1716(98.06)	988(98.21)	< 0.001
Non-white	156(2.84)	63(4.36)	41(3.16)	34(1.94)	18(1.79)	
<b>Marital status, N (%)</b>						
Married/Cohabiting	4068(73.98)	1033(71.49)	941(72.50)	1292(73.83)	802(79.72)	< 0.001
Other	1431(26.02)	412(28.51)	357(27.50)	458(26.17)	204(20.28)	
<b>Smoking, N (%)</b>						
Never smoker	2193(39.88)	519(35.92)	500(38.52)	722(41.26)	452(44.93)	< 0.001
Former smoker	2773(50.43)	755(52.25)	675(52.00)	873(49.89)	470(46.72)	
Current smoker	533(9.69)	171(11.83)	123(9.48)	155(8.86)	84(8.35)	
<b>Drinking, N (%)</b>						
Daily	2616(47.57)	607(42.01)	593(45.69)	867(49.54)	549(54.57)	< 0.001
1–2 times a week or more	748(13.60)	174(12.04)	184(14.18)	253(14.46)	137(13.62)	
1–2 times a month or less	2135(38.83)	664(45.95)	521(40.14)	630(36.00)	320(31.81)	
<b>Hypertension, N (%)</b>	3313(60.25)	873(60.42)	768(59.17)	1061(60.63)	611(60.74)	0.836
<b>Diabetes, N (%)</b>	2934(53.36)	720(49.83)	666(51.31)	964(55.09)	584(58.05)	< 0.001
<b>Stroke, N (%)</b>	2579(46.90)	626(43.32)	572(44.07)	852(48.69)	529(52.58)	< 0.001
<b>hypercholesterolemia</b>	3165(57.56)	792(54.81)	736(56.70)	1053(60.17)	584(58.05)	0.020
<b>Arthritis, N (%)</b>	3745(68.10)	989(68.44)	888(68.41)	1180(67.43)	688(68.39)	0.910
<b>Osteoporosis, N (%)</b>	2790(50.74)	692(47.89)	637(49.08)	901(51.49)	560(55.67)	0.001
<b>Cancer, N (%)</b>	883(16.06)	193(13.36)	192(14.79)	299(17.09)	199(19.78)	< 0.001
<b>Chronic lung diseases, N (%)</b>	2782(50.59)	675(46.71)	631(48.61)	904(51.66)	572(56.86)	< 0.001
<b>Heart disease, N (%)</b>	1569(28.53)	465(32.18)	372(28.66)	481(27.49)	251(24.95)	0.001
<b>Physical activity, mean (SD)</b>	66.30(34.66)	59.33(34.81)	63.70(34.39)	69.10(34.09)	74.79(33.43)	< 0.001
<b>Depressive symptoms, N (%)</b>	1144(20.80)	378(26.16)	293(22.57)	323(18.46)	150(14.91)	< 0.001

## Results

### Baseline characteristics and bivariate associations

Baseline characteristics are summarized in Table 1. Participants had a mean age of  $66.28 \pm 8.59$  years, with 54.34% identifying as female and 97.16% as White. The majority (73.98%) were married, and 34.75% had achieved A-level education or higher. The mean score for unstandardised physical activity was 66.30, and 20.80% scored  $\geq 4$  on the CES-D-8 scale, indicating depressive symptoms. Prevalence of chronic conditions was high, with hypertension (60.25%) and arthritis (68.10%) being most common. Bivariate analysis revealed significant differences in demographic and health characteristics across cognitive function quartiles, except for hypertension and arthritis prevalence ( $P > 0.05$ ).

### Associations between physical activity and cognitive function as well as depressive symptoms and cognitive function

After adjusting for demographic information (age, gender, education, ethnicity, marital status), lifestyle (smoking, drinking), and medical history (hypertension, diabetes, stroke, hypercholesterolemia, arthritis, osteoporosis, cancer, chronic lung diseases, heart disease), GEE analyses showed that higher physical activity levels were positively associated with global cognitive function ( $\beta = 0.151$ ,  $P < 0.001$ ), episodic memory ( $\beta = 0.074$ ,  $P < 0.001$ ), numeracy ( $\beta = 0.033$ ,  $P < 0.001$ ), and orientation ( $\beta = 0.039$ ,  $P < 0.001$ ), (Table 2). Depressive symptoms were negatively associated with global cognitive function ( $\beta = -0.099$ ,  $P = 0.007$ ), and episodic memory ( $\beta = -0.073$ ,  $P < 0.001$ ), but not with numeracy or orientation ( $P > 0.05$ ) (Table 2; Fig. 3).

**Table 2** Associations between physical activity or depressive symptoms and cognitive function

	Global cognition		Episodic memory		Numeracy		Orientation	
	$\beta$ (95% CI)	P	$\beta$ (95% CI)	P	$\beta$ (95% CI)	P	$\beta$ (95% CI)	P
<b>Model 1<sup>a</sup></b>								
<b>Physical activity</b>	0.277(0.244,0.309)	< 0.001	0.129(0.112,0.145)	< 0.001	0.070(0.053,0.087)	< 0.001	0.070(0.053,0.088)	< 0.001
<b>Depressive symptoms</b>								
Non-depressed	Reference		Reference		Reference		Reference	
Depressed	-0.144(-0.218, -0.071)	< 0.001	-0.085(-0.119, -0.052)	< 0.001	-0.066(-0.105, -0.026)	0.001	-0.022(-0.063, -0.019)	0.296
<b>Interact<sup>d</sup></b>	0.030(-0.043,0.104)	0.416	0.042(0.009,0.076)	0.012	-0.010(-0.048,0.272)	0.587	-0.001(-0.043,0.040)	0.947
<b>Model 2<sup>b</sup></b>								
<b>Physical activity</b>	0.173(0.141,0.205)	< 0.001	0.083(0.067,0.099)	< 0.001	0.042(0.025,0.060)	< 0.001	0.045(0.028,0.062)	< 0.001
<b>Depressive symptoms</b>								
Non-depressed	Reference		Reference		Reference		Reference	
Depressed	-0.117(-0.189, -0.045)	0.001	-0.079(-0.112, -0.047)	< 0.001	-0.036(-0.076, 0.003)	0.072	-0.024(-0.065,0.017)	0.255
<b>Interact<sup>d</sup></b>	0.019(-0.052,0.090)	0.593	0.035(0.003,0.067)	0.031	-0.006(-0.043,0.031)	0.754	-0.008(-0.050,0.033)	0.691
<b>Model 3<sup>c</sup></b>								
<b>Physical activity</b>	0.151(0.118,0.183)	< 0.001	0.074(0.058,0.090)	< 0.001	0.033(0.015,0.051)	< 0.001	0.039(0.022,0.057)	< 0.001
<b>Depressive symptoms</b>								
Non-depressed	Reference		Reference		Reference		Reference	
Depressed	-0.099(-0.171, -0.027)	0.007	-0.073(-0.106, -0.041)	< 0.001	-0.027(-0.067,0.012)	0.178	-0.020(-0.061,0.021)	0.346
<b>Interact<sup>d</sup></b>	0.015(-0.055,0.086)	0.669	0.033(0.002,0.065)	0.040	-0.007(-0.044,0.030)	0.696	-0.010(-0.051,0.032)	0.651

<sup>a</sup> Model 1 no covariates were adjusted<sup>b</sup> Model 2 adjusted for age(continuous), gender, education, ethnicity, marital status<sup>c</sup> Model 3 additionally adjusted for smoking, drinking, hypertension, diabetes, stroke, hypercholesterolemia, arthritis, osteoporosis, cancer, chronic lung diseases, and heart disease

CI, confidence interval

<sup>d</sup> Physical activity \* Depressive symptoms (interaction effect values only)

### Associations between physical activity and depressive symptoms

As previously mentioned, depressive symptoms were assessed as a dichotomous variable based on CES-D-8 scores, and Table 2 describes the effects of physical activity on cognitive functioning as well as the effects of depressive symptoms on cognitive functioning. Higher physical activity levels were inversely associated with depressive symptoms ( $\beta = -0.402$ ,  $P < 0.001$ ) (Table 3).

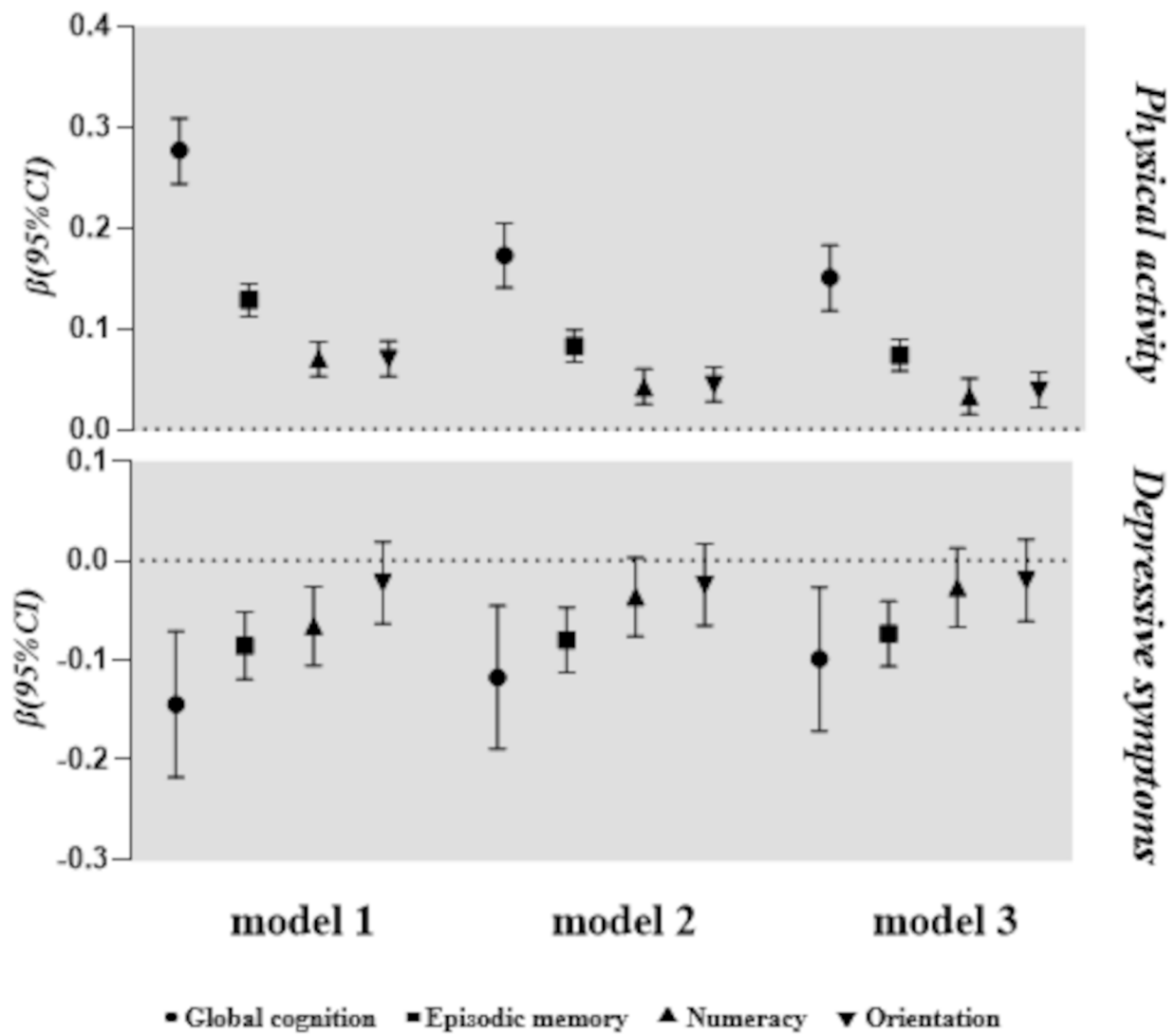
### Mediation analysis

After adjusting for covariates, mediation analysis revealed that depressive symptoms partially mediated the association between physical activity and global cognitive function. Specifically, with depressive symptoms accounting for 8.46% of the effect (Table 4). Mediation effects for episodic memory and numeracy were 8.31% and 12.73%, respectively (Fig. 4).

### Sensitivity analysis

In this sensitivity analysis, to test for effect modification by gender, we included an interaction term between

gender and physical activity in the GEE models. The interaction term was statistically significant ( $\beta = 0.074$ ,  $P = 0.026$ ), indicating that the association between physical activity and cognitive function differed by gender. A gender-stratified approach was used for in-depth study. Gender-stratified analysis showed that physical activity significantly improved cognitive outcomes in both men and women. Furthermore, depression symptoms were found to have a significant negative association with global cognitive function and episodic memory, but no significant association with numeracy and orientation (Fig. A.1). In the subsequent mediation effect analysis, it was observed that in the male population, depression symptoms only partially mediated the association between physical activity and global cognitive function as well as episodic memory. In the female population, however, depression symptoms acted as a mediator in the association between physical activity and global cognitive function, episodic memory, and numeracy (Table A.3). Overall, Depressive symptoms partially mediated the association between physical activity and cognitive



**Fig. 3** Associations ( $\beta$ [95%CI]) between physical activity or depressive symptoms, and cognitive function

**Table 3** Associations between physical activity and depressive symptoms

	Model 1 <sup>a</sup>		Model 2 <sup>b</sup>		Model 3 <sup>c</sup>	
	$\beta$ (95% CI)	P	$\beta$ (95% CI)	P	$\beta$ (95% CI)	P
Physical activity	-0.497(-0.544, -0.451)	< 0.001	-0.443(-0.493, -0.393)	< 0.001	-0.402(-0.452, -0.352)	< 0.001

<sup>a</sup> Model 1 without any adjustment  
<sup>b</sup> Model 2 adjusted for age, gender, education, ethnicity, marital status  
<sup>c</sup> Model 3 additionally adjusted for smoking, drinking, hypertension, diabetes, stroke, hypercholesterolemia, arthritis, osteoporosis, cancer, chronic lung diseases, and heart disease  
CI, confidence interval

function, with stronger mediation effects observed in men than women.

Sensitivity analyses using lagged variables revealed that physical activity at Wave t-1 was significantly associated with better cognitive function at Wave t ( $\beta=0.118$ ,  $P<0.001$ ); physical activity at Wave t-1 was significantly

associated with depressive symptoms at Wave t ( $\beta = -0.313$ ,  $P<0.001$ ), depressive symptoms at Wave t-1 were significantly associated with poorer cognitive function at Wave t ( $\beta = -0.284$ ,  $P<0.001$ ). These results support the robustness of our primary findings and suggest that the associations are not solely driven by reverse causation.



**Table 4** Direct and indirect effect between physical activity and global cognition mediated by depressive symptoms

Cognition function	$\beta$ (95%CI)	Mediation (%)
<b>Global Cognition</b>		
Indirect effect	0.016(0.010,0.021)	8.46%
Direct effect	0.170(0.139,0.201)	
Total effect	0.186(0.155,0.216)	
<b>Episodic Memory</b>		
Indirect effect	0.009(0.006,0.012)	8.31%
Direct effect	0.101(0.086,0.117)	
Total effect	0.110(0.095,0.126)	
<b>Numeracy</b>		
Indirect effect	0.004(0.002,0.007)	12.73%
Direct effect	0.030(0.013,0.047)	
Total effect	0.034(0.018,0.051)	
<b>Orientation</b>		
Indirect effect	0.002(-0.001,0.005)	-
Direct effect	0.039(0.022,0.055)	
Total effect	0.041(0.025,0.057)	

Adjusted for age, gender, education, ethnicity, marital status, smoking, drinking, hypertension, diabetes, stroke, hypercholesterolemia, arthritis, osteoporosis, cancer, chronic lung diseases, and heart disease

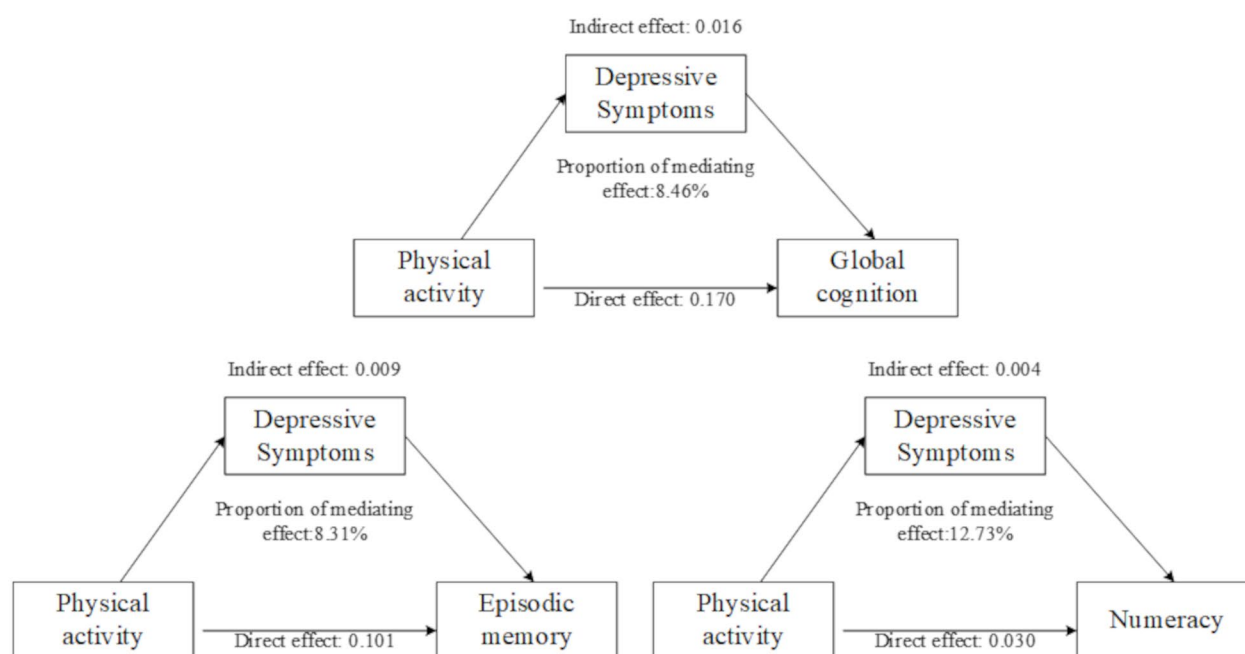
## Discussion

This study, involving 5,499 participants aged 50 and older in the UK, investigated the longitudinal associations between physical activity and cognitive function across various domains. It further examined the mediating role of depressive symptoms in this relationship. Our findings reveal that higher levels of physical activity are significantly associated with better cognitive function and

lower levels of depressive symptoms. Conversely, depressive symptoms were negatively associated with cognitive function, with varying effects across cognitive domains. Mediation analysis indicated that depressive symptoms accounted for 8.46% of the association between physical activity and global cognitive function.

Physical activity demonstrated significant positive associations with global cognitive function and its specific domains, even after adjusting for relevant covariates. These findings align with prior studies reporting the cognitive benefits of physical activity. For example, a cross-sectional study identified positive associations between moderate-to-high intensity exercise and various cognitive domains, including executive function, memory, numeracy, and orientation [21]; Similarly, a randomized clinical trial showed that dance interventions significantly improved global cognition, particularly memory [22]; A meta-analysis further confirmed that physical activity enhances cognitive function in patients with AD and those without cognitive impairment [23, 24]. Mechanistically, physical activity promotes the release of neurotrophic factors such as IGF-1 and BDNF, which support neuroplasticity and improve neurovascular function, particularly in brain regions like the hippocampus [25, 26]. Additionally, physical activity reduces inflammation, mitigates oxidative stress, and modulates endocrine functions, all of which contribute to cognitive preservation during aging [27].

The mediating role of depressive symptoms identified in this study highlights their importance in the

**Fig. 4** Intermediation effect diagrams



association between physical activity and cognitive function. Specifically, depressive symptoms mediated 8.46% of the association, emphasizing their influence on cognitive health. These results are consistent with findings from other cohort studies, which reported that maintaining adequate physical activity reduces depressive symptoms, while depressive symptoms negatively impact cognitive function and increase dementia risk [28–30]. Interestingly, the associations between depressive symptoms and cognitive domains varied, with a significant negative association observed for episodic memory but not for numeracy or orientation. This variability has been documented in prior studies analyzing specific cognitive domains separately [31, 32]. Moreover, prior research using mediation models has demonstrated that depressive symptoms significantly mediate the association between physical activity and cognitive outcomes [33]. Structural equation modeling has also shown that individuals who engage in higher levels of physical activity are less likely to experience depression and tend to exhibit better cognitive performance [34].

The cognitive benefits of physical activity are attributed to multiple mechanisms. Neurobiological effects include the upregulation of neurotrophic factors (e.g., BDNF, VEGF, IGF-1) and enhanced synaptic plasticity, neurogenesis, and vascular function [26, 27]. Additionally, physical activity modulates inflammation by reducing circulating pro-inflammatory cytokines, oxidative stress, and mitochondrial dysfunction. Psychological benefits, such as improved self-esteem, social support, and self-efficacy, further contribute to its antidepressant and cognitive-enhancing effects [35]. These synergistic effects underscore the importance of physical activity as a holistic intervention for cognitive health.

While our primary analysis included repeated measures to capture the dynamic relationships between physical activity, depressive symptoms, and cognitive function, we acknowledge the potential for reverse causation. To address this, we conducted sensitivity analyses using lagged variables, which supported the robustness of our findings. Future studies should explore more sophisticated modeling approaches, such as structural equation modeling or marginal structural models, to further disentangle these effects.

### Strengths and limitations

This study has several notable strengths. Data were sourced from the English Longitudinal Study of Ageing (ELSA), which provides a large, representative sample of 5,499 UK participants aged 50 years and older. The longitudinal design, with repeated measures of physical activity, depressive symptoms, and cognitive function, allowed for robust assessments of their associations over time. Furthermore, the study provides valuable insights

into the mediating role of depressive symptoms in these associations.

However, certain limitations should be acknowledged. First, participants with missing data on physical activity, cognitive assessments, or depressive symptoms were excluded, which may have introduced selection bias. Second, depressive symptoms were assessed using a simplified scale, potentially limiting the depth of evaluation. Third, physical activity data were self-reported, making them susceptible to recall bias, and this approach did not capture important dimensions such as duration and specific types of activity. Lastly, while this study focused on predefined hypotheses, other potential mediating and moderating factors were not explored.

### Conclusion

In conclusion, this study confirms the association between physical activity and cognitive function and highlights the mediating role of depressive symptoms in this association. Although depressive symptoms mediated approximately 8.46% of the association between physical activity and global cognitive function, the effect size is relatively small. This suggests that depressive symptoms play a partial role, other pathways and mechanisms are likely involved in the cognitive benefits of physical activity. These findings suggest that targeting depressive symptoms could mitigate cognitive decline and optimize cognitive outcomes, but future research should explore additional mediators and moderators to provide a more comprehensive understanding of these relationships. Interventions that integrate both physical and mental health strategies hold promise for comprehensive health management and cognitive preservation.

### Abbreviations

CES-D-8	8-item Centre for Epidemiological Studies Depression Scale
CI	Confidence Interval
ELSA	English Longitudinal Study of Ageing
GEE	Generalized Estimating Equations

### Supplementary Information

The online version contains supplementary material available at <https://doi.org/10.1186/s12889-025-22410-2>.

Supplementary Material 1

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### Author contributions

Zhang L.H. and Zhang J.C. designed and conceptualized the present study. Chen Q. and Cai X. curated the data. Zhang L.H. conducted the formal analysis and drafted the initial manuscript. Zu L.P., Liu L.Y., Li C.Y., Wu X.M. and Ma F. were involved in provided resources and supervision/oversight. Ma F. was involved in funding acquisition. All authors reviewed and substantially revised

the manuscript. All authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

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## Data availability

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

## Declarations

### Ethics approval and consent to participate

This study was performed in accordance with the Declaration of Helsinki. This study protocol was reviewed and approved by the NRES Committee South Central - Berkshire on 28th November 2013 (13/SC/0532), the South Central - Berkshire Research Ethics Committee on 23rd September 2015 (15/SC/0526) and the South Central - Berkshire Research Ethics Committee on 10th May 2018 (17/SC/0588). All participants in ELSA provided written informed consent to participate.

### Consent for publication

Not applicable.

### Competing interests

The authors declare no competing interests.

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